**UNIVERSITY OF CALIFORNIA,**
**IRVINE**

# Weighing the Future: An Ethnographic Examination of Epigenetics and Prenatal Interventions

DISSERTATION

Submitted in partial satisfaction of the requirements for the degree of

DOCTOR OF PHILOSOPHY

in Anthropology

by

Natali Valdez

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DEDICATION

Writing a dissertation and completing a doctorate is impossible to do alone. I thank all of the people in my life that helped me through this long journey. I am incredibly grateful to family and friends that walked this path with me.

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## EDUCATION

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| 2012 | School of Public Health, University of California, Berkeley<br>Collaborative Projects:<br>*A Nuanced Examination of Acculturation and Obesity Among Mexican and Mexican-American Women: Using National Health and Nutrition Survey Data from 2005-2008*. Natali Valdez and Lillian Sie |
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Commentaries and Encyclopedia Entries

Forthcoming "Latina Pregnancy." Lydia Zacher-Dixon, **Natali Valdez**, and Michael Montoya. *Oxford Bibliographies.* Oxford, England.

2015 Food, Fat, Fetus, and the Future: Histories of Weight Gain During Pregnancy in the US and UK. Society for Medical Anthropology, *Anthropology News.* May 2015.

Book and Report Reviews

2015 "*50 Great Myths of Human Sexuality* by Pepper Schwartz and Martha Kempner." West Sussex, U.K: John Wiley and Sons, Inc.

2015 "A Review of the 2015 CfSS Report: The Business of People: The Significance of Social Science over the Next Decade." *Anthropology in Action.*

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Ethnographer on a multi-sited randomized clinical trial

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Ethnographer, nutritional counselor, and volunteer staff member on multi-sited randomized clinical trial

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Staff member and community liaison of the Family Health and Self-Empowerment Project, a multi-million dollar community health intervention

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Co-ethnographer and research assistant on the international project titled, "The effects of a Western-Biomedical Clinic on the practices of traditional healers in an indigenous area in Mexico"

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**ABSTRACT OF THE DISSERTATION**

Weighing the Future: An Ethnographic Examination of Epigenetics and Prenatal Interventions

by

Natali Valdez

Doctor of Philosophy in Anthropology

University of California, Irvine, 2016

Associate Professor Kristin Peterson, Co-Chair

Associate Professor Michael Montoya, Co-Chair

The ethnography examines the different roles of epigenetics in both new scientific thinking and within clinical trials that test nutritional interventions on pregnant women deemed obese. Contrary to commonly held assumptions that underlie genetic determinism, epigenetics represents a paradigmatic shift through the study of how environmental conditions affect gene regulation. Research and prevention efforts around the growing public concern for childhood and adult obesity have recently shifted focus to a new population: pregnant women. Epigenetic science claims that women who are obese during pregnancy have a higher chance of having children who develop obesity and diabetes in adulthood. In the case of obesity during pregnancy, epigenetic researchers worry that a pregnant woman's diet and overall health may affect not only the health of her unborn child, but also the genetic development of future generations. My ethnography follows scientists as they navigate through the new epigenetic paradigm and investigate the effects of dietary interventions on obese pregnant women.

This project involved sixteen months of multi-sited ethnographic fieldwork, carried out between 2012 and 2014. I completed participant observation and interviews at two randomized clinical trials: the SmartStart trial in the United States and the StandUp trial in the United Kingdom. Both trials use epigenetic theories to test the efficacy of prenatal interventions. However, each trial uses different diet and exercise interventions on pregnant women. While the US trial implements a behavioral intervention that focuses on weight gain and calorie control, the UK trial emphasizes the need to control sugar consumption during pregnancy. Both trials also follow up with the participants' children to evaluate the effects of the prenatal interventions on the children's early development. I actively participated in the SmartStart trial in the US as a nutritional counselor and volunteer staff member. I was responsible for delivering the dietary intervention, recruitment, and data collection practices. At the StandUp trial in the UK, I observed twenty-four intervention visits and over twenty different data collection visits, as well as monthly staff meetings and weekly conference calls.

The ethnography juxtaposes the justification and design of the clinical trials on behalf of the staff and scientists, with the experiences of the pregnant participants. By examining the application of dietary prenatal interventions in contemporary science ethnographically, I aim to interrupt a pattern of thinking in which women's bodies are the inevitable targets of intervention to address future epidemics. Throughout the ethnography I examine themes related to how epigenetic science has influenced notions of the environment, "good mothering,"

"healthy eating," and the future risk of disease. In the ethnography I argue that individualizing dietary responsibility solely on the mothers obscures the social, gendered, and environmental factors that also influence their behavior and health. My findings show that epigenetic science is not a homogenous or monolithic paradigm. Rather, different scientists are applying particular aspects of epigenetics, which contribute to what I call the proliferation of epigenetic adaptations.

## Introduction:

## Epigenetic Logics: Scales, Relationality, and Risk

There are more randomized clinical trials (RCTs) testing behavioral interventions of diet and exercise now than ever before in history. In the United States the National Institutes of Health are funding over one hundred RCTs that test dietary interventions on pregnant women (NIH reporter 2014). I examined a trial called SmartStart, which was awarded a ten million dollar grant from the National Institutes of Health. It is one of seven trials in a consortium, in the United States. In the United Kingdom, the StandUp trial had a thirty million pound budget for ten years of research, which was awarded through the European Union and the National Institute of Health Research. The trials that I examined during my field research are part of a larger funding and scientific trend relevant to the new paradigm of epigenetics.

Epigenetics is currently hailed as a "new paradigm" in science. The field is taking shape through the various new journals, departments, disciplines, and studies that focus entirely on epigenetics. Social scientists are also publishing articles that make epigenetics legible and accessible across diverse social and human sciences (Nessa 2012, Stelmach and Nerlich 2015, Niewohner 2015, Darling et al. 2016, Muller and Kenny forthcoming). Excitement about epigenetics as a new paradigm, in part, comes out of the disillusionment with genetics as the "blue print" of life. The epigenetic paradigm marks a shift from genetic determinism to a world in which genes and gene expression are vulnerable to unpredictable change through environmental exposure in pregnancy, early development, and beyond. Epigenetics offers a molecular and epidemiological challenge to the idea that the environment stops at our bodies, and that our bodies have "defensible" and fixed boundaries from the "outside" (Cohen 2009). The emergence of the epigenetic paradigm falls under the "post-genomic era", which biologists

mark as the period following the sequencing of the human genome. Social scientists characterize the new era to include multiple shifts in biology and genetics including epigenetics and the Developmental Origins of Health and Disease (DOHaD) (Stevens and Richardson 2015).

Through epigenetic logics, which I explain below, pregnant bodies are environments and pregnancy is defined as a "critical period", or space-time that is vulnerable to epigenetic modifications. Sarah Richardson uses the term "epigenetic vectors" to describe the maternal body in epigenetic science (2015). However, the medical focus and surveillance on pregnant bodies is a well established and state endorsed project that existed prior to the emergence of epigenetics. In this ethnography I examine how older biopolitical strategies aimed at pregnant bodies converge with new epigenetic framings of pregnant bodies as environments. During my fieldwork I examined how pregnancy has become such an intensified locus for epigenetic knowledge. Encounters with pregnant mothers during my field research challenged me to analyze the complex relational contexts that situate pregnancy as a space for epigenetic knowledge production. The following encounter illustrates the anxiety and individualized responsibility that women have internalized regarding the role of "good" mothering and healthy nutrition in relation to their own bodies and their children's bodies.

"Did I do this to my daughter?"

- Annie, 3/8/16

In March 2016 I gave a presentation at an international workshop in Europe. I discussed the significance of nutrition in epigenetic modification, and I also talked about the importance of context, and the entanglement of environments. On the one hand epigenetic science claims that it

is possible to inherit behaviors and experiences trans-generationally, and that what women eat during pregnancy matters for genetic regulation in the developing fetus. However, I explained that epigenetics science also tells us that modifications in utero are unpredictable and influenced by multiple entangled environmental factors.

After my presentation a woman came up to me to talk about my research. She prefaced her question with a story about her first pregnancy. The woman, whom I call Annie, had a very traumatic first pregnancy. When I asked her why it was traumatic she told me that her doctors kept telling her that she was gaining too much weight and she was so worried. To try and limit her weight gain she walked everyday and watched what she ate. Annie avoided desserts, monitored her weight, and avoided eating too much, but the number on the scale kept going up. Annie felt very helpless and out of control. Her entire pregnancy was marked with fear from gaining too much weight.

At the end of her pregnancy Annie gave birth to a healthy eight-pound baby. Fast forward seventeen years, Annie tells me that her first-born is struggling with her weight. Annie's daughter cannot stop eating sugar, and does not have the taste or desire for anything else. "She just eats sugar" that is how Annie described her daughter. Then she finally asked me the question, "so did I do this to her? Did I make her want sugar because when I was pregnant I was so worried about eating sugar and gaining weight during pregnancy?"

I stood there and looked at Annie who was looking back at me with a worried face. My initial reaction was to say "no," but I knew that would not satisfy her. She wanted me to confirm her suspicion in order to come to terms with the guilt and responsibility she feels with her daughter's current eating habits and desires. I wanted to tell her that her feeling of responsibility and guilt for having "done this to her daughter" is based out of a long historical and political

framework that has influenced Annie's belief that she is responsible for her daughter's eating habits. What I did explain was that the risk and responsibility with regards to developing children and populations relates complexly to race, gender, history, and environments. Moreover, I explained that there is no possible way that she could solely control or be individually responsible for the health of her daughter.

My response to Annie relates to some of the questions that I examine in this ethnography. The focus on what women eat or how much they weigh during pregnancy is not a new topic. Maternal nutrition has a long and complicated and socially specific history. At one point certain foods were associated with having a boy or a girl. Certain food cravings during pregnancy were also thought to explain a child's food preferences later in life. Annie's story is no different than older folklores linking maternal nutrition to seemingly arbitrary outcomes for children. However, after hearing my presentation on epigenetics, Annie thought that this new paradigm could explain how her daughter could have inherited a strong desire for sugar. New ways of understanding pregnancy echo very old ideas about the maternal-fetal relations.

Mothers like Annie and many scientists underestimate the importance of contexts and scales of the environment to health outcomes. Although epigenetic science claims that women who are obese during pregnancy have a higher risk of having children that are obese and diabetic, epigenetics also claims that the risk is contingent and unpredictable. Despite the role of environmental factors that influence health outcomes, the interpretation of epigenetics by Annie as well as scientists in implementing prenatal interventions focuses entirely on pregnant bodies and behaviors.

To understand how pregnant bodies became a site for intervention in the postgenomic era, I examined trials that draw from epigenetics to test nutritional interventions on obese

pregnant women. Research and prevention efforts around the growing public concern for childhood and adult obesity have recently shifted focus to a new population: pregnant women. In the case of obesity during pregnancy, epigenetic researchers worry that a pregnant woman's diet and overall health may affect not only the health of her unborn child, but also the genetic development of future generations. In the ethnography I explore how scientists use epigenetic theories to test dietary interventions on obese pregnant women, in randomized clinical trials.

This project involved sixteen months of multi-sited ethnographic fieldwork, carried out between 2012 and 2014. I completed participant observation and interviews at two randomized clinical trials: the SmartStart trial in the United States and the StandUp trial in the United Kingdom. Both trials use epigenetic theories to test the efficacy of prenatal interventions. However, each trial uses different diet and exercise interventions on pregnant women. While the US trial implements a behavioral intervention that focuses on weight gain and calorie control, the UK trial emphasizes the need to control sugar consumption during pregnancy. Both trials also follow up with the participants' children to evaluate the effects of the prenatal interventions on the children's early development.

Although the scientists that I study focus on obesity during pregnancy as their broader research objective, I approach obesity during pregnancy as the background context to index the ways in which new epigenetic approaches are emerging within an existing techno-scientific environment. In Chapter One I trace obesity during pregnancy and the different national policies that exist in the United States and the United Kingdom to show how scientific theories and methods change over time, and have specifically changed as a result of epigenetics. By following the different ways in which scientists approach maternal nutrition and obesity, I show how the application of RCTs to test nutritional interventions during pregnancy is an emergent

phenomenon that developed in the past two decades. By doing so I argue that the scientific questions and methods used to produce epigenetic knowledge are using older and gendered notions of motherhood and pregnancy to design and test prenatal interventions. Consequently, I find that the questions and methods examining epigenetics are not aligned with the theories implicit to epigenetics.

The ethnography offers a few key interventions. Throughout the ethnography I argue that RCTs are not the appropriate tool for understanding epigenetics. I base this claim on the same theories that undergird epigenetics. Epigenetic modifications are unpredictable and indeterminate; therefore, a method aimed at finding causality is unsuitable for examining epigenetics. Moreover, epigenetic logics imply entanglements across scales of the environment and time-space. The linear and anticipatory temporalities that frame the RCT methods are not appropriate for understanding the relationality across environments and the transversality of time, all of which I explain below. In addition, I align myself with other scholars that bring attention to the gender biases and reductionist approaches that undergird epigenetic research (Landecker 2013, Lock 2013, Richardson 2015). I argue that targeting individual women's bodies for intervention undermines the potential of epigenetics and reproduces older forms of biopolitical surveillance. Finally, the ethnography offers an epistemological intervention that emphasizes the implications of disciplinary expertise in epigenetic science. I argue that the epigenetic paradigm is not homogenous. Rather, the different disciplinary approaches and expertise that frame study designs reflect a selective application of epigenetic theories and produce what I call epigenetic adaptations. In concluding, I offer alternative ways to frame pregnant bodies that prioritizes a relational approach to environments, inheritance, and risk.

**Prenatal interventions: surveillance, management, and disciplining**

Prenatal interventions are founded upon the biopolitical assumption that women's bodies should be disciplined, controlled, and managed. Broadly conceived, prenatal interventions include a range of reproductive practices, from restricted access to abortion, forced sterilization, birth control experimentation, sexual violence, (Briggs 2003; Lopez 2008; Petchesky 1985; Smith 2005). Such examples exemplify what Rapp and Ginsburg describe as "the intricate national and international connections among the rise of medical professions and industries, global markets in labor and pharmaceuticals, and ideologies and policies that explicitly link economic development to population control" (1991: 333). Therefore, prenatal interventions are already situated within larger systems of power and control that implement strategies of surveillance, management, and discipline on women's reproductive bodies (Foucault 1977, 1978, Donzelot 1979).

The scholarship on prenatal interventions is extensive and reflects a wide variety of contexts, technologies, and justifications and highlights the significant stakes involved in producing an assemblage of actors, methods, and networks to design and execute reproductive control. For example, Chinese women's bodies are sites of intervention and implementation of the One Child policy, a state policy also used to manage the size of the population body as a whole (Greenhalgh 2008). Controlling pregnancy through various forms of policies reflects the entanglement of individual and national scales, as well as the social and biological relations that justified the economically motivated policy. Scholars like Greenhalgh draw from Foucauldian theories of biopolitics and biopower to show how reproductive health and prenatal interventions are directly tied to national projects.

Foucauldian theory provides a useful lens for understanding prenatal interventions on pregnant bodies for many reasons. Foucault argues that as a result of a fundamental shift in nation-states and war during the 17th century, individual bodies became a key target for the maintenance of the nation. Individual and aggregate bodies became important for protecting and defining the state, nation, or population. Further, populations needed to be managed to maintain order and peace. In Foucault's lectures on *Security, Territory, and Population* he describes biopolitics as, "this very specific, albeit very complex, power that has the population as its target, political economy as its major form of knowledge and apparatuses of security [or dipositifs] as its essential technical instrument" (1978:108). Therefore, keeping individual and aggregate bodies healthy became crucial for the production of labor and the maintenance of the military. Biopower is also intimately connected to biopolitics. Biopolitics attempts to "optimize the life of the population as a whole", by focusing on the processes related to reproduction, health, and productivity (Inda 2005:5).

Another key aspect of prenatal interventions is the invasive justification of acting upon women's bodies. Foucault's idea of biopower is founded upon the regulation and systematic intervention on individual bodies (Foucault 1979:139). To maintain control over bodies, Foucault and others outline ways that power can be asserted over individual or docile bodies. Docile bodies are made through practices and processes of manipulation and surveillance. Docile bodies are also disciplined bodies that behave according to the structures of power. Bartky explains that "the production of 'docile bodies' requires that an uninterrupted coercion be directed to the very processes of bodily […] time, its space, and its movements" (63:1990). Producing docile bodies also occurs through the process of disciplining and defining proper hygiene, work ethic, breast-feeding, and child rearing behaviors. Moreover, biopower and

biopolitics constitutes the "localized routinized bodily practices in families, communities and institution" (Lock and Kaufert 139:1998).

In Foucault's later works he describes the family as a key target of surveillance and control by the state in order to ensure that children live long enough to reach adulthood.

> The family is no longer to be just a system of relations inscribed in a social status, a kinship system, a mechanism for the transmission of property. It is to become a dense, saturated, permanent, continuous, physical environment, which envelops, maintains and develops the child's body. Hence it assumes a material figure defined within a narrower compass; it organizes itself as the child's immediate environment, tending increasingly to become its basic framework for survival and growth (172-173:1980).

Foucault builds upon this idea of the "family" through various primary medical texts that explain how women – not men, grandparents, cousins, or other community members – should care for the children (1980). Foucault's description places the family as a focal point, but the actual interventions and education campaigns that he provides as evidence are focused entirely on women's behavior and child rearing practices. By describing the family as a "dense, saturated, permanent, continuous, physical environment, which envelops, maintains and develops the child's body", Foucault, perhaps unknowingly, illustrated a womb.[1] The metaphorical deployment of the womb or pregnant body as an institutional environment reflects how population health is literally and figuratively inscribed onto women's pregnant bodies. Foucault's description of the "family" indexes the ways in which women's bodies in particular have already been framed as the target site for intervention, long before the epigenetic paradigm began taking hold.

The justification to intervene on women's bodies in the name of population health or public health is not novel. Pregnancy has long been a site of medical intervention to ensure

[1] This conceptual thread is the basis of what I call epigenetic-politics, which points to the expanding forms of reproductive surveillance not just on bodies but on environmental scales.

healthy births and to control and direct women's reproductive capacities. Public health campaigns from the nineteenth and twentieth century targeted poor women to reduce fertility rates (Rafter 1992) and prescribed a 'domestic science' of house cleaning for germ prevention (Tommes 1999). Later campaigns focused on behaviors related to alcohol and tobacco consumption (Graham 1990) and breast-feeding (Schiebinger 2003). Not only are women's bodies sites for prenatal interventions, but there is an unequal focus on brown and black bodies in state and colonial biopolitical projects (Roberts 1997, Bridges 2011).[2] Most recently, the WHO proposed that any woman considering pregnancy should stop drinking alcohol months before conception. The persistence strategies of surveillance and monitoring are examples of bodily surveillance, control, and management, all of which characterize the biopolitics of governmentality (Foucault 1978, Cohen 2009, Thacker 2005, Clough and Willse 2011).

However a feminist approach highlights how women's bodies are made available for intervention. Anna Devin, a historian, argues that forms of surveillance focused not only on the family as a social institution but also on individual women--in the 18th and 19th centuries England (1978). Davin writes,

> "[I]f the survival of infants and the health of children was in question, it must be the fault of the mothers, and if the nation needed *healthy future citizens* (and soldiers and workers) then mothers must improve.[…] Thus the solution to a national problem of public health and of politics was looked for in terms of individuals, of a particular role – the mother[…]. This obscured to an extent

[2] In Dorothy Roberts' book *Killing the Black Body* she argues that Black women's reproduction in particular has a long and extensive history of surveillance, control, and management. She draws on specific examples during times of slavery, and also more recent strategies of reproductive control in the late 1980s. During the time of the "crack" epidemic and the war on drugs, South Carolina decided to arrest pregnant women who tested positive for smoking crack. Roberts' analysis shows that all but four women arrested were Black.
Khiara Bridges book *Reproducing race: an ethnography of pregnancy as a site of racialization* examines the ways in which prenatal nutritional interventions are politically and racially charged (2011). Bridges explores how poor women covered under Medicaid/PCAP in New York are surveyed and managed through prenatal care services. Women receiving Medicaid are required to eat specific foods, gain specific amounts of weight, complete regular physical examinations, submit information about their partners' citizenship status, and share extensive personal and medical history (Bridges 2011).

> which now seems astonishing the effects on child health of poverty and environment." (Davin1978:12 my emphasis*)*

Davin highlights that the justification to survey women's bodies has a long history. Where Foucault fails to highlight the significance of raced and gendered bodies in processes of biopower and biopolitics, feminist scholars provide a critical expansion on the topic. For instance, feminist literature shows how the 'microphysics of power' does not capture the complicated relationship between bodies, docility, resistance and oppression (Boddy 1998). Likewise, Sawicki comments that within a context of biopower the body is neither active nor passive, it is not autonomous or enslaved (Sawicki 1991). In addition, the complexity of oppression and resistance is illustrated in Barbara Bush's work on how Caribbean slave women would use their bodies as 'political weapons' to resist the oppressive structures of slavery (1993). According to Bush, "these women developed a politics of resistance by quietly killing the infants in their wombs by means of well-known plant poisons" (11:1993). By terminating their pregnancies, enslaved women limited the production of 'man-power' that fueled the colonies (Bush, 1993). Bush's work relates to the ways in which women knew their bodies were used to socially and biologically reproduce labor. The feminist emphasis on how biopolitical strategies have primarily focused on brown and black female bodies or bodies that lie outside the platonic ideal, reflects the role of larger systems of control and power. The persistence and ubiquitous use of prenatal interventions in medicine and research is part of a larger system of biopolitics and power.

The same goals of ensuring the health of future citizens, or the next generations are motivating contemporary dietary experiments on pregnant bodies. The growing interest in targeting pregnancy for diet and exercise interventions reiterates older forms of surveillance and

management, but under a "new" epigenetic paradigm. My research builds on feminist literatures to show how pregnant bodies are made available for new forms of intervention, and specifically examines how epigenetics frames pregnant bodies *as* environmental sites for intervention. The randomized clinical trials that I examined are also forms of prenatal interventions that are informed by biopolitics, race, and gender. Drawing from epigenetic logics I argue that if taken seriously the environment does not stop at women's bodies, but rather, women's bodies are also porously and continuously entangled with larger political, social, and culture environments. Further, the implications of centering population health approaches on women's bodies and abilities to biologically reproduce reifies the distinctions between social and biological reproduction, between sex and gender, and between nature and culture. Instead of addressing structural factors related to the industrialization of food production, water politics, and the social phenomena of food deserts, the trials that I examined focus large quantities of time and money to target pregnant bodies for nutritional interventions. In general prenatal interventions are broad and diverse projects involving biopolitical forces. As a result of particular interpretations and applications of epigenetics prenatal dietary experiments are expanding.

In what follows I trace the intellectual genealogies relevant to epigenetics and the limits and possibilities of epigenetic theories. Then, I explain my analytical framing of the ethnography. I also include a critical perspective on the history of the RCT method, which is the primary method used to examine epigenetics in prenatal interventions. Finally, I explain my methodology and end the introduction with a summary of each chapter.

**Situating Epigenetics**

*Intellectual Genealogy and Orientations*

The emergence of epigenetics is intimately related to genetics. The Greek prefix "epi" literally means "on top" or "above" genetics. Conrad Waddinton, a British scientist, coined the term epigenetics in the 1940s to unite the fields of developmental biology and genetics; two fields that had previously been studied as separate disciplines during the nineteenth century (Szyf 2009, Holliday 2006, Hurd 2010). Waddington explored developmental biology and genetics with the intention to understand how the same genotype or the same genetic information could produce different phenotypes or physiological manifestations.

The motivation to unite the fields of developmental biology and genetics came out of the much older debates on epigenesis and preformation dating back to Aristotle. Epigenesis explained how an embryo gradually became more complex through repetitive cell division. Preformation, on the other hand, proposed that an embryo is a fully formed individual at all stages of development.[3] The debate between preformation and epigenesis shifted with the invention of advanced microscopes, which provided visual evidence that an embryo was not a fully formed individual.[4]

In a letter to the journal *Nature*, Waddington explains the significance and derivation of the term epigenetics in relation to the debate on epigenesis and preformation:

[3] Aristotle argued that organic "form" comes from something that was "unformed" before it, which is guided by "vital cause," which Hurd cites in his article titled "Briefings in functional genomics" (2010: 425-428).

[4] Microscopes may have produced a new way of seeing embryos but historians of embryology highlight that preformation ideas still undergird current debates on the classification of embryos as living entities. Classifying material as preformed and living is related to notions of fetal personhood and the struggle between fetal/maternal rights (Maienshein 2012). Microscopes and sonograms are forms of technology that produce "modern science" (Haraway 1991). These technologies have significant consequences for the "personification of fetuses and the effacement of the 'maternal vessel' within which its gestational life is lived […]"(Rapp 1997:32). The role fetal-maternal relationship is also (re)configured through the logics of epigenetics, and the preformation/epigenesist debate is part of that history.

> The study of the 'preformed' character nowadays belongs to the discipline known as genetics; […] Admittedly the word 'genetics', which was coined by Bateson to cover the 'the physiology of descent', might have been used so as to embrace both [biological and genetic] development; but in practice it has not been widely employed in [the genetic] sense" (Waddinton1956:1241).

The emphasis that Waddington places on epigenesis and its interaction with "preformed characters" is the connection between genetics and developmental biology. Therefore, Waddington argued that epigenetics is the study of the interaction of the "preformed characters" or genes and epigenesis. Waddington's work is significant because it established a framework through the term 'epigenetics' to examine the connection between genetic and biological aspects of development.

Waddington turned to the notion of acquired characteristics, or genetic assimilation, to illuminate epigenetic processes. He explained that genetic assimilation occurs as a result of complex genetic interactions during development, and that this process is flexible. His publications on the topic of genetic assimilation explored developmental modifications, environmental stress, and inheritance (Waddington 1959, 1961). For instance, Waddington claimed that an organism could assimilate or rather "remember" or embody environmental stress that occurred in past generations.

The notion of assimilation or acquired characteristics is similar to an older concept of biological development credited to Jean-Baptist Lamarck (1744-1829). Lamarck argued that the environment played a role in physiological and anatomical development. A common example of Lamarckism is the length of a giraffe's neck. He claimed that a giraffe acquired a long neck by stretching and stretching to reach leaves on trees. After a few generations of giraffes stretching their necks, a new generation of giraffes would have longer necks. Lamarck published his

findings in the early 1800s, which are contributed to an understanding of "soft inheritance." Soft inheritance is defined as the inheritance of acquired characteristics.

In the late 1880s another theory of inheritance emerged, and was developed by Alfred Wallace and Charles Darwin. In his 1871 book Darwin proposed his theory of evolution, which specified that changes in species occurred through the process of "descent with modification" (1981). Interpreters of Darwin's theory focused on how natural selection played a significant role in determining the survival of the species. Darwinian theories prevailed, and Lamarkism dissolved as a legitimate theory. However, there were still some limitations to the Darwinian theories of inheritance, namely the concept of the gene.

Around the same time that Darwin was publishing Gregor Mendal established his theory of genetic inheritance. Through pea plant models, Mendel developed our modern understanding of how certain genes are inherited from one generation to the next. In addition, Mendelian inheritance was fundamental to developing the field of genetics. In the genetic paradigm genes were understood as fixed forms of information. "Hard inheritance" was defined as the inheritance of genetic material from only your mother and father (Meloni 2014).

Some scholars comment that the age of epigenetics marks a revival of Lamarckism. In part this revival is due to the epigenetic focus on the environment (Lock 2011). The environment played a key role in Lamarck's understanding of biological development. Likewise, for Waddington, the environment is significant to biological and genetic development. Therefore, in contemporary epigenetic paradigm the environment is also featured in stimulating genetic modifications or epigenetic changes to genes. Epigenetics defines the environment as exposure from inside the body like "the cytosol around the nucleus, the tissue milieu of the cell, the uterine environment of the fetus, the gut microbiota - or the outside - some element of the macro

environment" (Landecker and Panofsky 2013: 339). In the new epigenetic paradigm, cells, tissues, uteruses, guts, and the "outside" or natural built environment represent different scales of the environment. Moreover, food or hormones can act as environmental signals to cells and these changes are inheritable, but not in the same way that genetic information is inherited (Landecker 2009). The epigenetic definition of the environment looks very different than the genetic paradigm.

The disenchantment with genetics opened up a space for the emergence of the epigenetic paradigm.[5] By the twentieth century, genetics emerged as a significant field of science marked by hope. Once the human DNA sequence was mapped 1999, people were surprised by the results. The Human Genome Project concluded that humans have far less genes than originally estimated, and only a fraction of the genome consists of genes that are used to make proteins (Barnes and Dupré 2008). The results from research on genetics in the twentieth century challenged the notion of Mendelian inheritance. Mendelian inheritance claims that discrete genes are passed from one generation to the next in a pattern influenced by chance and probability. Experimental studies that test environmental signals on plants, animals, and humans confirm that genetic modifications play a significant role in gene expression and inheritance. Therefore, genes

[5] The international interest and exploration of epigenetics and epigenomics, which includes both the genome and the cellular levels of epigenetics, is reflective of a paradigm shift in the Kuhnian sense. In *Structures of Scientific Revolution*, Kuhn examined historical records to argue that scientific discoveries are not isolated or distinct events credited to individuals but part of a larger process and development of knowledge production. Paradigms have two main characteristics: first, they must be unprecedented to attract people towards it and away from competing notions; second, the achievement must also be open ended enough to allow the 'profession' to solve problems (Kuhn 1962:11). Additionally, Kuhn discusses the issue of incommensurability by stating that "differences between paradigms are both necessary and irreconcilable" (1962:103). New paradigms also offer different views on nature, and in the case of epigenetics ontological understandings of being. I highlight three ways in which epigenetics represents a Kuhnian paradigm shift. First, epigenetics as a paradigm is defined as different from and incommensurable to the genetics paradigm. Second, epigenetics is still an emerging field open for exploration. Third, as scientific revolution, it is changing ontological and epistemological worldviews. By ontological and epistemological I mean that epigenetics is changing the ways in which we understand, for instance, our relationship with our bodies and past generations, and the potential for understanding disease manifestation across generations. Another way of framing this is by looking at Callon's concept of obligatory points of passage (1986). Susan L. Star's work on ecologies of knowledge is also another example (1995).

that are past down from one generation to the next do not account for most of the phenotypic variation.

The new paradigm of epigenetics is marked not by the distinction between phenotype and genotype but by the distinction between genetics and epigenetics. One of the key distinctions between genetics and epigenetics is inheritance. As I mentioned above, epigenetic modifications stimulated from the environment are inherited in a particular way. Whereas, genetics is defined by Mendelian inheritance, epigenetics is defined as non-Mendelian inheritance. Szyf explains, "it was generally believed that most of the epigenetic information is erased during early gestation, and if this erasure were complete then errors in the epigenetic markings would not be transferred across generations" (2009:9).[6] He goes on to say that due to unpredictable modifications, or stochasticism, on parts of the DNA it is possible that these changes can actually be inherited across generations and be latently triggered later in life (Szyf 2009). In another definition, Waterland and colleagues write that "rather than heritable change in gene expression epigenetics encompasses heritable changes in gene expression *potential*" (2007:366 my emphasis). Framing heritability through the concept of "potential" highlights the flexibility, unpredictability, and malleability of biological and genetic development. The new forms of heritability and malleability of genetic develop characterize the post-genomic era, a broad title that includes epigenetics.

The potentiality of non-Mendelian inheritance implies a new kind of relationality between and across generations, environments, bodies, and cells. Niewohner describes this relationship through his definition of environmental epigenetics. He writes that environmental epigenetics asks, "how changes in the social and material environment have a physiological

[6] Also, Paul Hurd writes, "The inherited preformed or predetermined genetic program provides information about what is possible, but regulation of genetic expression involves interpretation. It is the latter that is epigenetic" (2010:425)

impact on individuals and on forms of sociality" (Niewohner 2011: 284). Through epigenetics the environment is an entanglement of different scales of the environment. As Van Speybroaeck notes, environmental epigenetics reveals a hierarchy of different levels of context (2000).

Another key difference between genetics and epigenetics relates to the ways in which causal pathways are connected between genotypes, phenotypes, and disease manifestation. Genetic determinism prioritizes genetic information and difference to explain the increased rates of obesity or diabetes in particular ethnic or racial groups (Montoya 2011). Instead, epigenetics explores how socioeconomic status and social conditions might be a predictor of chronic disease and low birth weight.[7] For example, the concept of the "thrifty phenotype" (as opposed to the thrifty genotype) offers a way to incorporate the social, political, and environmental concerns that influence development in early life and the lasting effects into adult hood. The "thrifty phenotype hypothesis" claims that if the fetus is in a nutrient deficient environment, its development will react in a "thrifty" way to prepare for the "postnatal environment", which could result in neo-natal fat storage (Hales and Barker 1992). Understanding the causal mechanisms for how social conditions can signal genetic modification lies at the boundaries of epigenetic knowledge.

The development of epigenetic theories for understanding the inheritance of disease manifestations across different generations and across the life span came out of observational studies on the effects of famine or under-nutrition across different generations.[8] During the late

[7] There is also a contrasts between genetic determinism and epigenetic logics reflected in the differences between the "thrifty genotype" and the "thrifty phenotype" hypotheses. The "thrifty genotype hypothesis" is way to reify racial categories as biological and deny the material and biological effects of social, cultural, and historical inequalities (2011). Further "there is no evidence that the differential rates of diabetes across global populations are a result of a thrifty genotype" (Montoya 2011: 160).

[8] In developing this hypothesis Barker explored retrospective observational studies on women from Hertfordshire, United Kingdom. In the records Barker found that babies with low birth weight have a higher risk of adult cardiovascular heart disease (CHD). These findings are corroborated with many other retrospective studies as well. Barker also looked at different population records in India and in the Netherlands. For instance, with the "Dutch

1980s and early 1990s, British scientist David Barker published a series of articles that are largely credited for developing the fetal origins hypothesis and the notions of fetal programming and latency. Fetal programming is the idea that the epigenome can be programmed in the uterine environment. The epigenome includes the chemical changes to the genetic sequence. The fetal origins hypothesis proposes that fetal programming during pregnancy can permanently impact the metabolic development of the offspring (Barker 1992). Latency refers to the ways in which the fetal programming can have permanent effects that may or may not be triggered later in life (Hales and Barker 1992 and 2001). Although, other accounts of the emergence of epigenetics do not highlight the work by Barker, as a result of earlier published research on latent health effects, I incorporate Barker's work because the principle investigator in the UK trial references Barker's work on the fetal origins hypothesis in her speeches and discussions of her research.

Barker's work along with other scientific research on nutrition during pregnancy and famine studies helped develop the study of developmental origins of health and disease (DOHaD).[9] DOHaD claims that the, "maternal diet and nutrition status during pregnancy also predict risk for [cardiovascular risk, blood pressure, insulin resistance, type 2 diabetes, and a tendency to deposit fat in the central, metabolically active fat deposit]" (Kuzawa et al. 2009: 133). In addition, the DOHaD theory explains that epigenetic modifications during pregnancy can manifest into metabolic disorders later in life.[10] The DOHaD is a theory within the

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hunger Winter group in Amsterdam", he showed that exposure of pregnant mothers to famine left a legacy of chronic disease in their children" (Barker et al., 2013:1).

[9] One of the foundational famine studies is a longitudinal study from Sweden. The Swedish scientists found different relationship between famine and chronic disease across generations. The study analyzed over one hundred years of records that contained detailed medical information of all the residents in one town. The retrospective study found that grandparents' exposure to hunger or famines could influence the cardiovascular risk in their grandchildren (Bygren et al., 2014**).** The Scandinavian cohort study is often cited as support for the presence of inherited epigenetic modifications due to environmental stimuli in past generations.

[10] At the molecular scale, epigenetics is characterized as "those processes that ensure the inheritance of variation (-genetics) above and beyond (epi-) changes in the DNA sequence" (Bonasio et al., 2010:612). The molecules responsible for changing, altering or modifying the genetic sequence during translation are methyl groups (one

epigenetic paradigm that draws an explicit connection between women's diets during pregnancy and health risks in future generations.

The focus on pregnant women is a result of both non-Mendelian inheritance and the notion of a critical period in epigenetics. Epigenetic science focuses on specific times and spaces that are most vulnerable to epigenetic changes. A space-time in which epigenetic modifications can occur are called critical periods. A critical period is defined as, "a time in an organism's development when environmental input strongly influences a morphological or behavioral outcome; these same input before or after the critical period has little effect" (Landecker and Panofsky 2013: 340). Waterland and colleagues note that the embryonic state is full of potential - it is totipotent or multi-potent (Landecker and Panofsky 2013: 340). For instance, if an embryo develops into a fetus that has ovaries the potential for epigenetic modifications is extended across three generations. The embryonic stage is one example of a critical period that takes place in the uterine environment during gestation.

The embryonic stage is a critical period because of fetal programming. Fetal programming refers to the changes in the genetic sequence that can occur in the uterine environment during pregnancy. Epigenetic modifications during fetal programming are stimulated from the uterine environment, pregnant body and behaviors, and the natural built environment. As a result of non-Mendelian inheritance these changes during pregnancy (although not proven in humans, only in animal studies) can cause chronic disease in the development child, adult, and across generations.

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carbon atom with four hydrogen atoms). Attaching or detaching methyl groups to certain parts of the DNA or genetic sequence can "silence" or inhibit parts or activate other parts, which make up the epigenome. Moreover, "epigenetic signals are responsible […] for the cell's ability to **"***remember* past events, such as changes in the external environment or developmental cues" (Bonasio et al., 2010:612, my emphasis).

A key question in the field of epigenetics today is whether epigenetic changes "during gestation or later in life are stochastic or programmed" (Szyf 2009: 11). The term stochastic is defined as "interactions that follow a random probability distribution that can be analyzed statistically but not predicted precisely" (Landecker and Panofsky 2013: 339). Stochasticism is the unpredictable way that epigenetic changes can occur during critical periods. This area of epigenetics lies at the borders of scientific consensus.[11] For now, however, stochasticism still applies as a way to explain epigenetic processes. Assuming stochasticism is relevant and significant to epigentics, the temporality that it implies is not linear or determinate. Landecker and Panofsky highlight that, "stochasticism - the idea that many biochemical processes […] are inherently unpredictable and nondeterministic - is an essential *but often overlooked* caveat in discussing cause and effect in epigenetics" (2013:339 my emphasis).[12] Therefore, epigenetic science suggests that changes to DNA sequence can be unpredictably inherited across different generations and can occur outside of the womb/body/uterine environment. From my ethnographic research, the infusion of epigenetic theories within evidence-based medicine often overlooks the issue that epigenetic modifications are inherently unpredictable and inherited in unpredictable ways.

The unpredictability of non-Mendelian inheritances challenges causal or predictive links from one environmental exposure at any given time or space to future disease. Scientists are also pointing to early development outside of the uterine environment, as a critical period that can change biological and genetic development throughout the lifespan (Weaver et al., 2006). In

[11] No doubt soon the suspense of whether epigenetic changes are stochastic will be resolved, since dwelling in the uncertainty is not a techno-scientific priority).

[12] The unpredictability and non-determinate temporality inherent to stochasticism is similar to the ways in which Nietzshe, Bergson, Deleuze, and Irigaray frame temporality as open, becoming, uncertain, unpredictable, and non-linear (Grosz 2005). Elizabeth Grosz's book *The Nick of Time,* argues for the ontology of a temporality that does not depend on the past to determine or produce a certain future (2005). She emphasizes that the potentiality to diverge and produce unpredictable futures is immanent in our past and present, which is in line with the temporality implied in the logics of epigenetics. See also Heidegger, Derrida, and Harvey.

addition, more research is emerging that if epigenetics modifications are already happening at the embryonic stage, then interventions to prevent adverse modifications in utero should target women before they even get pregnant. The last point implies an expansion of the "critical period" to include a women's entire lifespan. By this logic, the justification for interventions on women's bodies and behavior could occur earlier and earlier.

*Limits and Possibilities of Epigenetics*

My work informs and is informed by an existing literature that highlights the epistemological and ontological limits and possibilities of epigenetic science.[13] Here I will discuss a few different examples. In the article titled "The Epigenome and Nature/Nurture reunification: A Challenge for Anthropology", Lock writes that the "[epigenome] has the potential to incite new forms of reductionism that may well result in inappropriate moral attributions, stigmatization, and discrimination, largely because social and economic variables external to the body are, from the outset, set to one side" (2011: 296). She warns that the prioritization of biomarkers in Alzheimer research reflects a reductive approach in epigenetic science that elides other cultural, historical, and political factors that facilitate the diagnosing and treatment of Alzheimer (2011)[14]. Lock's earlier work on local biologies also proves useful to think with in the epigenetic paradigm (1993). Her focus on how health and disease is embodied in local contexts directly maps on to epigenetic explanations of socio-cultural marks of genetic expression. However, Lock brings attention to the fact that although epigenetics offers a way to understand how social and historical inequalities can leave material traces on the body, the

[13] I also engage with the science studies literature that highlights the potential of epigenetics as a subject and object of scientific inquiry that can facilitate a cross-disciplinary move beyond the nature/nurture divide (Nessa 2012; Landecker and Panofsky 2013; Stelmach and Nerlich 2015; Niewohner 2015).

[14] See also: Lock, M. 2013. The Alzheimer Conundrum: entanglements of dementia and aging. Princeton University Press. Princeton, New Jersey.

research does not necessarily focus on the political, structural, and historical systems that perpetuate inequality.

Drawing from Lock, Meloni's work also critically examines the possibilities and limitations of epigenetics. Meloni argues that epigenetics has the potential to dissolve disciplinary boundaries between biological and socio-cultural approaches to health and disease (Meloni 2014). However, he warns that using epigenetics could also lead to the reclassification of social classes. In his book *Political Biology: Science and Social Values in Human Heredity from Eugenics to Epigenetics* Meloni traces scientific understandings of heredity along with political movements that focus on the universality of human nature (2015). Meloni shows how scientific theories of heredity reflect human values, and in turn how historical and social contexts influence paradigmatic shifts in science.

Meloni's concern with epigenetics is directly related to Flipchencko's warnings from the 1930s. In the Russian context, Flipchencko, a Soviet geneticist, denounced Lamarckism as a legitimate theory of inheritance because of the underlying political implications (Graham 1978). Flipchencko believed that the theory of acquired characteristics could be used to justify the divisions of social classes between elites and the proletariat. If taken seriously Lamarckism could be used as a way to justify the stratification of society based on different environmental or social contexts. For Flipchenko, heredity through acquired characteristics implied that the elite class was also biologically and genetically different because they had inherited different social characteristics.[15] Meloni claims that through epigenetics we can now examine questions like "do the poor have different patterns of methylation than the rich?" (Meloni 2014: 2). However, he warns that these kinds of research questions reify social differences across socio-economic classes, which results in the essentialization of poverty through inheritance. The potential of

[15] Flipchenko's position was rejected by the Lysenkoist USSR (Graham 1978).

classifying poor groups of people into "risky" populations is already manifesting in current research. From a different perspective, Maderspacher a prominent biologist published a comment in *Current Biology* claiming that the heightened attention around the context-based inheritance in epigenetics is directly related to liberal politics that characterize our contemporary climate (2010).

At a conference I attended on "Big Data" in 2016, a social scientist from the UK presented research on how big data could be used to address social inequalities. The UK researcher introduced the concept of "community vital signs," which uses patients' addresses to compile information on neighborhood environments and social context. For instance, doctors could use a program that identified patients' neighborhoods and provides information on the number of parks, grocery stories, fast food chains, or crime rates. Drawing from this information the primary care physician could customize her message on healthy eating and activity to different patients. Alternatively, primary care doctors could use the data to identify "at risk" populations and potentially spend more time with them.

The response to the presentation was mixed. As a result of epigenetic influences in big data approaches to healthcare, the social context of individuals' are now framed as directly related to health risk and outcomes. Therefore, concepts like "community vital signs" are framed as important to health. On the one hand the epigenetic paradigm emphasizes how important environmental conditions are for peoples' health. However, on the other hand the emphasis on neighborhoods, addresses, and social context could produce unintended stigma. One audience member, also from the UK, stated that associating individual's neighborhood data with their medical records was appalling and could stigmatize poor people. In the UK context, socio-economic stratification has a long and complicated history where addresses come to represent

more than just "healthy" or "unhealthy" environments. For instance, assuming that particular health risks are inherently a part of particular neighborhoods can perpetuate social biases. However, it could also open up the different ways in which social context is incorporated into primary care. The presentation of "community vital signs" in a post-genomic era reflects the limits and possibilities of using epigenetics in approaching health. Moreover, it is an example of how different interpretations, adaptions, and applications of epigenetics will produce unpredictable consequences.

In this ethnography, I argue that the exponential growth of studies that focus on pregnancy and early development in the past decade is related to the growing emphasis on "critical periods" derived from the epigenetic paradigm. Therefore, the clinical trials that I examine are testing interventions of diet and exercise on obese pregnant women, and are following up with the born children. The trials hypothesize that changing diets during pregnancy can reduce the risk of pregnancy complications and the risk of obesity and diabetes in the developing children. However, there are a few caveats to consider when applying epigenetic theories in clinical trials. One aspect that is often overlooked is that intervening on pregnant women in the present does not consider the possibility that the embryo or fetus might have already inherited epigenetic changes from a previous generation. Epigenetic modifications to genetic sequences are understood by scientists to be stochastic, inherently unpredictable, and nonlinear. Therefore, RCTs aimed at finding causal relationships are potentially inadequate in understanding epigenetic effects of prenatal interventions.

**Epigenetic Logics**

In this ethnography, I use the concept *epigenetic logics* to highlight how epigenetics is not just a mechanistic discovery of genetic modification and inheritance. Rather, I demonstrate that epigenetics implies a way of thinking, framing, and knowing the world. Epigenetic logic refers to the epistemological and ontological implications of knowledge production and practice. Logic, according to the Oxford English Dictionary (*OED*), pertains to reason, conduct, and practices that are made or assessed based on principles of validity. Institutional logics refers to the ways in which society, history, and culture influences practices, values and belief, all of which contribute to the creation of social worlds (Thornton and Ocasio 1999). Authors Alford and Friedland first described institutional logics in order to illustrate the ways in which capitalism, bureaucracy, and political democracy influenced social order and reality (1985).

I use logics to refer to the guiding principles that are inherent in epigenetic knowledge. By doing so, I highlight how scientific knowledge production also shapes social realities and provides guiding principles. The ethnography examines the different logics that are inherent in the practice and examination of epigenetics, and also reflective of larger principles that carry implications for knowing, being, and thinking. In what follows I situate three kinds of epigenetic logics: environment, inheritance, and risk. My analysis emphasizes how, for instance, the epigenetic understanding of the environment is a kind of scalar project and why scales of the environment are essential to epigenetics. Additionally, I illustrate how epigenetics has fundamentally changed understandings of "soft" and "hard" ideas about inheritance. By doing so, I argue that epigenetic inheritance represents new forms of relationality across time-space and nature-nurture. Finally, I explore how epigenetics frames and underscores the "transversality" of risk, a concept on which I elaborate below.

The description and explanation of the epigenetic logics comes from archival review of scientific understandings of epigenetics as well as from my ethnographic research. I explored how scientists apply and approach epigenetics in prenatal interventions. The experimentation of prenatal interventions in clinical trials offers a space to examine scientific knowledge production and epigenetic logics that are co-constructed through the processes of practice and experimentation. Further, I found that the different disciplines that draw on epigenetics mobilize new kinds of connections across different networks of science. Bringing together odd bedfellows, like physiologists and psychologists, to implement behavioral interventions on pregnant women results in what I call the "proliferation of epigenetic adaptations." Different scientists take on and integrate different aspects of epigenetics, and the disciplinary influence of the scientists that design each clinical trial and shape the way epigenetics is applied and understood.

Throughout the ethnography I argue that, if taken seriously, epigenetics can offer a new way of approaching scientific research questions, methods, and health policy. However, I show that the scientific methods have not comprehensively incorporated the logics that are inherent to epigenetics. Together, the conceptual understanding of epigenetics and the practices of epigenetics produce possible ways of opening up and limiting epistemological and ontological approaches to health and disease. One of the aims of the ethnography is to illustrate the different manifestations and adaptations that shape the emerging field of epigenetics, as well as provide new ways of using epigenetics to guide future research and policy questions. In addition, I trace how epigenetics has changed approaches to prenatal testing in the context of obesity during pregnancy and randomized clinical trials.

Although have I have categorized epigenetic environment, inheritance, and risk and into distinct categories, the implication is not that these logics work in discrete ways, nor are they the only ones that exist. On the contrary, the epigenetic logics that I examine in the ethnography are intimately entangled. For instance, while Chapter Two focuses on illustrating the significance of scale at one trial site, Chapter Three focuses on the relationality across different scales of the environment. The distinction between each chapter relates to the ways in which different scientists apply and practice epigenetic logics in particular ways. In addition, the logic of risk is connected to both environmental scale and relationality.

*Epigenetic Environment: a scalar approach*

The definition and framing of the environment has fundamentally changed as a result of epigenetics. For instance, epigenetics defines the environment as exposure from inside the body like "the cytosol around the nucleus, the tissue milieu of the cell, the uterine environment of the fetus, the gut microbiota - or the outside - some element of the macroenvironment" (Landecker and Panofsky 2013: 339). The cells, tissues, uterus, gut, and "outside" represent different scales of the environment. Not only are there multiple scales of the environment, but these scales are related to each other. The molecular environment and the natural built-scale of the environment interact to show how environmental stimulation or stress can "come into the body" and change genetic sequences (Landecker and Panofsky 2013:349). For instance, through the epigenetic logic of environment, the uterus is now conceived of as a "uterine environment." The uterus and, by extension, women's bodies are regarded as environments that can change or modify genetic sequences in the developing fetus. As Gluckman and Hanson explain, "the environment of the fetus is created by its mother and, once developed, by the placenta" (Gluckman and Hanson,

2005: 530). This description of the environment breaks down the fetal environment to include the individual (mother) and tissue (placenta). There are different ways of referring to this scale of environment; some scientists that draw from epigenetics say the "environment of the fetus," while others use the phrase the "uterine environment."

Another way to signify the environment is the phrase "maternal metabolic state," which describes the metabolic processes that influence the uterine environment. The "maternal metabolic state" refers to the mother's ability to maintain a particular kind of metabolic state, or a condition defined by behaviors, food, politics, and multiple scales of environment, including the fetal/uterine environment. In Chapter Two, I show how the prioritization of particular scales narrows the focus onto individual women as opposed to larger political, structural, social and gendered environments.

A scalar analysis of epigenetics prioritizes the idea that there are multiple scales of the environment that relate to particular times and spaces. In this way, I suggest that environmental scale is relational with respect to time and space. The uterus is one particular time-space, or scale of the environment, as well as the maternal metabolic state, or the natural built environment. Scale, as an analytic, allows me to zoom in and out of different contexts of epigenetic influence and explore how different scales of the environment are related.

To examine the environment through scale, I draw from Anna Tsing's work on global connections and environmental conservation. Tsing states that her "story inquires into the makeshift links across distance and difference that shape global futures, and ensure their uncertain status" (2005:2). By doing so, Tsing focuses on "the links between heterogeneous projects of space and scale making" (2005:12). Similarly, my approach to the environment exposes epigenetics as a scale-making project. I trace how epigenetics is unfolding in a new post-

genomic era, and show how "makeshift" links of risk in the present are used to shape assumptions about future disease. Like Tsing, I aim to emphasize how the links across time, space, and scale are unpredictable and uncertain, particularly in the context of epigenetics. In addition, I aim to explore the different politics of comparisons and hierarchies of value that focus on particular scales over others (Choy 2011).

Although I emphasize how the epigenetic logic of the environment is inherently relational and scalar, in practice, scientific research that applies epigenetics aims solely at finding linear and causal correlations. However, finding linear patterns of causation is inherently impossible in epigenetics because epigenetic modification is by definition stochastic (or unpredictable). Therefore, approaches that target only specific scales of the environment deny the relationality between and among different scales of the environment. I argue that while epigenetic logic implies multiple entangled environmental scales, the implementation of the clinical trials I examined only target specific scales of the environment such as the uterine and molecular scales.

*Epigenetic Inheritance: a new form of relationality*

The epigenetic form of inheritance is fundamentally different than genetic inheritance. Specifically, epigenetics is changing the ways in which we understand relationality through inheritance. Moshe Szyf explains: "It was generally believed that most of the epigenetic information is erased during early gestation, and if this erasure were complete then errors in the epigenetic markings would not be transferred across generations" (2009:9). Due to unpredictable modifications on parts of the DNA it is possible that these changes can actually be inherited across generations. It is also possible that the inherited changes can be reversed by pharmacotherapy or behavioral therapy (Szyf 2009). Overall, epigenetic science claims that

epigenetic modifications are unpredictably inherited across generations, and inheritance is no longer isolated to the genetic information from one sperm and one egg.

There are many scientific studies that explore epigenetic relationships between inheritance, behaviors, and genetic modifications in animal models. In one such study, cited by over three thousand times, the authors concluded that stress response is inherited by the offspring after birth and outside of the womb (Weaver et al., 2004). The offspring's ability to respond to stress differently in adulthood depends on the maternal behavior during early development. Weaver and his colleagues Moshe Szyf and Micheal Meany proposed the "molecular conduit model" to help explain the relationship between behaviors and epigenetic changes. The model shows, "how behavior is embodied in molecules that themselves go on to pattern behavior in the future; in other words, the conduit goes into the body (from behavior to gene methylation), but it also runs back out again (from gene methylation to behavior)" (Weaver et al. in Landecker and Panofsky 2013:341).

In examining epigenetic inheritance I emphasize the role of relationality. For instance, there are two aspects of epigenetic inheritance that reflect new forms of relationality. First, the conduit model shows a tangible pathway between behaviors and biochemical changes from one body to another across time and space. Then there is another relationship between the epigenetic changes that results in an altered behavioral response to stress later in development. Both aspects of genetic modification through behaviors are inherited outside the body. The conduit model redefines inheritance to include both biological and behavioral relationships between mice and their offspring. The role and potential influence of the mother's behavior on the offspring's development maintain significant consequences for the gendered responsibility placed on

mothers to behave in a way that produces healthy and well-adapted offspring. The latter point is one that I return to in Chapters Three and Four.

Another example of epigenetic inheritance relates to the fetal/maternal relationship. The material and mechanistic relationships between a pregnant person and a developing fetus are completely different as a result of epigenetic logics of inheritance. For instance, since the 1980s, it was generally believed that the body and behaviors of a pregnant person had a uni-directional effect on fetal development. However, scientific research now claims that the fetal/maternal relationship is bi-directional, or that the fetus also affects and leaves marks on the maternal environment. Put another way, the pregnant person can inherit or take on cells from the fetus, which can change health outcomes later in life. For example, Boddy and colleagues found that fetal cells stay in the body long after pregnancy (2015). Fetal cells found in bodies are said to influence autoimmune responses, milk production, cancer risk, and mental and emotional health. Through the porous entanglement of different environmental scales, the fetal and maternal environments are imbued with a new form of relationality. Further, the study of fetal microchimerism, or the coexistence of genetically distinct cells in the same body, also implies that the bodies can have cells from grandparents and great-grandparents, which is a material illustration of the relationality that exists across time and space.[16]

In my examination of epigenetic inheritance, I highlight the different forms of relationality in trans-generational inheritance, fetal/maternal relationships, and the social/biological realms. I draw from anthropological and philosophical literature that examines the blurred boundaries between nature-culture and time-space, as a framework for relationality. For instance, Lefebvre's analytical framing of space claims that space is *both* natural and cultural, subject and object, immediate and mediated, given and artificial (1984:85). Lefebvre's

[16] See Lee Nelson's Lab at the University of Washington

approach to space challenges binary dualities imposed by the philosophical lineages of Descartes and Kant. In examining the relationship between time and space, or space-time, Nigel Thrift argues that space-time is a relational process of becoming that is constantly performed through daily practices (1996). Phenomenological approaches also prioritize a form of relationality by challenging divisions between mind/body, nature/culture, and space/time (Csordas 1994; Merleau-Ponty 1962). From a different approach, Elizabeth Grosz also emphasizes the relationality through her ontological approach to time. Grosz uses an ontology of time to expand or reimagine the ways we think of cemented nature/culture divisions that shape relations relevant to, but not only, reproduction and sexual difference. She takes seriously evolutionary biology without naturalizing reproduction to female bodies and she does this by examining space and time (2004). Finally, it is significant to note that the development of relationality across time-space and nature-culture is well established in non-European or Anglo contexts (Escobar 2014; Blaser 2013)

Similarly, I argue that a relational approach to epigenetics helps us see other forms that connect and link different bodies, organisms, and space-times. Although social scientists have proposed various approaches to understanding the entanglements of the social and the biological in relation to health disparities and institutionalized racism (Gravlee 2009, Williams and Colins, Chavez 2004), epigenetic inheritance offers another tool or heuristic for understanding how the "outside gets inside" (Landecker 2011). Or, rather, how there is no outside or inside but instead a complex web of scalar connections across time and space that reflect the relationality of material conditions, social histories, and different bodies. By focusing on relationality, I emphasize how epigenetic inheritance can shape new questions, but in order to do so we will need to explore new scientific methods that do not rely only on linear, causal correlations.

*Epigenetic Risk: a transversal approach*

Through epigenetics, different theories have emerged to explain the risk of disease across the lifespan. Drawing from both the "fetal origins hypothesis" (FOH) and developmental origins of disease theories, Kuzawa and colleagues claim that the "maternal diet and nutrition status during pregnancy predicts cardiovascular risk, blood pressure, insulin resistance, type 2 diabetes, and a tendency to despot fat in the central, metabolically active fat depot" (Kuzawa et al. 2009: 133). During the late 1980's and early 1990's, British physician David Barker published a series of articles that are largely credited for the developing the notion of fetal programming, fetal origins hypothesis, and latency, which later became part of a widely accepted analytic framework described as developmental origins of health and disease (DOHaD). Fetal programming is the idea that the epigenome, which includes chemical changes to genetic sequences, can be programmed in the uterine environment.

Fetal programming is a key aspect to the fetal origins hypothesis that claims that fetal programming during pregnancy can permanently impact metabolic or nutritional processes in developing offspring (Barker 1992). In addition, the epigenetic modifications that happen during pregnancy can manifest into metabolic disorders later in life, which is intrinsic to the notion of latency. Latency refers to the ways in which the fetal programming can have permanent effects that *may or may not* be triggered later in life (Hales and Barker 1992 and 2001). These concepts came out of studies that focused on nutrition during pregnancy and famine studies. The risk of disease is therefore connected to actions and conditions in past and future generations. The epigenetic relationship between time, space, environments and inheritance challenges linear and speculative notions of risk. The process of tracing chronic disease in adulthood to exposures that

occur in the womb, or exposures to famine in a previous generation, reflects a new form of epigenetic risk.

The epigenetic logic of risk informs the ways in which my informants approach obesity during pregnancy. For instance, the principal investigator in the UK explicitly draws from the Barker hypothesis to claim that the "maternal metabolic state plays a very important role in the *future risk of disease* in the developing child."[17]Through epigenetics risk during pregnancy could potentially carry consequences for more than one generation.

Throughout the ethnography I show that the clinical trials I examine draw from epigenetics to justify prenatal interventions. The justification is grounded in the prevention and anticipation of future risk if people change their diets while pregnant. The speculative approach to risk, even in a new epigenetic paradigm, is not novel. Adams, Clarke, and Murphy point out that the speculative turn in the life sciences is characterized by anticipatory regimes (2009). They argue that efforts to anticipate the future are increasingly defining the present. They argue that the individualization of responsibility upon gendered bodies is characteristic of a techno-scientific regime framed by speculation and anticipation (2009). The authors apply their notions of anticipatory regime and temporal politics to the issue of HPV and the expanding market of Gardasil. The vaccine is said to be more effective if it is administered to people *before* they are sexually active. Therefore the vaccine is aimed at young girls. In the case of HPV vaccination, girlhood becomes the site and time of intervention in an effort to prevent the cost and risk of cervical cancer in the future. Although the anticipation of risk is not new, I argue that the novel component of epigenetic risk is the notion that one person's behaviors in the present or past can have material implications for future generations. Therefore, risk is not only speculative, but retrospective, and also deeply embedded in the present.

[17] 6/18/14 LP my emphasis

I use the concept of *transversality* to explain how epigenetic risk crosses through the spatio-temporal planes of the past, present, future. Transversal is a geometric term that describes how space and time can intersect.[18] In the context of epigenetic risk, the risk exists and is distributed across the past, present, and future, and across different generations. For instance, pregnant women in the present, whether they are obese or not, could have already inherited epigenetic modifications from their grandparents or great-grandparents, which could have already affected fetal development in the present and future. Through epigenetics, risk can simultaneously be inherited from the past, experienced in the present, and potentially passed onto future generations. However, in the trials that I examine it is impossible to capture the transversal component of epigenetic risk. The clinical trial method cannot capture, assess, measure, or predict the potential risk that runs through the past, present, and future. Further, the scientific method is not able to assess transversal risk because the questions that frame the method are focused on finding causal correlations, not transversal connections.

The epigenetic risk incorporates both the potential and limits of epigenetic science. On the one hand epigenetic risk provides an alternative view for thinking about risk in the past, present, and future across time and space. On the other hand, the application of epigenetics using RCT methods focuses only on future risk, and not on the potentiality of transversal risk. Finally, in the clinical trials that I examine, pregnant women who are obese or diabetic are not just risky because of their own individual conditions, but they are risky more importantly because of their potential to (re)produce a generation at risk of obesity and diabetes. This later characteristic of risk is explored in Chapter Four.

[18] Although I use geometry as a point of reference, there are many others such as, the American Indian tradition that denies linear progression of time, or the cyclical temporality inherent in Hindu traditions of reincarnation.

The common thread across epigenetic environments, inheritance, and risk is space-time. There is a spatio-temporal aspect to the different epigenetic logics that I have outlined above and expand on in the rest of the ethnography. In this way, the epigenetic environment includes both time and space. Likewise the relationality in trans-generational inheritance is also spatio-temporally significant. Finally, as I described above, the transversal potential of risk in epigenetics also reflects the role of time and space. I return to the implications of the spacetiem for prenatal interventions in the Conclusion.

Next, I turn to the topic of randomized clinical trial design, the gold standard of evidence-based method. Doing so will help illustrate one of my key arguments about the concepts and theories inherent to epigenetics and the contradictions involved with using RCT method for the purposes of examining epigenetics and prenatal interventions. If we take seriously the logics of epigenetic environments, inheritance, relationality, and risk, then the randomized clinical trial method is an inadequate tool for examining the non-linear, unpredictable, and transversal characteristics of epigenetic logics.

**Randomized Clinical Trials (RCTs): The Gold Standard of Scientific Knowledge Production, Experimentation, and Validity**

This section aims at outlining key aspects of RCTs, its development, and role in scientific knowledge production. I argue that RCTs are the dominant form of producing valid scientific medicine in the Global North. The dominant position of the RCT method is not a naturally occurring phenomenon. Below I provide a snapshot of the historical development of RCTs

throughout the twentieth century. Tracing the development and expansion of RCTs helps explain why RCTs are the primary method for testing epigenetic theories.

The key component of the randomized clinical trial method is its purpose. The method is designed to find causal correlations between an intervention and an outcome. Also, RCTs require at least two groups for comparison. One group is called the control and the other is the experimental. Both groups need to be the same, except for the intervention that is applied to the experimental group. If the groups are the same, then any differences that result between the two groups is contributed to the intervention. The RCT is called the "gold standard" of evidence-based medicine because it is perceived as the main method for producing valid scientific evidence. As a result, the RCT method is the main approach within biomedicine for influencing and testing health policies. The current dependence on RCTs in the fields of obesity and diabetes, education, economics, and global health is well established (McGoey et al. 2011, Cartwright and Hardie 2012, Cohn et al. 2013,). However, the production and implementation of an RCT is very expensive. More RCTs are funded and designed by scientists in the Global North than anywhere else in the world. Consequently, there exists a disproportional influence on global health policies by the Global North (Anderson 2006, Petryna et al. 2006, Sunder-Rajan 2006, Palmer 2010, Pollock 2014).

One genealogical origin of the RCT method can be traced to the work of Claude Bernard. Bernard wrote the book *Introduction to Experimental Medicine* in 1865*,* which proposed a model for using laboratory experiments to test and examine theories of chemical processes and disease. Pivotal to his model was the notion of comparison. He established the need for a control and experimental group. Experimental comparison is foundational to the RCT design. The

comparative approach of a control and experimental group further developed in the fields of experimental psychology in the late nineteenth century.

In the early twentieth century, experimental methods continued to develop and spread into other geographic areas.[19] For instance, "the sites of laboratory research expanded from their original places in the European heartland […] to the outermost reaches of the European empire, where field laboratories used to study exotic tropical disease that afflicted settlers resulted in networks of research institutes dotted throughout Africa, Asia, and Latin America" (Nguyen and Lock 178:2010). The spread of research networks in colonial areas reflects the intimate relationship between experimental medicine and colonialism. Hygiene campaigns focused on "teaching" or disciplining people in colonial areas. For instance, Pasteur's theories of hygiene were applied, tested, and used to treat populations in French colonies (Nguyen and Lock 2010).[20] The expansion of comparative experimental models in the colonies also helped further develop the method itself.[21]

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[19] See E. Greenwood, and F.S. Chapin's work on experimental interventions in sociology and education.

[20] Latour's book *The Pasteurization of France* (1993) documents the networks of people and germs related to the spread of hygiene campaigns in France and the colonies. Pasteurization became a method as well as a concept. For instance, "hygienist movement came to be identified with the man Pasteur, and ultimately, following a very French habit, the man Pasteur was reduced to the ideas of Pasteur, and his ideas to their 'theoretical foundations'" (1993:23). This method, practice, and strategy swept through France and eventually fed into the discipline of microbiology.

[21] With the support of the Rockefeller Foundation in the United States, hygiene campaigns on hookworm were experimented, tested, and implemented in the Caribbean and Central America (Palmer 2010). Palmer argues that these sites were also used "as a laboratory for discovering and testing the elements of a global health system for the twentieth century" (2010:1). Similarly, Anderson found that hygiene campaigns in the Philippines by US soldiers functioned as laboratories of hygienic practices (2006). He argues further that the making of the modern citizen subject involved the making of clean hygienic subjects. The laboratories of hygienic practices, established by white male military and public health officers, were not only teaching and disciplining hygiene but they were also testing the effectiveness of subject formation (2006). Overall the laboratories in colonial setting set the stage for experimentation at the disciplinary, subject formation level and at the level of scientific knowledge production through comparative models of controls and treatment. These practices expanded to global networks of institutes that fed into the London School of Hygiene and Tropical medicine, the Antwerp Institute for Tropical Medicine, and the Royal Tropical Medicine in the Netherlands (Lock and Nguyen 2010). All of which became part of a movement towards global epidemiology and eventually the establishment of the World Health Organization (2010). I do not expand on it here, but many scholars note that the development of global health as we know it came out of testing hygiene models in colonial areas.

The international efforts to develop what we now call the RCT method were further developed domestically. Contrary to common perceptions, the RCT method was not based solely in the clinic or in medical laboratories. In the 1920s, F.S. Chapin from the University of Minnesota and Ernest Greenwood at Columbia University used experimental methods to study topics related to rural education, public housing, and social interventions for "delinquent" boys (Oakley 1998). The underlying premise of using the randomized control design was to show a connection between social interventions and improved outcomes among different populations. Greenwood and Chapin's efforts helped create a pedagogical movement across the United States that promoted the design and application of experimental models in sociology (Oakley 1998).[22]

The RCT method was further developed in agricultural studies. In 1935, Fisher published a book called *Design of Experiments*, which was based in agricultural research but ended up fundamentally changing medical research. Fisher is credited with incorporating the randomization aspect of randomized control trials (Mark 1997). Fisher claimed that randomizing samples, or subjects, into control and experimental groups would limit the bias on behalf of the staff and investigators. Today this approach is another fundamental aspect of the RCT method. In addition, Fisher combined probability statistics from Gregor Mendel's work on pea plants to measure the uncertainty of experimentation (Howie 2002). Testing the randomized experimental method on plants soon expanded to other organisms. Fischer's work on the RCT design was taken into the clinical setting and his plants were replaced with human bodies—bodies that became experimental subjects upon which and through which reliable, valid, and statistically significant knowledge was produced.

The RCT design experienced another phase of development after World War Two. In 1946, Austin Bradford Hill became among the first to develop a way to standardize the

[22] See Ernest Greenwood book *Experimental Sociology* 1945

implementation of randomized control trials. While exploring the effects of drugs on patients infected with tuberculosis, Bradford used a randomized control design to develop drugs and disseminate them at a large scale (Cooper 2011). The standardization of the RCT design helped promote its use in medical care spaces. The randomized control trial became synonymous with the term randomized clinical trial, which is subtly different but reflects the clinical and medical research focus on human subjects.

By the mid-twentieth century the RCT design went from laboratory experimental use to large-scale standardized and regulated applications in drug development and medical research. This trajectory had significant implications for the development of healthcare structures and health research. Melinda Cooper argues that the expansion of mass drug markets, national health care, and biomedicine could not be possible without the development and institutionalization of the RCT method. Through the growth of industrial production, she argues that it became "feasible, for the first time, to manage large volumes of clinical data, and to produce, measure and predict events on the scale of whole populations" (2011:83).[23] Moreover, Sturdy and Cooter argue that the post World War II environment shaped the standardization, implementation, and regulation of RCT methods in the National Health system in the United Kingdom (Sturdy and Cooter 1998).

Because of the rapid growth of using RCT method to test public policy, the period following WWII is also characterized as the "golden age of evaluation" in the United States

[23] Drawing from Rheinberger's definition of RCTs, Cooper states that the RCT "lies somewhere between the test and experiment, since it seeks to both provoke and standardize novel therapeutic interventions" (84:2011). Implicit to this process is the introduction of risk and the assumption of an 'average man' (Cooper 2011). That is, the inclusion and exclusion criteria are designed to eliminate as many variables as possible. The issue with the standardization of RCTs and measurements used and tested in RCTs is that it assumes an 'average human subject' or seeks out particular subjects that are assumed to be the same or representative of the normal. For instance, in the development of average/ healthy weight chart the measurements of height and weight were modelled off of men in the military. The measurements and standards of average height and weight came from the assumption that the male militarily fit body was the 'average human subject', which defined the terms of the normal height and weight and the deviations form that norm (Thompson 2005).

(Oakley 1998:1240). Research studies used the randomized clinical trial design to study work incentives, the maintenance of labor force participation, household behavior with regards to savings, and the costs and benefits of supporting disadvantaged workers (Oakley 1998). The focus of these interventions aimed to improve the economy and effectiveness of particular social polices. Oakley also notes that the studies using the RCT method often found minimal effectiveness of the interventions, which helped justify the budget cuts to social welfare services in the 1980s (Oakley 1998). However, it is not well understood whether the policies were not effective, or if the RCT method was not the appropriate tool to capture long-term nuanced effects of social interventions.

The twenty-first century reflects the proliferation of RCTs in the realm of prenatal interventions. Regardless of inconsistent results or failures in trials, there are more RCTs testing behavioral interventions on pregnant women now than ever before. Large studies on weight management, nutrition, child rearing, and depression are all targeting pregnant women for clinical trial interventions. Epigenetic theories claiming that mothers' behaviors during pregnancy and early development can affect mental and physical outcomes in the future provides a new and vital justification for the intervention on women's bodies and behaviors. For instance, a recent consortium of RCTs funded by the European Union, draws from epigenetics to justify interventions on women's children rearing practices. The study is based on the idea that trauma during pregnancy or in early development can affect "aggressive" behavior in adulthood.[24] Although I suggest that we are in an epigenetic paradigm, the methods and questions currently wielded by scientists are not that different from studies in the 1920s. As I noted above, Chapin used RCTs to study "delinquency" in poor families, which is similar to contemporary interest in finding causal links between aggressive behaviors in adulthood to pre and post-natal care. Older

[24] See the AGRESSOTYPE trial based in the European Union: http://www.aggressotype.eu/

assumptions about mothering, behavior, and delinquency are still present in contemporary applications of epigenetic theories.

Overall, the current techno-scientific climate is dominated by the production of RCTs in the Global North. The development of the RCT design over the past century focuses entirely on finding a causal effect of an intervention in an experimental setting. However, as I show throughout this ethnography, even with advanced statistical programs for "controlling" various variables, it is very challenging to maintain ideal control and experimental groups in an RCT designed to test nutritional interventions during pregnancy. More importantly, I argue that epigenetics is emerging in biomedical fields that are completely dependent on certain scientific methods. Furthermore, using a scalar approach to epigenetic environments helps us zoom out to critically examine the social and scientific context within which epigenetics is emerging. Next, I expand on my own methodology for examining RCTs that test nutritional interventions on obese pregnant women.

**Methodology**

Between 2012 and 2014 I collected ethnographic data at two clinical trials. In the United States I examined a trial called SmartStart, which was awarded a ten million dollar grant from the National Institutes of Health, and it is one of seven trials in a consortium. In the United Kingdom, I examined the StandUp trial, which was awarded thirty million pounds for ten years of research. The funding for the StandUp trial came from the European Union and the National Institute of Health Research in the UK.[25] I frame both trials as separate case studies to show how epigenetic knowledge is applied in different ways depending on the scientific background and

[25] Even though trial design and information is publically available, I have changed the names of all sites, staff, and participants to protect the identity of my informants.

expertise of the principal investigators. In addition, the trials reflect the divergent positions on maternal nutrition and weight gain that are maintained in each national context.

The incorporation of both trials in my research is not aimed at making analytical comparisons across each site. Instead, I frame my fieldwork as a multi-sited ethnography, which emphasizes the connections across different spaces, networks, and actors (Marcus 1998). Multi-sited projects emphasize the relationality between and among networks, without reifying geographic and national boundaries (Sunder-Rajan 2006). The incorporation of both the StandUp and SmarStart trials came about through conversations with key informants. I followed the connections and networks across the StandUp and SmartStart. The collaborators and principal investigators at each trial knew of each other tangentially and had published together. While I was in the field, another trial funded by the European Union approached both of the PIs at the StandUp trial and SmartStart trial to participate in a larger consortium of trials collecting nutritional data on pregnant women. Both PIs met each other at the European data consortium. The networks and trials involved in prenatal interventions are small but also competitive with each other. There is an awareness of the different experts working in prenatal interventions and running large RCTs. However, the collaborations are hard to build and there are internal tensions regarding the different approaches to obesity during pregnancy.

In an effort to embed myself into the world of prenatal interventions and obesity during pregnancy, I had to learn the "language" of my informants. Applying a science technology and society approach required an understanding of the relevant scientific methods and theories at my research sites. Therefore, I enrolled into the school of public health at University of California, Berkeley. My course work focused on epidemiological study design and methods. My assigned advisor had thirty years of experience working on maternal nutrition and clinical trial design. I

worked with her long term and created independent study focused on obesity during pregnancy, epidemiological methods aimed at testing prenatal interventions, and international maternal nutrition and weight policies.

Through my preliminary conversations and research I was introduced to the problem of "consensus" regarding obesity during pregnancy. The key difference in approaching obesity during pregnancy emulated from the distinct policy recommendations in the US and UK. The scientists that I worked with in the US did not understand why the scientists in the UK promoted totally different approaches to obesity during pregnancy. One collaborator for the SmartStart trial felt so passionately about the topic that she flew to England to discuss the matter with principle investigators conducting studies in the UK. Prior to going to England, she assumed that everyone believed in the same approach to obesity during pregnancy. When she confronted the PI of the StandUp trial in the UK, she realized for the first time that the perspectives and approaches were totally different. When I interviewed her, she told me that she did not understand the divergent approaches because both national settings used similar data. I followed the fault line between the US and UK, and doing so lead me to the two trials that I examine here.

*Access and Analysis*

In order to design, develop, and execute empirical research on two separate international clinical trials on pregnant women, I had to build relationships with elite networks of scientific researchers. After two years of building relationships and trust with different scientists in relevant fields, I was finally connected with two different principal investigators that were implementing large-scale trials on an obese pregnant population.

Once I had permission from each PI, I submitted my research proposal to three different ethical review committees including my university, and to the ethical research committees at each trial site.

To illuminate the structure and implementation of the trials from the perspective of the research participants, I followed the pregnant women through their clinical trial journey. I observed processes of recruitment, consent, data collection, and the implementation of each intervention. In order to understand the design and implementation of the trial from the staff's perspective, I interviewed forty individual staff members and collaborators for both sites, including research assistants, research midwives, consultants, health trainers, interventionists, and principal investigators. Those responsible for designing and monitoring the trial implementation came from a range of scientific disciplines (e.g., epidemiology, psychology, physiology, nutrition, obstetric gynecology, and midwifery). In addition, I collected archival materials from biomedical literature in the areas of epigenetics and behavioral interventions during pregnancy.

As an independent researcher I recruited all participants for my study and collected all the interviews and observations.[26] However, I was able to observe more women at the StandUp trial because it was based at a large teaching hospital and it had access to approach hundreds of women each month. In addition, the StandUp trial was in its fourth year of implementation when I collected most of my data, and the SmartStart trial was in its first year of implementation and recruitment. During the beginning phases, the SmartStart had a very difficult time meeting its

[26] The ethnographic material for this chapter draws from participant observation, interviews, and published articles by Dr. Elizabeth, the principle investigator of the trial in the UK. In order to preserve the anonymity of my informants I do not cite or quote from published articles directly, but instead paraphrase key messages. All names of people and the trial itself have been changed. Although, the people I worked with at my field-sites are public scholars and the information about the trials are also public, I make a concerted effort to protect their identities by not disclosing specific quotations and citations.

recruitment goals each month. Therefore, I was only able to observe six different pregnant women, whereas at the StandUp trial I observed seventeen. In addition to observing individual pregnant participants, I also observed twenty-seven intervention visits in the StandUp trial, and twelve intervention visits in the SmartStart. For all of the observational field notes I used thick description to illustrate the context, people, and practices of each clinical trial. In total I spent eight months at the SmarStart trial and six months at the StandUp trial.[27]

Only at the SmartStart trial did I have dual roles. While I was collecting my own ethnographic data, I also worked on the SmartStart trial as a "volunteer staff member." I was trained as a nutritional counselor and I worked closely with the intervention staff and principle investigator, who were all "unblinded", which means that the staff in charge of delivering the intervention knew who was receiving the intervention, but the staff that collected the anthropometric data did not know who was in the control or experimental group. At the SmartStart trial, the nutritional counselors are in charge of delivering the nutritional intervention. The staff meets with pregnant participants, randomized into the intervention group, every two weeks from sixteen weeks gestation until their eight month of pregnancy. Depending on the rate of gestational weight gain, the nutritional counselor may meet with the pregnant women until delivery. Tracing the experiences of the women in the intervention during their pregnancy gave me insight into their everyday routines, challenges, and social conditions.

My analytic methods are premised on processing the ideas that emerge from my ethnographic data. After completing the data collection phase of the research, I organized, transcribed and coded the data. I experimented with using textual programs for coding, like NVivo. However, in order to have a more intimate understanding of the data, I preferred going

[27]I was only given permission to stay in the UK for 6 months.

through all of it by hand. I analyzed all of the transcribed notes, highlighted themes and created a codebook. To distill key themes, I repeated this process multiple times.

When analyzing my data I used a triangulation method to corroborate and situate my particular case study and ethnographic vignettes. This method helps provide perspective on how my particular case studies relate to other understandings of trials testing prenatal interventions. In addition, I include themes that are counter to ones that I have found, or "negative information." Using negative information challenges holistic or "complete" depictions of my field site and informants (Strathern 1988).

My perspective and analysis of epigenetic science aims to co-exist with other similar or contradictory perspectives on scientific knowledge production. By situating my methodological approach I intend to reveal the taken for granted and often obscured roles of race, gender, and power that are inherently apart of the production of scientific knowledge. Therefore, by describing the ways in which my informants and pregnant women narrate what they do, my intention is not aimed at critiquing their work or lives. Rather, I focus on illustrating the everyday practices and cultures of science through the lens of a feminist ethnographer (Franklin 1995, Haraway 1991, Traweek 1988).

*SmartStart and StandUp trials: structure and design at each site*

The SmartStart trial was structured very differently from the StandUp trial because of different health systems in each national setting. The SmartStart trial had to build relationships with public and private prenatal and reproductive care clinics within a fifty mile radius. Gaining permission from each clinic to recruit in their offices was a big challenge, and then getting women to consent was the other big challenge. The SmartStart trial organized lunches, and

raffles to get the nursing staff at the participating clinics to refer eligible pregnant women to the trial. There were about six to seven participating clinics that allowed the staff to sit in the lobbies and recruit pregnant women. I had experience recruiting pregnant participants in both public and private clinics. From my observations, the majority of the pregnant participants were recruited from the public Medicare clinics, which served low-income or unemployed women. In addition, once we did recruit the women from different clinics, depending on the distance, we would have to travel to different locations to deliver the intervention. I traveled thirty-five miles regularly to recruit and deliver the intervention.

In contrast, the StandUp trial was centrally structured and it was based in a large teaching hospital within the National Health System (NHS). Being embedded within the NHS helped the StandUp trial with recruitment, resources, and labor. The trial recruited, implemented the intervention, and analyzed the data in the same building. There was also a different attitude towards participating in clinical trials in the UK because of the NHS. I often heard women at the StandUp trial stating that they wanted to participate in the trial to give back to the NHS and to science. Also, women said they participated in the UK because they had access to a midwife, and could ask her questions about their pregnancy. In the US the "giving back" sentiment did not exist, and the US did not have any national or standardized form of midwifery care incorporated into standard prenatal care. I will expand on the different relationships across each site in the conclusion.

The main similarities across each site had to with the clinical trial design. Since both sites used the "gold standard" of evidence-based medicine their trial designs were the same.[28] The

[28] Except for the "semi-blinded" aspect of the SmartStart trial. The StandUp trial did not blind any of their staff members. Everyone at the StandUp trial knew who was randomized into the control or intervention groups. As a result of the NHS, the staff members in the StandUp trial included midwife researchers whereas, the SmartStart study did not have any midwife researchers. In addition, the SmartStart trial had a completely different hiring

similarities across each trial had to do with the RCT. Both trials had a control group and an experimental or intervention group and every participant is selected randomly, rather, each participant has a fifty/fifty chance of getting selected into the control or experimental group. The control group received the same standard prenatal care that any other pregnant received at each respective location. The trials both wanted a diverse range of ethnic and socio-economic representation in their population. In addition, each trial is designed to follow the children of the pregnant participants up to three years after delivery. Both intended to collect bio-samples from the children to evaluate their blood glucose levels, and other hormone and metabolic activity related to obesity and diabetes. In this way, both studies are testing epigenetic mechanisms in the long term. However, each PI has a particular agenda attached to this broader goal of understanding epigenetics. The distinct agendas on behalf of Dr. Sally and Dr. Elizabeth have to do with the types of data they prioritize. Different types and amounts of data can bring different kinds of meaning to epigenetic knowledge production.

For instance, there are different types of data that are collected at each trial. Behavioral data includes the data from the intervention delivery. The bio-sample data includes blood, urine, cord blood, and placenta samples along with biometric data.[29] Each site is also part of the larger consortium, however, the main difference is that in the UK consortium everyone collects the same types of behavioral and bio-sample data. In the US consortium different sites can collect their own individual data, and also "common core measurements", which are required from all participating sites. The common core measurements include weight, height circumference, blood, urine, and placenta. There are also site-specific data that each PI wants to collect, for instance,

strategy, which often involved word of mouth or university job postings in search of research assistants and health promoters in the community.

[29] This includes weight, height, and circumferences of legs, thighs, waist, hips, neck, and arms. The UK site also takes skin fold measurements.

Dr. Sally is collecting the majority of the behavioral data, with one other colleague. There are only two sites in the US consortium that are doing an intervention with meal replacements on obese and overweight pregnant women. Other sites are also collecting breast milk, but Dr. Sally said she would not collect breast milk.[30]

Another difference in the data collection is that Dr. Elizabeth is collecting blood samples five times during pregnancy and Dr. Sally is only collecting blood three times. As one of the collaborators told me in an interview, the committee thought that it would be too much of a burden on the pregnant participants to draw blood more than three times during pregnancy. During my observations and training as a staff volunteer at the US site, I was told to clarify to all participants exactly how many tubes of blood would be drawn at each collection. Many of the women resisted having their blood drawn and saw it as a downside of participating. Besides the burden of collection, Dr. Sally did not want to risk detouring women from participating in the trial by collecting blood more than three times. The women in the UK would also mention how much blood they had to give during pregnancy. However, the midwife researchers on the StandUp trial would always say "during pregnancy women produce thirty percent more blood, so you have extra to give." I explore the details of each trial throughout the rest of the ethnography.

**Chapter Summaries**

Chapter two traces four distinct phases in maternal nutrition and weight across the US and UK during the second half of the twentieth century. In this chapter I show how science and policy change in relation to social and historical contexts. The first part of the chapter highlights the divergent approaches that the UK and US have in addressing obesity during pregnancy. The

[30] Recently, another researcher contacted Dr. Sally to see if they could collaborate and collect feces from the pregnant participants and their children before and after delivery. The study is intended to explore the popular topic of the microbiome. The idea is that the gut bacteria in obese women are contributing to their obesity.

second part of the chapter focuses on explaining, through the analysis of scientific causal diagrams, how the emergence of the epigenetic paradigm shifted approaches to maternal nutrition and weight. As a result of epigenetic logics maternal nutrition and weight are now framed as environmental factors that can influence fetal development. Framing maternal nutrition and weight as potentially risky environmental factors for fetal development justifies the implementation of nutritional interventions during pregnancy.

Chapter three explores epigenetic environments and scales. Through epigenetic logics "the environment" includes many different scales, from cells, uteruses, maternal bodies, and atmospheres. In this chapter I apply a scalar approach to examine the different environments that are prioritized or elided in the design and implementation of the StandUp trial. By doing so, I show how particular scales of the environment, like the uterine and molecular scales, are targeted for intervention. Targeting certain scales of the environment over others reflects the selective application of epigenetic logics. The PI at the StandUp trial is an expert in physiology and animal studies which directly influences which scales of the environment she prioritizes. In this chapter I show that epigenetics is not a homogenous paradigm. Rather, the applications of epigenetics varies in relation to research design, animal modeling, and data collection. Overall, the chapter highlights a key tension in the knowledge and application of epigenetic science in prenatal interventions: although epigenetic logics claims that there are multiple entangled scales of the environment, in practice scientific studies using RCT methods can only target narrow and discrete scales of the environment.

Chapter four examines how nature, nurture, nutrition and the environment are interconnected through the epigenetic logic of inheritance. In Chapter Two I focus on a scalar approach, and Chapter Three emphasizes the relationality across scales of the environment.

Exploring the SmartStart trial I show how the expertise of the PI influenced the design and implementation of the trial intervention, which focuses entirely on controlling calories or exposure to food. Researchers carefully monitored pregnant persons' diet in an effort to reduce the risk on the developing fetus and by temporal extension the future child. The assumptions undergirding epigenetic ideas of nature, nurture, and nutrition is the gendered and bias concept of "good mothering." I argue that the SmartStart trial reflects the ways in which social and gendered ideas of motherhood are folded into scientific research questions. The unexamined issues related to motherhood, gender, sexuality, and heteronormativity limit the possibilities of epigenetic knowledge.

Chapter five examines the paradox of pregnancy, which I describe through the entanglement of individual responsibility, risk, and control. Drawing from feminist literature and social theory, I argue that pregnant persons are targeted as individuals who have complete responsibility and control over fetal health outcomes. However, epigenetic logics explain that in fact pregnant persons cannot control or influence health outcomes due to epigenetic changes. Epigenetic logics of risk are transversal, that is epigenetic risk crosses through time and space. The transversality of epigenetic risk emphasizes the potential for epigenetics to have already been inherited in the past, triggered in the present, and inherited into the future. However, the trials that I examined do not acknowledge the transversal risk intrinsic to epigenetics. Instead the trials I examined individualize future risk to pregnant persons. The data that I explore in Chapter Four focuses on the narratives of the pregnant participants in the StandUp trial. By doing so I aim to emphasize the complex ways in which pregnant persons subject themselves to systems of surveillance. Furthermore, the paradox of individual responsibility, risk, and control impacts the legal and political policies around prenatal surveillance, health and treatment.

## Chapter 2:

## Knowledge and Standards: Approaches to Maternal Nutrition and Weight

The growing public concern over the obesity epidemic, and emerging epigenetic paradigms of fetal programming claiming that women's weight and diets during pregnancy can have permanent effects for their children and grandchildren, puts pregnant women at the center of a two-pronged debate: Should weight be monitored during pregnancy? And if so, how much weight should women gain? Currently, there is no international consensus on how to address these questions, which are also key aspects for examining obesity during pregnancy.

Obesity during pregnancy is defined as having a body mass index, or the ratio of height to weight, equal to thirty or more at the beginning of pregnancy. The issue of obesity during pregnancy is a priority for the National Institutes of Health (NIH) in the United States, and the National Institute of Health Research (NIHR) in the United Kingdom. Both the NIH and the NIHR are investing millions of dollars and pounds into understanding the potential risks associated with obesity during pregnancy, as well as metabolic syndromes that may develop in the children of obese pregnant women.

In the United States, focusing on weight during pregnancy is intimately entangled with prevention efforts and relevant clinical research. The Institute of Medicine (IOM) in the United States has published standards and recommendations for how much women should gain during pregnancy in 1970, 1990, and 2009. By contrast, weight is not regarded as a significant measurement for pregnant women in the United Kingdom. What is more, the World Health Organization (WHO) does not give recommendations for weight gain during pregnancy. The WHO aligns itself with the UK National Institute of Clinical Excellence (NICE), which claims

that there is not enough evidence-based science to prove any positive health outcomes of routinely weighing and monitoring women's weight during pregnancy (NICE guidelines 2010). It is expected that the WHO may change its position on this issue in the coming years, as it draws on evidence-based medicine, and, importantly, on randomized clinical trials (RCTs).

My fieldwork focused on two clinical trials in the US and UK that were guided by respective national health guidelines on weight during pregnancy. The US-based trial requires pregnant participants to monitor their weight daily, control calories, and record physical activity, in order to reach their weight gain goal of half a pound per week throughout their pregnancy. The gestational weight gain (GWG) goal of a half a pound per week comes from the IOM's most recently published guidelines, which are based on the claim that the rate of weight gain during pregnancy is causally connected to pregnancy complications for the mother and long term health outcomes for the child.[31]

In contrast to the US trail's emphasis on weight gain, the UK trial focuses on glycemic control. Glycemic control aims at lowering pregnant participants' intake of sugar, saturated fat, and carbohydrates, while also increasing exercise. Drawing from the NICE guidelines, the UK trial does not monitor weight during pregnancy and does not provide any weight gain recommendations for its participants. The UK maintains that weighing women during pregnancy is not a necessary practice. Furthermore, the scientists and health professionals I interviewed in the UK believe that weighing women during their pregnancy causes unnecessary anxiety. Health professionals working on the UK trial did not regularly monitor participants' weight gain or give any specific recommendations for how much weight they should gain.[32]

[31] See diagram in Part II.

[32] However, this is changing, and it varies widely across different hospitals in the UK, some hospitals will monitor the weight of women who have a BMI over 30, but will not give them specific ranges for how much they should gain or loose, instead some women with high BMI are sent to obesity clinics during their pregnancy.

The UK is among a minority of countries that do not offer pregnant women such guidelines. In a previous research collaboration, my colleagues and I found that most countries around the world implement formal or informal policies for obese pregnant women based on the IOM recommendations (Simons et al., 2014). Of the countries that had formal or informal policies for maternal weight gain, 80% monitored gestational weight gain (Simons et al., 2014). If countries did not have any formal policies that were supported by their government, then the alternative would be to use the IOM recommendations. Our results reflected that there is an international concern over obesity during pregnancy, but that there is no consensus on how to address it. Even if there are formal policies in a country, the way these are executed at different levels of medical care is not uniform.

These different approaches to obesity during pregnancy have political implications for the production and influence of scientific knowledge and health policy. For instance, one expert surveyed in this previous research commented that her country – located in the Global South - had no formal policies on weight gain during pregnancy. Consequently, she and other healthcare workers had to draw their recommendations and policies from "developed countries," like the US and UK. The global significance of IOM recommendations with regard to obesity during pregnancy reflects the unequal amount of resources that the Global North can spend on obesity research. Other testimonies from the data that were not published stated that they trusted the IOM recommendations for weight management during pregnancy, because the guidelines were based on extensive and reliable scientific research.

Health policies and research designed and funded from the Global North reveal two key issues. First, evidence-based medicine in the form of randomized clinical trials are the main sources of data for policy recommendations. The dependence on RCTs in the fields of obesity

and diabetes, education, economics, and global health is well established (Cartwright and Hardie 2012; Cohn et al. 2013; McGoey et al. 2011). In addition, clinical trials are expensive methodological tools of knowledge production, and are primarily designed by countries in the Global North (Anderson 2006, Petryna et al. 2006, Sunder-Rajan 2006, Palmer 2010, Pollock 2014,). Therefore, the existing distribution of research and policy development with regard to obesity during pregnancy results in a disproportionate influence of knowledge production and policy influence disseminating from the Global North.

The unequal production and influence of knowledge and international policies is an underlying theme in this chapter, but not the main focus. This chapter focuses on the convergent and divergent approaches to obesity during pregnancy within the Global North. Although other countries trust the US guidelines, the UK does not. The UK-based NICE guidelines specifically state that their guidelines on weight management before, during and after pregnancy, on maternal nutrition, and on antenatal care draw from evidence-based medicine only. NICE argues that the IOM guidelines are based on observational studies and not randomized clinical trials. NICE states that there is not enough evidence-based medicine to support the focus on gestational weight gain as a key indicator of healthy pregnancies (2010). At stake in these differing approaches to obesity during pregnancy are questions of what counts as legitimate knowledge for policy-making.

The international concern and contestation over obesity during pregnancy provides fertile ground for tracing how divergent clinical practices become standardized in some places but not others. If science is universal, and if both the US and the UK draw from similar scientific studies, why are there such different approaches to nutrition and weight during pregnancy? This chapter is organized around two key segments. The first part of the chapter traces four historical shifts in

approaches to and recommendations for maternal nutrition and weight. The second part examines two key documents to show how the epigenetic paradigm influenced the current guidelines for obesity during pregnancy, as well as the field of maternal nutrition writ large.

Part one is organized around the narrative theme of maternal nutrition and weight. How have medical and scientific approaches to maternal nutrition and weight changed across time and space? To answer this question, I draw from national health reports and scientific studies from the second half of the twentieth century across the US and UK. Four shifts in this history include: the risk of toxemias due to gestational weight gain, the risk of delayed development due to low gestational weight gain, the risk of overweight infants due to high gestational weight gain, and finally an approach to maternal nutrition and weight that is marked by the epigenetic paradigm. I argue that the four main shifts in maternal nutrition and weight are framed by particular scientific trends and historical events. Tracing the ways in which maternal nutrition has changed over the past 60 years also reflects changes in scientific knowledge and practices.

Part two highlights the emergence of the epigenetic paradigm and the ways in which epigenetic science influenced US policies and approaches to obesity during pregnancy. I analyze causal diagrams from two IOM reports published in 1990 and 2009. By comparing these two diagrams, I highlight the significant logics of epigenetics that appear in the most recent report. The emergence of epigenetics is indexed by three key themes. First, in the 1990 report the environment did not play a key role in obesity during pregnancy, whereas the in 2009 report illustrated different scales of the environment. Further the environment is an overarching principle connected to the fetus/child, mother, and disease. Second, whereas the 1990 diagram associated excessive gestational weight gain with obesity risk for the mother, the 2009 diagram associates obesity risk for the mother, fetus/child, and future adult. Finally, the 2009 diagram

includes a relational connection between the fetus and mother that did not exist in the 1990 report. Overall, I argue that these key themes related to environmental scale, risk, and relationality index the ways in which epigenetic logics are changing scientific research questions, health treatments, and interventions on pregnant women. I show how the scientific and medical changes in approaching obesity during pregnancy are a result of the epigenetic paradigm. Part two also provides a link to later chapters that further examine the significance to the epigenetics paradigm.

## Part 1: How Science Changes: Tracing Four Key Shifts in Maternal Nutrition and Weight, 1950-2010

*Associating gestational weight gain with the risk of toxemia: the parasite theory and the dangers of gaining too much weight during pregnancy*

Associating maternal diets with infant characteristics extends beyond nineteenth-century medical history. What a woman eats when she is pregnant has been linked to different fetal outcomes like sex, birthmarks, and even personality traits. However, in the 1950s, what a woman ate, or maternal nutrition, did not figure as a significant health factor. What mattered more during the 1950s across the United States and the United Kingdom was how much a woman gained each week of gestation, not what she ate. There are two key reasons for this strong emphasis on weight gain and the insignificant attention to maternal nutrition.

During the 1950s the comprehensive medical theory for both the US and UK was that gestational weight gain, or weight gained during pregnancy, indicated a much more serious illness. Doctors and scientists at the time thought that rapid weight gain over one or two weeks of pregnancy was associated with one of the toxemias of pregnancy called preeclampsia. Preeclampsia if undetected during pregnancy can develop into eclampsia. Eclampsia can cause

seizures in pregnant women, early delivery, and hemorrhaging, all of which result in a high risk of infant and maternal mortality. Preeclampsia was, and remains, a serious pregnancy complication.

From one doctor's testimony in the 1950s, he said that during his training in medical school if a woman gained two pounds per week they thought she "was about to die" (Brewer 1969). There was a high risk and fear associated with weight gain during pregnancy, which resulted in strict weight gain recommendations. As a result of fear and widespread anxiety related to gestational weigh gain and toxemia, both research and health professionals promoted weight restriction and weight monitoring for pregnant women in the US and UK. The scientific consensus was that if women's weight was restricted and monitored during pregnancy, then rates of maternal mortality would decrease (Bell 2010). This association between preeclampsia and weight gain resulted in a proliferation of medically recommended diets and prescribed diuretics during pregnancy.[33]

Doctors and scientists from the 1950s believed that weight restriction through calorie control and diuretics were safe and healthy because of the "perfect parasite" theory. The "perfect parasite" theory claimed that no matter what a pregnant woman eats, and no matter the restriction on her weight, the fetus would be able to supplement or take what it needs from the mother's body (Susser and Stein 1994).[34] If the mother had a protein deficiency, the fetus would take

[33] However, we now know that preeclampsia is not caused by rapid weight gain, and that rapid weight gain does not mean women have preeclampsia. Rather, spikes in blood pressure and other pregnancy complications are used to currently diagnose preeclampsia.

[34] The parasite theory does not have a clear intellectual history. Most narratives are found in child maternal educational books as a way to situate the history of diet restriction during pregnancy. For instance the educational text titled *William's Essentials of Nutrition and Diet Therapy* by Schlenker and Gilbert defines the parasite theory in its introduction to the development of diet therapy during pregnancy (2014). The same text also draws from the fetal origins hypothesis to situate the approach to dietary therapies during pregnancy in the present. In addition, the parasite theory came hand in hand with the "maternal instinct theory," which assumed that women would instinctively know what to eat based on an unexplained connection to the fetus (Schlenker and Gilbert 2014).

certain amino acids from her blood. The parasitic like absorption of maternal nutrients appears in other contexts. For instance, forensic anthropologists can identify when a woman was pregnant by analyzing samples of hair. Women's hair analysis from Egyptian tombs reflect highs and lows of nitrogen and amino acids. The sharp lows in the chemicals reflect a period of gestation during which a growing fetus absorbed nutrients from the pregnant body.

As a result of the "parasite theory", physicians promoted restricting women's weight during pregnancy. They did not think this would harm the growing fetus because the fetus would naturally absorb its nutritional needs. The perfect parasite notion neutralized any fear or risk between weight restriction and fetal growth. The fetus as a perfect parasite could protect itself from the effects of weight and calorie restriction, which were intended to prevent the main pregnancy complication of toxemia. The parasite notion along with the association of toxemia with weight gain helped frame weight restriction and dieting as healthy forms of prevention in 1950's US and UK. The parasite theory and weight restriction characterized approaches to maternal nutrition and weight during the mid-century in the US and UK.

*Historical Interlude: Post WWII context*

To historically and culturally situate the medical approach to weight restriction during pregnancy I will review some key events that were also taking place at this time. Scholars characterize the time period following the end of World War II, as a time of rebuilding for both the US and the UK. The most influential policy that followed WWII was the 1946 National Health Service Act in the UK. The act states that the National Health Service or the NHS was intended to "secure improvement in the Physical and mental health of the people of England and

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Through the parasite theory, the fetus would communicate to its host to eat particular foods, which was understood as the "maternal instinct." There is still a lot to unpack there and I plan to explore it further in the book project.

Wales and the prevention, diagnosis and treatment of illness" (NHS ACT 1946). The health services were provided to the public free of charge and it also reorganized the administrative management of hospitals under regional board of governors (Warren 2000). In the same year the National Insurance Act was established. The act legally made previsions for welfare services for the sick, unemployed, women in maternity, and people of old age. Within a year of establishing the NHS the British government also legislated new laws for the professionalization of nurses and midwives.

Likewise, in the 1950s, the United States developed professional organizations dedicated to the fields of health and medicine. However, it did not involve nurses or midwives, but obstetrics and gynecologists. In 1951 the American College of Obstetricians and Gynecologists (ACOG) was established, and it was and still is responsible for developing standards of care for pregnant women. Unlike the UK, the US reflected a growth in private obstetric clinics and private insurance programs. In the 1950s, the establishment of insurance was made possible only for the middle class and not equally for all (Ward and Warren, 2006).

However, the 1960s mark a significant shift in American and British history. A social awakening related to civil rights and women's rights caused civil unrest and protest against the Vietnam War. In 1963, John F. Kennedy established the Maternal and Child Health and Mental Retardation Planning Act of 1963. The act established prenatal care services for "high-risk mothers in low-income rural and urban areas," which included medical care, nutrition social services, and education under the Maternal and Infant Care (MIC) program (Ward and Warren 2006:117). Whereas the UK was continuing to develop their National Health System, the MIC was the only comprehensive form of healthcare provided to the pregnant American population.

However, one key aspect remained the same for both the US and UK: maternal and infant mortality continued to rise. Further, efforts to that focused on weight restriction were not improving maternal death from preeclampsia. Therefore, much of the research and approaches to maternal nutrition and weight started to shift in the 1960s and 1970s.

*Associating maternal nutrition and weight with the risk of delayed development and infant morality: The dangers of weight restriction and small babies*

By the 1970s, the overall position in the US and UK was that weight restriction could cause low birth weight and adverse health outcomes, therefore weight restriction should not be recommended to women during pregnancy. Changing common beliefs about the benefits of weight restriction was difficult and required extensive research. To change the public and medical sentiment around weight restriction and maternal nutrition, studies started returning to the relationship between weight restriction and small babies. I write returning because the relationship between weight and small babies was an existing idea that studies in the 1800s explored.

In 1803 a medical scientists named Brunninghausen argued that there was an association between weight restriction during pregnancy and small babies. The idea was that the fetus ate whatever the mother ate and therefore careful attention to the mother's diet was fundamental to pregnancy. This was in direct contrast to the "perfect parasite" theory.

The return to studying weight, nutrition, and small babies is apparent in one 1933 study by Edward Mallaby. He argued, "nutrition is the most important of all environmental factors in childbearing whether the problem be considered from the point of view of the mother or that of the offspring" (1933:2). Drawing from this text, Frank Hytten and Isabella Leitch's *The Physiology of Human Pregnancy* (1964) became a hallmark for the shifting views on maternal

nutrition, weight, and small babies. This book, as one of my key informants claimed, was the "bible" for anyone working on pregnancy and nutrition (2/26/15 Interview with BA). The authors, Frank Hytten and Isabella Leitch, were from the UK and they did most of their research in the UK. Hytten and Leitch's international influence on the field of maternal nutrition reflects the ways in which the scientists in the US and the UK drew from each other's research to inform contemporary health recommendations. This research, along with other studies that confirmed that weight restriction did not prevent toxemia, facilitated the end of practicing weight restriction during pregnancy.

Another study that helped shift perspectives on weight restriction during pregnancy was the Motherwell study in Scotland. The Motherwell study is still regarded in the field of maternal nutrition as a key example for the design and implementation of large-scale nutritional interventions during pregnancy. Dr. Grieve, the main obstetrician in the town of Motherwell, recruited thousands of women from the town of Motherwell for scientific experimentation. Dr. Grieve designed a unique nutritional intervention for pregnant women. The women in his study were not allowed to eat bread, potatoes, prunes, plums, bananas, canned fruit, nuts, or dates, and they were not allowed to smoke (unlike other women at the time). The diet also limited milk consumption to 10 ounces/day and more importantly all the women in the study were required to eat one pound of red meat each day (Grieve 1974). Dr. Grieve also gave all the women strict weight gain limitations. Grieve implemented his "program" from 1960-1976.[35]

[35] Dr.Grieve's study reflects the ways in which science, and, in this case, ideas about health, nutrition, and pregnancy, are based on cultural and somewhat arbitrary ideas. Dr. Grieve's diet was developed out of his whimsical ideas about nutrition and health. His ability to produce scientific knowledge that was seen as novel, innovative, and systematic at the time was based on the fact that he was an influential, trusted, white man in the town of Motherwell, Scotland. He did not perform any kind of informed consent, and he did not seek any external ethical review from the town. His position of authority facilitated his intervention onto women's bodies and diets for over fifteen years. Regardless of ethical concerns, the scientific community gathered significant results from the Motherwell study to inform a new direction in maternal nutrition and weight.

The study found that although women were weight restricted, and had restrictive diets, they still had better health outcomes than neighboring towns. These results contributed to changing ideas about the benefits and health concerns around weight restriction during pregnancy. The complex results of the Motherwell study reflected a more dynamic relationship between weight restriction and "good" health outcomes. One explanation is that weight restriction due to war, famine, or poverty, was not the same kind of weight restriction due to nutritional interventions. Other contemporary studies on pregnant women during times of war and famine found adverse health outcomes related to poor nutrition and underweight women (Lumey et al. 2007). The research on famine, maternal nutrition, and weight found that the children of the women had higher rates of infant and maternal mortality, and that the babies were smaller. The research on pregnancy during times of war and famine helped support the idea that poor maternal nutrition and weight restriction during pregnancy was harmful to fetal development.

The Motherwell study also helped us understand how maternal weight was associated with fetal outcomes but in different ways. The women who experienced famine had very different living conditions compared to women enrolled in the Motherwell study. Further and more complicated is the idea that the women who were enrolled in the study received extra prenatal care as a result of the intense surveillance on their daily eating habits. The variety of outcomes from limiting weight gain during pregnancy reflected a nuance to nutritional interventions during pregnancy that were not well understood at the time.

Two aspects emerged as somewhat competing ideas on the approach to maternal nutrition and weight. The study found that the women in the Motherwell study did have smaller babies compared to woman and infants in neighboring towns. Therefore, scientific consensus further

developed around the idea that restricting weight affected birth weight, or the baby's weight. But the women in the study still had better rates of infant and maternal mortality compared to other women in the area. Therefore, even though the study reflected a link between restricting weight during pregnancy and higher instances of small babies, the study does not reflect a straightforward link between weight restriction and maternal mortality. Put another way, maternal weight both does and does not directly affect birth weight and infant mortality. In the 1970s, maternal weight was associated with both negative and positive outcomes depending on the context and living conditions of the mother. I argue in the rest of the ethnography that the influence of context and living conditions remains a significant factor even in our contemporary examinations of epigenetics and pregnancy.

As a result of many different studies on maternal nutrition and weight the Institute of Medicine and the National Academy of Science in the US published a report titled *Maternal Nutrition and the Course of Pregnancy,* in 1970.[36] The report aimed at explaining the scientific understanding regarding rising neonatal and infant mortality rates. One aspect that emerged as a central focus in the 1970 IOM report was birth weight. To acknowledge or get around the different studies that challenged the possibility of drawing causal links between birth weight and infant/maternal mortality, the IOM report encourages the classification of birth weight as a "surrogate outcome." A surrogate outcome is an end result that may be correlated with, for instance, maternal nutrition and fetal/infant survival but necessarily directly or causally (Rush

[36] One other study that was also influential in the conversation of maternal nutrition and health, among many other research topics was the US Collaborative Perinatal Project (CPP). The CPP study was implemented during 1950's and 1960's, and it focused on ways to improve pregnancy through different approaches. The CPP study concluded that along with famine and war other factors also contribute to infant and maternal morality (Rush 2001). Namely, the project reflected how negative health outcomes during famine did not just have to do with weight, diets, or calorie intake, but rather that the social conditions during pregnancy played a role in child maternal health. Both the Motherwell study and the Collaborative Perinatal Project influenced the design and implementation of future studies about pregnancy and maternal nutrition. See J.B.Hardy's article from 2003 titled, The collaborative perinatal project: lessons and legacy. Annals of Epidemiology. Vol.13. issue5:303-311.

2001). Birth weight is used as a "surrogate" because, as we learned from the Motherwell study, maternal nutrition and fetal/infant survival cannot be directly related to each other. Surrogate categories, or indirect measurements, were fundamental to the changing trends in maternal nutrition and weight during pregnancy throughout the second half of the twentieth century.

In developing birth weight as a "surrogate outcome" the report also drew a connection between maternal pre-pregnancy weight gain and birth weight. The 1970s report stated that women's weight before pregnancy had more of an effect on birth weight no matter how much she gained during pregnancy.[37] At this moment in time both the US and UK emphasized the importance of pre-pregnancy weight in relation to birth weight. In fact, the UK still emphasizes pre-pregnancy weight in their 2010 pregnancy recommendations. The most recent NICE guidelines state that pre-pregnancy weight is a better indicator of pregnancy complications. However, the current US recommendations are different now than they were in the 1970s, which I highlight below. It is at this juncture that the US and UK diverge in their approaches to maternal weight and nutrition.

Once scientific consensus was reached around the dangers of weight restriction, practices of routinely weighing women during pregnancy began to change, but only in certain places. As a result of these findings from the 1970s, British doctors and midwives eventually stopped routinely weighing women and monitoring weight during pregnancy. A veteran midwife from the UK, reflecting on her early days working with pregnant women, explained to me: "By the late 1980s they started throwing the scales out of the [clinic] room."[38]

However, instead of relying less on weight as an indicator of health, the US strengthened their support around measuring and targeting gestational weight gain. Although the 1970 IOM

[37] The exact quote is "higher pre-pregnancy maternal weight reduced the impact of gestational weight gain (GWG) on birth weight" (IOM 1970).
[38] AN 3/10/14

acknowledged that pre-pregnancy weight had more of an effect on birth weight than the weight gained during pregnancy, the IOM made unique recommendation. To ensure the prevention of low birth weight, the 1970s IOM report stated that women should gain between 20-24 pounds during their pregnancy. The numerical measurement that the 1970s IOM report established was a key step in a new direction for the US. The formal establishment of recommending a minimum amount of gestational weight gain is a fundamental turning point in the history of maternal weight gain and nutrition policies in the US and UK.

*Historical Interlude: Impacts on maternal weight and nutrition from research and politics after the 1970s*

During the late 1960s and early 1970s the US made some progressive policies around social and healthcare services. Lyndon B. Johnson declared a "War on Poverty," which resulted in what is today known as Medicaid. In addition, as a result of the various studies like the Motherwell study in the UK and the 1970s IOM report, the US established the Women, Infants, and Children Program (WIC) (Rush 2009). The WIC program emphasized maternal nutrition during pregnancy and in early child development. As a result of WIC, and other health and welfare programs established in the 1970s, rates of infant mortality and low birth weight improved (Ward and Warren 2006).

However, the promotion of welfare services dramatically changed in the 1980s. In the midst of an economic recession and a resurgence of social and political conservatism led by Ronald Regan, Medicaid and other social services were cut. A significant portion of women that were covered by Medicaid during pregnancy in 1975,were no longer covered in the 1980s (Ward

and Warren 2006). The antiabortion debate also grew in strength and there was a general movement to cut government spending on reproductive health.[39]

The UK also saw increased health disparities and inequalities in the 1980s among their low socio-economic working class. The 1970s left the UK in an "ungovernable" state with national budgets and democratic processes in decline (Collinicos 2015). Both the US and UK were recovering from the Cold War. Margaret Thatcher took over in the late 1970s and slashed pensions, welfare services, and any other services that were deemed socialist. Thatcherism, or the new conservative right in the UK, grew in strength during the 1980s and into the 1990s. Thatcherism moved to centralize the NHS, and in turn embrace privatization of business and deregulation of trade (Collinicos 2015).

Although social conservatism thrived in the 1980s in both US and UK national contexts, studies continued to reflect that infant mortality was in fact a social issue. The publication of the IOM report in 1988 on prenatal care argued that infant mortality should be approached as a "social issue with biological manifestations" (Ward and Warren 2006:121). Manifesting the idea that infant mortality was a social issue in research and policy proved, and still proves, to be difficult.

While some research highlighted how the social matters and how stress and poverty affect our bodies and our babies, other research trends worked at undermining that message. For instance, the position that infant mortality is a social issue becomes undermined by new research in the 1990s. The Latina Paradox emerged as a popular public health idea in the 1990s, and it assumes that recently migrated women of low-socioeconomic status have protective advantage

[39] Rosalind Petchesky's book titled *Abortion and Women's Choice*, she argues that abortion continues to represent the "fulcrum of a much broader ideological struggle in which the very meaning of the family, the state, motherhood, and young women's sexuality are contested" (1990:xi). See also, the book *Dangerous Pregnancies* by Leslie Regan published in 1970.

against infant and maternal mortality. If the "Latina Paradox" holds ground then why should the government invest in social services for poor mothers? In addition, new randomized clinical trials showed that social services had not improved birth outcomes. The results from RCTs in the 1990s justified the cuts to prenatal care services for low-income women.

Tracing the different trends in maternal nutrition and weight shows us how scientific knowledge production does not follow coherent linear lines of inquiry and solutions. As the next section shows, maternal nutrition and weight experience another phase of changes during the 1990s. The 1990s reflect the consequences of the 1970s approach of ensuring that women gained a certain amount of weight during pregnancy. The next phase of maternal weight and nutrition takes a slightly different tone. Maternal weight is still associated with birth weight, but low birth weight no longer becomes the problem.

*Associating gestational weight with the risk of overweight babies: the impact of the obesity epidemic on maternal nutrition*

In the twenty years since the last significant shift in approaching maternal nutrition and weight, the demographics of populations changed in the US and UK, and individual bodies changed. Immigrant populations from Latin America and Asia continued to grow in the US, and in the UK there was movement from different African countries, the Caribbean, and Southeast Asia. In addition, the debates around obesity during pregnancy were instituted into national discourse and health policy.

In 1990 the Institute of Medicine in the US published another report titled *Nutrition During Pregnancy*. The report gathered research from different places to create new guidelines

for maternal nutrition and weight, which they now referred to as maternal nutritional status (IOM 1990). The justification for changing the guidelines for maternal weight in the US came from data that reflected significant increases in gestational weight gain. In the 1970 report, the recommendations gave a minimum gestational weight gain in pounds to prevent low birth weights. However, in 1990 it was apparent that telling women in the US to gain at least 24 pounds during pregnancy produced some unintended consequences. The report states that recommendations for "gestational weight gain has nearly doubled during the past 50 years – from 15lbs in the 1930's to a range of 25-35 lbs in the 1980's" (IOM 1990: 53). In addition, the 1990 report notes that the demographics of the population changed. The report claimed that since the 1970s, white women gained more weight during their pregnancy and had larger babies, and so did black women, but only slightly. There was no data for any other ethnic or racial group of women.

As mentioned above, the previous phase in maternal nutrition emphasized the risks and dangers around restricting weight gain during pregnancy. Small babies were the key issue, and birth weight became the key standard in evaluating fetal growth and maternal nutrition. The shift in the 1990s is marked by a different standard. In the 1990s the US established gestational weight gain as a key standard for understanding the relationship between maternal nutritional status and birth weight. To create a more manageable and mobile standard the 1990 committee narrowed down the definition of gestational weight gain to three main points: " weight just before delivery minus weight just before conception; total weight gain minus the infant's birth weight; rate per week, weight gained over a specified period divided by the duration of that period in weeks" (1990:13). The clarified definitions came from studies that focused specifically on the effects of weight gain on the mother and the child. The studies claimed that gestational

weight gain was fundamental to understanding child mortality, morbidity, and physical and mental performance.

In the US gestational weight gain became directly related to "fetal and infant mortality [and] increased risk of giving birth to a growth-retarded infant" (1990:30). In addition, the committee stated that the amount of energy in-take, or calories, is directly related to gestational weight gain. The calorie in calorie out approach to nutrition dominates the intervention at the SmartStart trial in the US. Even though alternative notions of metabolism are currently emerging they have not reached the space of maternal nutrition. Implicit to these notions of energy balance is the framing of the body as a machine that needs energy to expend energy (Martin 1987). In addition, by framing gestational weight gain in terms of calories, the nutritional substance of food becomes standardized into quantifiable units of calories. Foods provide calories, and women's behavior during pregnancy needs to be surveyed in order to make sure that she gained a "healthy" amount of weight during pregnancy.[40]

The new approach to maternal nutrition places gestational weight gain as the main measurable outcome, but only in the US. Gestational weight gain is not a key standard in the UK. One practical issue for the lack of standardization in the UK is the fact that the UK does not routinely weigh pregnant women. To establish gestational weight gain as a reliable measure doctors and scientists were required to regularly collect and record weight during pregnancy. Over the second half of the 20th century gestational weight gain becomes a significant and

[40] As Mol and Law (2002) argue, the calorie is a unit of analysis based on social relations, and requires laboratory practices, nutritional science, thermodynamics, and infrastructures like nutritional labeling (Dumit and de Laet 2014). Basing gestational weight gain on the understanding of calories as a dependable unit of analysis reflects a larger layering and building of standards from other standardized units. The focus on energy intake and expenditure is a production of extensive scientific and medical coordination, and choreography of producing "good science" (Thompson 2013).

fundamental measure for child and maternal health through the constant vigilance and clarification by the scientific community in the United States.

However, one key difference in approaching maternal weight gain emerged in the 1990 report. The report stated a need for measuring and monitoring high gestational weight gain, not just low weight gain. Whereas the 1970s focused on the risks of low weight gain and restriction of weight gain, the 1990 incorporated the focus on the risks of women gaining too much during pregnancy. Since the common understanding in the US was that weight gain during pregnancy correlated with birth weight, high weight gain resulted in large babies, and low weight gain resulted in small babies. Following this line of inquiry the experts on the 1990 IOM committee decided to define the standard for an ideal or "favorable" birth weight as 6-8 lbs (1990: 4).[41]

To assess gestational weight gain the 1990 report recommended routinely weighing women and the monitoring of body mass index, based on height and weight measurements. The 1990 report defines BMI as "a better indicator of maternal nutritional status than is weight alone" (1990:5). Like in the 1970s IOM report, body mass categories were not well established yet. The 1990 report used body mass categories from the 1959 Metropolitan life insurance company to define gestational weight gain recommendations. The body mass data that was collected by the Metropolitan insurance company were not based on any diverse sample size, nor did they include pregnant women. Overall, the recommendations for gestational weight gain in pounds, and based on BMI included 28-40lbs for low BMI, 25-30lbs for normal BMI, and 15-25lbs for high BMI (1990: 10). Therefore, the new approach to maternal nutritional status stratifies the amount of

[41] The committee defined a "healthy" or ideal birth weight in order to prevent any pregnancy complications related to macrosomia. Macroscomia is defined as having a baby that is larger than the IOM recommendations of a "favorable" birth weight. Any baby larger than eight pounds is classified as having macrosomia. One of the health risks related to giving birth to a large baby included, emergency caesarean sections.

healthy weight gain for women based on their pre-pregnancy BMI. So if a woman is classified as having a low BMI, then it is recommended she gain 28-40 pounds.

Anthropological and sociological studies of science emphasize that the making of standards are pregnant with meaning. Standards, like how much a baby should weigh at birth, or how much a woman should gain during pregnancy, characterize aspects of a social world (Bowker and Star 1999), and a shared reality (Busch 2011) The production of standards, related to gestational weight gain and birth weight are also crucial to the production of scientific knowledge (Timmermans and Berg 2003, Latour 1987). In tracing the narrative of maternal nutrition and weight I show how standards have been vital to the health recommendations and research questions regarding what a woman should eat during pregnancy and how much she should weigh. Emphasizing the role that standards play in how science changes renders the work that standards do visible and facilitates the accountability of the meaning and making of standards (Lampland and Star 2009).

In a chapter by Joseph Dumit and Marianne de Laet, titled "Curves to Bodies: The Material Life of Graphs", the authors show how graphs, recommendations, and standards all play a vital role in shaping bodies (2014). They analyze two key examples regarding recommended caloric intake for men and women, and the growth charts for infants. In their analysis of caloric tables differentiated by sex they problematize the ways in which male bodies set the standard for what is counted as the norm, and therefore claim that women's caloric intake should be less. Despite evidence showing that across the board women and men consume comparable amount of calories, the tables represent a division between the sexes (Dumit and de Laet, 2014). This division embodies the social construction of both sex and gender. Similarly, in this chapter I show how the standard recommendations for how much women should weigh during pregnancy

not only changes over time but is different for pregnant bodies. The tables and diagrams in the IOM reports on maternal nutrition and weight also shape pregnant bodies.

In addition, their examination of growth charts for babies underlines how "healthy" growth is different in different places and changes over time (Dumit and de Laet, 2014). More importantly they state, "the charts have agency, as commanding parts of these babies' lifeworlds; [the growth charts] speak and order, imposing a will to act on those involved the infants' care" (Dumit and de Laet, 2014:80). Drawing from STS literature the authors bring attention to how charts, standards, and recommendations have agency in shaping not only bodies, but also behaviors and approaches to child rearing, eating, cooking, and social relations. Likewise, the recommendations imposed on pregnant women to gain certain amounts of weight in different contexts also shape the ways in which they eat, behave, and perceive risk.

However, I focus on the distinct ways in which standards and recommendations command order not just on bodies and behaviors, but also on risk. The causal framing of gestational weight gain with particular health risks is problematic. For instance, there are no clear causal correlations between maternal weight gain and birth weight. Women who gained too much weight, or higher than the IOM recommendations, can still deliver a small babies. This phenomenon is found among low and high levels of socioeconomic status in African American women (Collins et al., 2004). Moreover, women who are underweight and have low gestational weight gain can have larger babies (Boney et al., 2005). Gestational diabetes is just one factor that contributes to the paradoxical relationship between maternal weight and birth weight.[42]

[42] Gestational diabetes is the manifestation of diabetes during pregnancy. It is currently diagnosed through the application of a glucose tolerance test at around 24 or 28 weeks gestation, depending on the local standard protocol. No one knows why or how women develop gestational diabetes, but it is associated with weight and nutrition. In addition, gestational diabetes may disappear after birth, and or return later in life. It is possible that women who gain rapid amounts of weight in their first trimester or start pregnancy at a higher BMI will develop gestational diabetes, but it is also possible for women who start pregnancy at a "normal weight", and do not gain too much weight can also develop gestational diabetes. The studies that I examine fall on two sides of this debate. The US study claims

Further, these recommendations that change over time regarding gestational weight gain are constantly in flux depending on the production of new scientific literature. The implication of causally framing recommendations on gestational weight gain to health outcomes not only shapes pregnant bodies, but it also frames how we understand risk and therefore medical approaches to maternal nutrition and weight. The problems with the 1990 recommendations emerged over the next twenty years, when the most recent and final phase of maternal nutrition and weight was established in 2009 and 2010.

*Historical Interlude: The Obesity Epidemic and the Emergence of Epigenetics (1990-2010)*

A significant change that occurred in between 1990 and 2010 was the public and international acknowledgement of the obesity epidemic. During this time period the number of headlines of newspaper articles with the words "obesity epidemic" exponentially increased. Abigail Saguy writes that from 1990 to 2000 the number of titles literally increased from zero to sixty (Saguy 2013:46). Saguy's book entitled *What's Wrong with Fat*, draws from international health policy reports, media representations and scientific trends to trace the growing public concern over the obesity epidemic.

In 2000 the World Health Organization (WHO) published a report entitled "Obesity: Preventing and Managing the Global Epidemic." The report claimed that obesity was replacing "traditional" global health problems like infectious diseases and under nutrition. The report introduces a brief history of obesity from the committee's perspective, which highlights the contradiction between increased efforts and research on diets, exercise, and surgery and the increasing rates of obesity. The growth of the billion-dollar diet industry also marks a significant

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that limiting weight can prevent instances of gestational diabetes, and the UK study claims that limiting weight does not prevent gestational diabetes, but that reducing saturated fats, and sugars can prevent gestational diabetes.

change in the two decades during which obesity became a public health concern. As the WHO highlights, there are more diets, nutritional programs, exercise programs, and research on weight and weight management then ever before in history; yet, the rates of obesity are still higher than ever before in the Global North and increasingly in the Global South.

Along with the growing concern around obesity, there was another shift developing at the same time: the epigenetic paradigm. To expand on how epigenetic science started emerging and influencing maternal nutrition I will focus on a few key scientific studies, historical accounts, and interviews with experts in the field of maternal health and nutrition. I find that the emerging field of epigenetics entered the space of obesity during pregnancy and maternal nutrition around the late 1980s with the publications of David Barker's studies on maternal nutrition.

David Barker was a British scientists and physician who examined various nutritional studies from the past. His retrospective analysis emphasized the long-term effects of maternal nutrition. For instance, Barker went back to the Motherwell Study mentioned above. He found that the children of the women in the Motherwell study had higher rates of cardiovascular disease. Barker also completed retrospective studies on different population records in India, Holland, and the UK. In the UK he explored studies on women from Hertfordshire. The records at Hertfordshire showed that babies with low birth weight also had higher risks of cardiovascular disease, similar to the Motherwell study. These findings are corroborated with many other studies like the Dutch Hunger Winter group in Amsterdam. All of his work contributed to the idea that "exposure of pregnant mothers to famine left a legacy of chronic disease in their children" (Barker, 2013: 347).

The causal mechanisms of how weight restriction during pregnancy, due to interventions like the Motherwell Study or environmental conditions like famines, affects cardiovascular

disease risk in future generations is not very well understood. Scientists understand that weight restriction is related to lower birth weights, and that lower birth weights are related to cardiovascular disease. Although linkages appear in the retrospective studies between women's diets during pregnancy and chronic disease in future generations, there are no current evidence-based studies that have found causal links between nutrition in pregnancy and chronic disease in humans. Overall, Barker published several articles explaining his theory of fetal origins through fetal programming, and the significance of maternal nutrition on the future risk of disease for the developing fetus, child, and adult (Barker et al., 1993). Many other scholars credit Barker with the fetal origins hypothesis; however, as Almond and Currie comment, Barker's popularity can also be attributed to his "proselytizing zeal that helped make his name synonymous with the fetal origins hypothesis" (2011: 156).

Mainstream media also reflects the impact of epigenetics in relation to maternal nutrition and obesity during pregnancy. In the last few years newspaper articles in the US and UK have titles like "Pre-pregnancy diet 'permanently influences baby's DNA'" (Briggs 2014); and "Increase in severe obesity among expectant women raises concerns" (Cullen 2016). The way that the news articles explain epigenetics focuses on how the "mother's nutrition can leave permanent marks on her child's genome, and on all the cells of the body" (Briggs 2014). Popular journalism emphasizes that maternal nutrition can affect the long-term risks of health in future generations.[43]

[43] See book titled *Evolving Ourselves: How Unnatural Selection and Mutation are Changing Life on Earth, by Juan* Enriquez, Steve Gullans 2015.
See also, P. Dominguez-Salas, Se. E.Moore, M.S. Baker, A.W.Beren et al., "Maternal nutrition at conception modulates DNA Methylation of Human Metastable Epialleles" Nautre Communications. 5(3746). 2014
D.S. Feig, J.Hwee, B.R. Shah, G.L. Booth et al., "Trends in Incidence of Diabetes in Pregnancy and Serious Perinatal Outcomes: A large population based study in Ontario, Canada, 1996-2010." Diabetes Care 37(6):1590-96. 2014.

In addition, experts at my field sites reflected on the influence of the epigenetic paradigm. The principle investigator at the UK study stated, "over the past twenty years or so the Barker hypothesis has grown credence, at first I didn't believe it but now I am completely convinced."[44]The PI in the UK focuses her research on tracing the epigenetic effects of nutrition during pregnancy to future chronic disease, which I explore further in the next chapter. Moreover, collaborators and experts on the US trial also reflected on the growing belief around Barker's work to me. One of the key collaborators on the US trial, an expert in maternal nutrition, told me that when she first read Barker's papers in the late 1980s, she did not believe him. However, now she is also convinced of the significance of maternal nutrition. She reflects that as a researcher on maternal nutrition her work is in high demand because of the epigenetic implications of nutrition as an environmental factor to fetal development. Barker's work on the fetal origins hypothesis was clearly important for the experts at my field sites, and this was significant because it allowed the PIs to design trials that focused specifically on engineering pregnant diets. Drawing from Barker helped justify the dietary interventions, as well as promote a new way of targeting pregnant women during pregnancy. In what follows, I show how the last shift in approaching maternal nutrition drew connections between what pregnant women eat during pregnancy and future risk of obesity in their developing children.

*Associating maternal nutrition and weight with future risk: epigenetics and the (re)production of obesity*

To describe the most recent phase of maternal nutrition and weight I will focus on two key reports. One by the US IOM, published in 2009, and one by the UK NICE, published in

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D.Martinez, T.Pentinat, S.Ribo, C.Daviaud et al., "In Utero Undernutirtion in Male Mice programs liver lipid metabolism in the Second-generation offspring involving altered LZRA DNA methylation. Cell Metabolism 19(6):941-51. 2014.

[44] LP speech 6/18/14

2010. Both of these reports claim that obesity during pregnancy is a public health concern and that more research is needed to understand the effects of obesity during pregnancy longer term. However, there are very different ways of addressing the same problem in the US and UK. In the US weight features as a significant focus, with the intervention and treatment targeting calorie control and gestational weight gain limits. In the UK the guidelines focus on promoting "healthy" eating and nutrition, but do not support counting calories or regularly weighing women. The difference is epistemological. One approach quantifies food into measurable units of energy, and the other focuses on the quality or nutritional substance of food in the form of carbohydrates, sugar, and saturated fat. The epistemological difference is further explored in chapter four.

Overall, I show that this fourth and final phase of maternal nutrition is marked by a focus around what is "healthy" weight gain during pregnancy. Furthermore the final phase of maternal nutrition is marked by potential health risks that can affect the next generation. In both the US and the UK the message of "Eating for two" is no longer an acceptable public health message in the context of the obesity during pregnancy and maternal nutrition.

The 2009 IOM report justifies its approach to maternal nutrition and weight with the following excerpt:

> It has become clear that heavier women could gain less weight and still deliver an infant of good size. Since [the 1990 report] the obesity epidemic has not spared women of reproductive age. In our population today, more women of reproductive age are severely obese (obesity class III; 8 percent) than are underweight (3 percent), and their short- and long-term health has become a concern in addition to the size of the infant at birth. Clearly the time had come to reexamine the guidelines for weight gain during pregnancy (IOM 2009: ix).

The justification and framing of this report relies heavily on the obesity epidemic. Highlighting how obesity "has not spared women of reproductive age" points to a significant shift in the ways

in which obesity and reproduction are associated and framed in relation to each other. In past framings of maternal nutrition and weight, obesity did not play a significant role. However, the issue of obesity during pregnancy has come to characterize the 2009 weight gain recommendations for women in the US. Further, they start off the first sentence by stating their main intervention in maternal nutrition and weight that "heavier" women do not need to gain as much weight as they were previously advised by the 1990 recommendations. Not only can heavier women "afford" to lose less, but the actual guidelines claim that women with a BMI over 30 do not need to gain *any* weight during pregnancy. This new shift might appear familiar to the 1950s promotion of weight restriction during pregnancy, but it is occurring in a completely new context and in a completely new scientific paradigm.

The new scientific paradigm is characterized by the diverse disciplines represented on the 2009 IOM committee. For the first time in history, experts in the fields of nutrition, genomics, family medicine, social policy, epidemiology, maternal/fetal medicine, and obstetrics and gynecology all came together to discuss the topic of maternal nutritional status. A key difference in the disciplinary composition of the 2009 committee is that it includes experts in genomics. In 1990 the field of genomics was not formally recognized, consulted, or incorporated in the committee. Now, genomics, and not genetics, influences the new approach to maternal nutrition and weight.[45]

Another development pertains to the BMI categories. The 2009 report draws from the updated BMI categories established by the WHO in 2000. The new categories contain four categories instead of the three categories developed by the Metropolitan life Insurance group. The recommended ranges of gestational weight gain are also based on the new BMI categories.

[45] The World Health Organization defines genetics as the study of heredity, and genomics as the study of genes and their mechanistic functions (WHO 2002). The main difference is that genomics looks genomics explores the relationship or interaction between and among genes to better understand the combined effect on development.

Therefore, maternal nutrition and weight is still defined and approached through gestational weight gain and BMI in the US.

However, in the 2010 NICE report the UK offers different recommendations, but still acknowledges obesity during pregnancy as health issue. The report states "if a pregnant woman is obese this will have a greater influence on her health and the health of her unborn child than the amount of weight she may gain during pregnancy. That is why it is important, when necessary, to help women lose weight before they become pregnant" (NICE 2010:61). The problem is the same, but the approach is different. Women's weight should be targeted before they get pregnant, but not during pregnancy. The NICE guidelines support their recommendations by stating that "the US Institute of Medicine guidelines [are] based on observational data" (2010:20). They go on further to say that there are no evidence-based guidelines in the UK for gestational weight gain.

The 2010 report is titled the *Weight Management Before, During and After Pregnancy*. The title of the report is confusing because although the report tells women to manage their weight before they get pregnant, it does not give gestational weight gain recommendations during pregnancy. The only kind of recommendation that the NICE report gives in regards to "weight management" are nutritional meal plans and portion control. Moreover, the report emphasizes that women should not lose more than half a kilogram of weight each week during pregnancy. The minimum weight limit is difficult to measure since the report also states that there is not enough evidence-based science to support the routine weighing of women during pregnancy. Therefore, women are not routinely weighed during pregnancy unless they have a BMI of over 30 and have been recommended to see a dietician.

The difference in approaching obesity during pregnancy in the US and UK is apparent in the 2009 IOM and 2010 Nice reports. However, both reports address and acknowledge the impact of the obesity epidemic on pregnancy women. The emphasis on obesity during pregnancy characterizes the most recent phase in maternal nutrition and weight management. In addition, I show in the next part how this most recent shift is also directly related to the emergence of epigenetics.

## Part 2: The Emergence of Epigenetics: obesity during pregnancy before and after epigenetics

To examine how the epigenetic paradigm has fundamentally shifted understandings and framings of obesity during pregnancy, I focus on the 1990 and 2009 IOM reports. I compare how these reports link gestational weight gain to risky health outcomes in different ways. The documents that I chose to analyze reflect a time period before the emergence and broad dissemination of epigenetics, in 1990, and after the paradigmatic shift in 2009. By examining these artifacts of scientific development, I argue that the emergence of epigenetics is associated with three key themes of environmental scale, risk, and relationality. Overall, the most recent shift in approaching maternal nutrition and weight is a direct result of the epigenetic paradigm.

In both the 1990 and 2009 IOM reports on maternal nutrition and weight there are two key diagrams that outline causal pathways of obesity during pregnancy from the mother to the fetus and developing child. These diagrams aim at explaining the health outcomes of obesity during pregnancy for the mother, fetus, and child. In this section I approach these diagrams of causation as artifacts of scientific trends in 1990 and 2009. Both diagrams are ideal for comparison because the 2009 report draws directly from the 1990 report to re-create the same

causal diagram, but with more recent scientific information. The changes between the 1990 and 2009 diagrams focus on expanded definitions of the environment and greater risks of maternal nutrition and weight. More importantly the 2009 diagram connects long-term health risks of gestational weight gain to obesity in developing children. The risk or health outcomes of gestational weight gain are now extended beyond the mother and the developing fetus, to include future generations.

The 1990 report drew most of its research from earlier decades before the dissemination of epigenetic literature in maternal nutrition. However, the 2009 IOM diagram reflects research since 1990, which includes epigenetic literature that was disseminated in the 90s, and early 2000s. The significance of both diagrams is that it illustrates the justification for why the IOM recommended interventions upon gestational weight gain in 1990 and also in 2009. Despite new epigenetic information, the 2009 report still justifies a narrow focus on gestational weight gain.

My analysis examines three key themes related to environmental scale, risk, and relationality. I show how the differences between the 1990 and 2009 diagrams of causation reflect an expansion of the environmental scale, a change in longitudinal associations of risk from the mother to future generations, and a difference in the relational framing of the mother and fetus.

*Finding Causation: The 1990 Diagram*

The 1990 report states that its main aim is to find "potential causal relationship" between nutritional interventions, gestational weight gain, maternal factors, and health outcomes for mother and child (1990:4). The 1990 report proposes two similar diagrams, but I only focus on one. The diagram that I focus on describes the specific interventions that the 1990 report

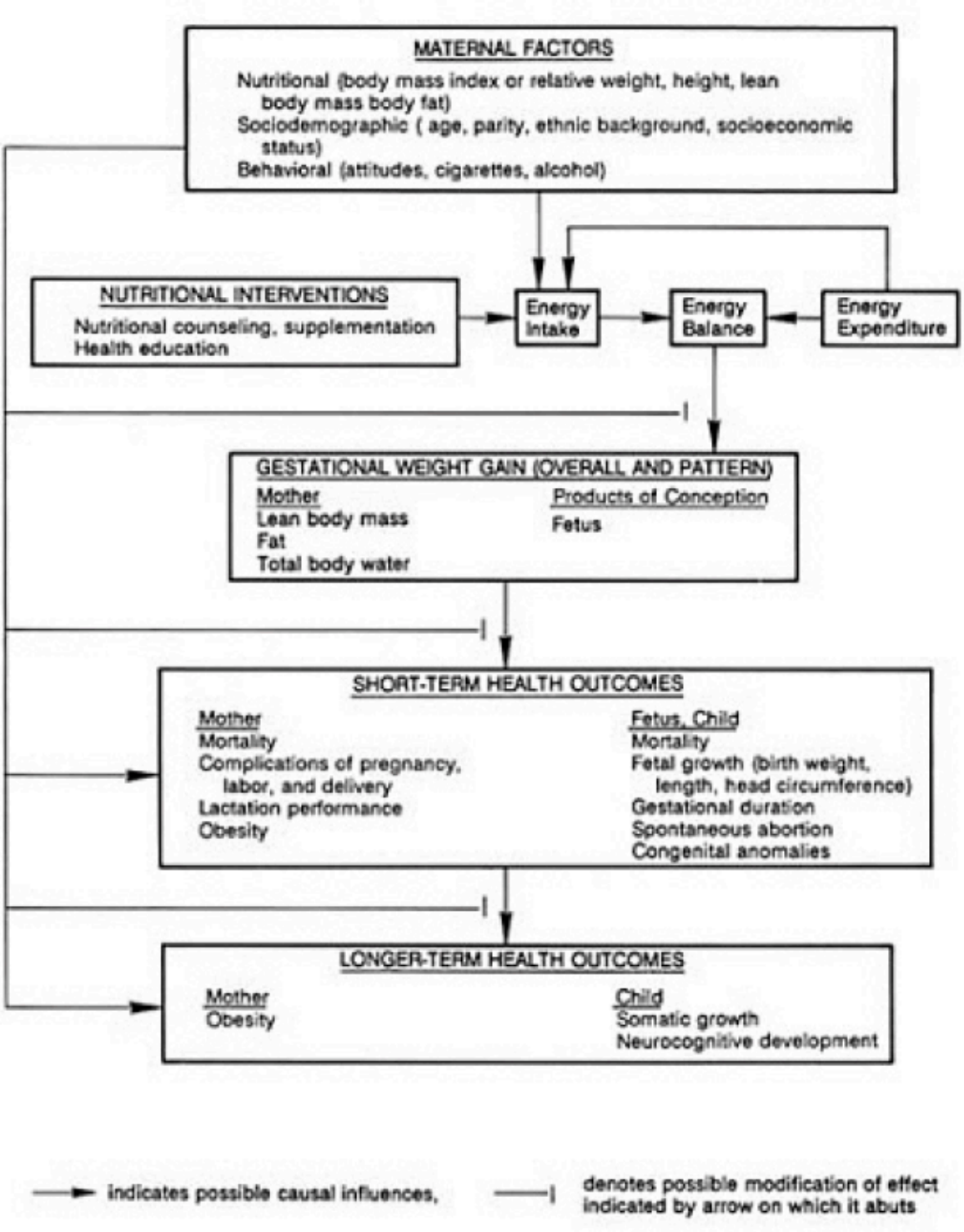
intended to address, and it is titled "Schematic that the 1990 specifically addresses" (1990:33). The diagram that is not included here is a general diagram of causation, which includes a broader list of factors that can cause gestational weight gain, and health outcomes that result from gestational weight gain. The focus in both the general and specific diagrams of causation is gestational weight gain.

The diagram in Figure 1 includes various boxes with text and arrows that represent possible causal influences, and lines with no arrows, which represent effect modifiers. An Effect modifier is an epidemiological term that reflects a relationship between two factors, but not necessarily a causal one. The diagram in Figure 1 is hierarchical with the box on top labeled "Maternal Factors." In the general diagram, not pictured here, there are seven factors including genetics, environment, and prenatal care. The IOM states that the genetic factor does influence gestational weight gain, but does not explain how. In addition, the 1990 report defines genetics as "anything other than height and ethnic background" (1990:33). The role of the environment is also limited in the 1990 report. Environment is defined as anything related to climate, geography, or the natural-built space (1990:33). In Figure 1, the environmental factors and genetic factors are not addressed.

The diagram below focuses specifically on the maternal factors related to gestational weight gain, including nutrition, behavior and "sociodemographics" (1990:33). These maternal factors influence gestational weight gain through energy intake and energy expenditure, or calories in and calories out. The calories in/out are then causally linked to gestational weight gain. The report states that the short-term consequences of gestational weight gain for the mother are "mortality, complications of pregnancy, labor, and delivery, post partum nutritional status, lactation performance" (1990:33). The short-term outcomes for the "fetus/child" that result from

gestational weight gain are “fetal growth, (birth weight, length, head circumference) gestational duration, spontaneous abortion, congenital anomalies, mortality and morbidity” (1990:33). Finally, at the bottom of the diagram it states that the long-term consequences of gestational weight gain for the mother is obesity. However, the long-term consequences for the fetus/child are not the same, and do not include obesity. This is a significant change from the 1990 and 2009 diagrams. Another specific change between the two reports is that the 1990 report does not include any extensive discussion on environmental or genetic factors related to gestational weight gain.

Figure 1. Schematic that the 1990 Specifically Addresses




*Source: Nutrition During Pregnancy* Institute of Medicine 1990.

*Analyzing the 1990 Diagram:*

As I described in the introduction, epigenetic logics include ways of framing the world, and not just mechanistic explanations for the trans-generational inheritance of epigenetic modifications. The three types of logics, or epistemological frameworks, that I examine here are environmental scale, risk, and relationality. For example, I argue that epigenetic science has multiplied and expanded notions of the environment to include different scales from the cellular level to the natural built level.

However, the expansion of environmental scale is not present in the 1990 report, nor is it included in the 1990 diagram pictured above. The 1990 report defines the environment as "climate and geography," and does not associate the environment with gestational weight gain. This definition of the environment does not include many aspects that epidemiologists and epigenetic scientists would include in current conceptualizations of the environment, which are redefined in the 2009 diagram to include natural built environments, behaviors, food, stress, and toxic exposure. Another aspect to highlight is that the concept of the intra-uterine environment is not included in the broad environmental factors. In the rest of the report the term 'intrauterine environment' is referred only in relation to fetal growth. The 1990 diagram is an artifact of the scientific period prior to the emergence and proliferation of the field of epigenetics.

The diagram also does not reflect a clear designation of fetal risk in relation to gestational weight gain. The risk of obesity as a result of gestational weight gain is directed at the mother only and not the "fetus/child." The report states that "[i]n emphasizing gestational weight gain as a potential cause of maternal and fetal outcomes, the subcommittee in no way wishes to impugn its [GWG's] potential value as a *marker* of risk for adverse pregnancy outcomes" (1990:34). The statement reflects a hesitation to frame gestational weight gain as a risk for pregnancy

complications. The ambiguous framing of risk in the 1990 report disappears completely in the 2009 report. With the emergence of epigenetics, and nutritional epigenetics in particular, a woman's diet and weight gain during pregnancy is clearly framed as risky.

One thing to note about these consequences or causal relationships between gestational weight gain and health outcomes for the mother and "fetus/child" is that they are based on research from the 1960's surgeon general report on smoking and health. The committee notes that there are lots of other possible relationships between GWG and birth weight. For instance, some women with large placentas will have large or fast growing fetuses with excess amniotic fluid, and they will appear to gain more weight. The 2009 IOM report is defines this as "reverse causality" because their schematic diagram suggests that increased GWG will cause larger fetal growth, not that large fetuses will cause increased GWG. This is an important aspect that underlines an unspoken fetal/maternal relationship.

In both diagrams the pregnant woman is framed as "mother" and the fetus is framed as the "fetus/child." Although the report does not explicitly state this, it is assumed that there is a causal relationship between the "mother's" energy intake and or nutritional behavior and the size/ growth of the "fetus/child" this relationship is framed as uni-directional. However, as part 1 of this chapter detailed the relationship between maternal nutrition and birth weight is not causal. In addition, the 1990 report does not explain how the fetal/maternal relationship changes or transmits energy across the placenta; or how exactly the placenta mediates the causal relationship between energy in/out and gestational weight gain. The relationality of the fetal/maternal relationship changes and is expanded upon in the 2009 report.

*Finding Causation: The 2009 IOM Diagram*

After the broad dissemination of epigenetic publications in the field of maternal nutrition the 2009 IOM committee drew from multiple studies that found a link between environmental exposure, epigenetic modifications, and the trans-generational inheritance of chronic disease. The diagram in Figure 2 reflects the fetal origins theory or the Barker hypothesis, which states that children from obese pregnant women are at a higher risk of developing obesity because of permanent fetal programming in utero (Barker 1991). This schematic is an artifact of the epigenetic shift in science and the ways it has influenced child maternal health.

For instance, in the 2009 schematic the overarching factors are no longer just the maternal factors outlined in the 1990 report. The recent report includes a box titled "social/built/natural and life-stage environment" at the very top of the diagram. The environment is the overarching category for the rest of the diagram. The definition is expanded to include characteristics of social conditions that are associated with a broader more complex understanding of the "environment."

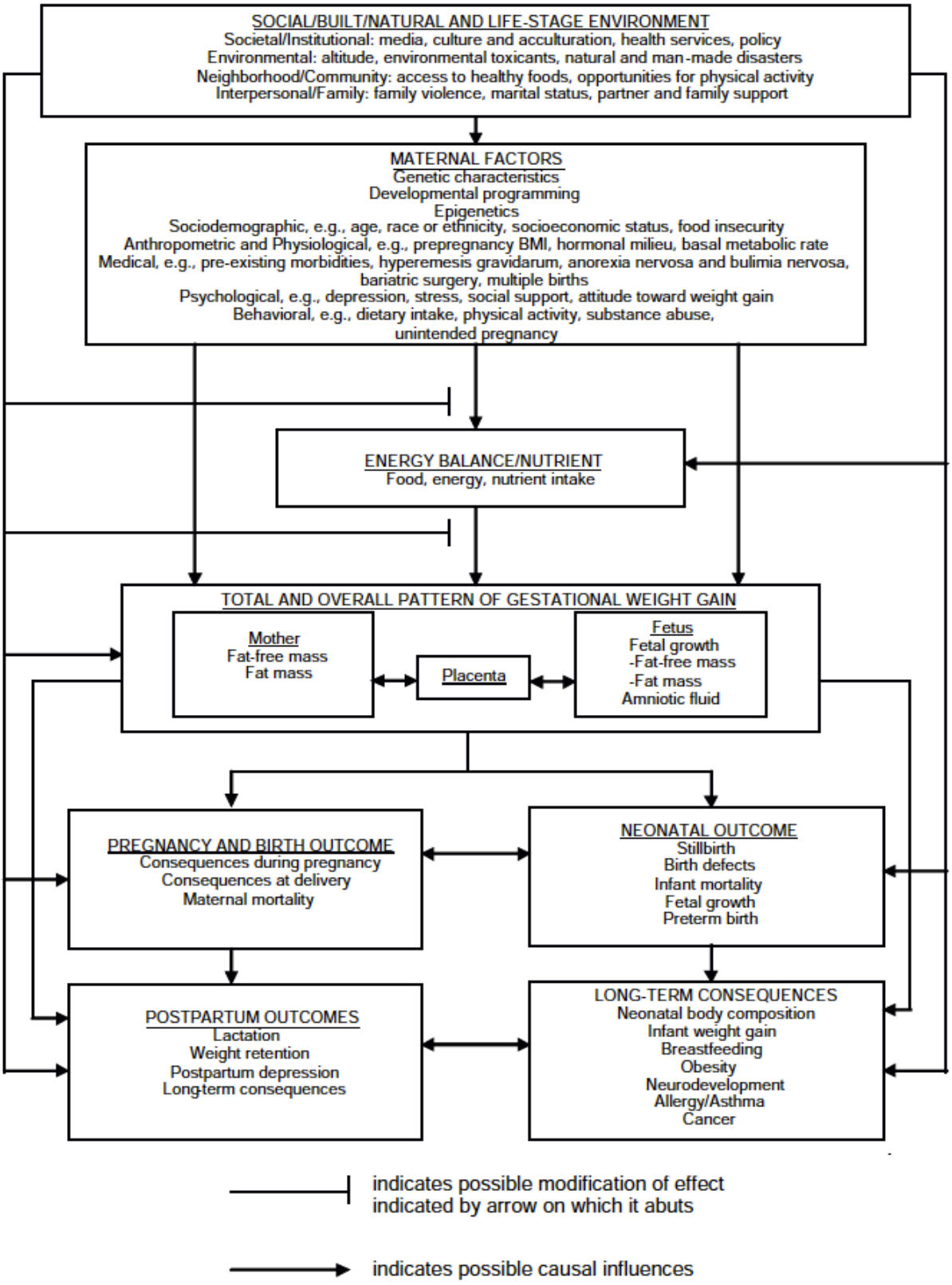
Another significant change is the placement of the environment on the diagram. In the 1990 diagram the environment factor was not included. However, in the 2009 diagram the environment is placed at the top and the maternal factors are underneath. The placement of "the environment" signifies the larger role that it takes in 2009 compared to the 1990. Furthermore, the maternal factors are changed to include "genetic characteristics, developmental programming, and epigenetics" (2009:6). The inclusion of these three fields reflects a fundamental change in scientific paradigms. The description uses the term "epigenetics" and not just genetics.

However, there are some things that remain consistent in the 2009 report as in the 1990 report. Although the environment becomes a focused on gestational weight gain, it is still framed

as under maternal control through the process of calories consumed and expended. The underlying emphasis on maternal control over maternal nutrition, and hence gestational weight gain individualizes epigenetics and the environment. Despite the knowledge that epigenetics has different scales of environment and that these different scales are entangled, the focus remains on gestational weight gain and maternal control.

One descriptive difference between the 1990 and 2009 diagrams are the double-ended arrows. It may seem like a simple detail, but the fact that the arrows are double ended, reflects a larger paradigmatic shift. The double-ended arrows and the entangled arrows open up more pathways. In this way both the environment and fetal/maternal relationship reflect a more dialectical and relational fluidity that did not exists in the 1990 diagram.

Figure 2: Schemata summary of potential determinants and consequences for gestational weight gain, IOM 2009




*Source: Weight Gain During Pregnancy, Reexamining the Guidelines* IOM *2009*

*Analysis of 2009 Diagram:*

The 2009 diagram reflects multiple scales of the environment. It illustrates the environment as an influential factor across pregnant bodies, food, behavior, and neighborhoods. The definition of the environment is expanded to include " culture, acculturation, media, toxins, policy, access to health food, [and] family violence" among many other factors (2009:6). In addition, the diagram draws a causal link between the "environment" and gestational weight gain. In the 1990 diagram the environment was mediated by maternal factors and it did not have such a direct link to outcomes. Before the emergence of epigenetic science the environment did not have a direct link to short and long term fetal outcomes. In the 2009 diagram the environment is connected to short and long term health outcomes for both mother and child, across different scales. The expanded role of the environment in the new diagram is reflective of epigenetic logics.

In addition, the fetal/maternal relationship features more significantly in the 2009 report compared to the 1990 report. I argue that the more dynamic relationship between the fetus, child, mother, and environment reflects the epigenetic logic of relationality. Through epigenetics we can examine the fetal maternal relationship as one that is dialectical and not uni-directional, and one that is vulnerable to environmental factors at different scales. The diagram and the 2009 text itself represent an open "dialogue" between the mother, fetus, and environment- environment both as sociocultural built environment and the intrauterine environment. In the 2009 schematic under a central box titled the "total and overall pattern of GWG" there is the incorporation of the "placenta" as a key player in the relationship between the mother and fetus. The placenta was not mentioned very much in the 1990 report and it was not included in the schematic diagram. In addition, the 2009 IOM report includes an entire chapter related to the "dialogue between the

fetus and the mother" (2009: 71). The relationship between the mother and the fetus is also characterized by a double arrow mediated by the placenta the double ended arrows signify that the direction of "causal influence" can go both ways. In the 1990 schematic there are no double arrows. This conceptual difference in the potential for causal influence to be reversed or to move in more than one direction is also a fundamental change in the notion of "causal pathways."

Along with the more open and dialectic framing of the environmental scale, and relationality between mother, fetus and developing child, there is also an underlying implication of risk. The key difference in risk is that in the 2009 diagram the fetus/developing child is framed as at risk for obesity in the future. The long-term consequences for the developing child include obesity, cancer, asthma, and allergies. All of which are associated with the gestational weight gain of the mother and broader environmental exposures. However, in the case of obesity during pregnancy, risk for the developing child is associated to the maternal control of energy intake and gestational weight gain. The new epigenetic notions that denote a kind of openness and permeability between bodies, placentas, and the environment are still limited to the focus on gestational weight gain. Moreover, the future risk of obesity for children is directly associated with the diets and behaviors of pregnant women in the present. This framing of risk selectively focuses on the future risk without acknowledging the transversality. The epigenetic logic of risk explains that epigenetic changes can across time, space, and bodies.

**Conclusion**

Parts one and two of this chapter both focus on illustrating how scientific and medical approaches to maternal nutrition and weight have changed over time and are different in the US and UK. In part one I trace four key phases that reflect fundamental shifts in maternal nutrition.

The four different approaches that I examine in part one have one main aspect in common: all of the phases focus on associating health risks with gestational weight gain and maternal nutrition.

The first phase is framed by the 1950s promotion of weight restriction during pregnancy and the "perfect parasite" theory. In addition, scientists and medical practitioners during this time associated excessive gestational weight gain with the risk of toxemia. Then I highlight the scientific move away from thinking that gestational weight gain was risky. The second phase focused on how low gestational weight gain was linked to adverse health outcomes for the child namely, delayed development, infant mortality, and low birth weight. The different risks to infant development motivated scientific and medical experts to change their approach and recommendations. As a result women were told to gain a minimum amount of weight to ensure a "healthy" birth weight.

It is during the second phase that the US and the UK diverge in their approaches to maternal nutrition and weight. Following the second phase, the UK did not require a minimum weight nor did they implement routine weighing of pregnant women. However, the US continued to routinely document and record gestational weight gain. The different approaches in the US and UK are further solidified in the 1990s. The third phase examines the1990 IOM report, which claimed further restrictions on gestational weight gain, in the US only. The 1990 IOM report states that in order to avoid excessive weight gain during pregnancy, women should have their BMI calculated at the beginning of pregnancy. The BMI at the beginning of pregnancy indicated how much a woman should gain during her pregnancy.

The UK did not and still does not implement gestational weight gain recommendations. In 2010 the UK published its own guidelines, which were implemented in the UK trial. The guidelines discourage routine weighing of women and dieting during pregnancy. Even when

participants in the UK trial asked the staff about how much weight they should be gaining, the staff members repeatedly responded by saying that the UK did not have any recommendations for gestational weight gain. In chapter four I argue that despite the NICE guidelines in the UK, pregnant participants in the UK were interested in managing their weight gain and were worried about gaining too much weight.

The last and final phase of maternal nutrition and weight is marked by the emergence of the epigenetic paradigm. Due to epigenetics, the fourth phase examined how the risk of obesity is also associated with developing children, adults, and not just with mothers who gain too much weight during pregnancy. Part two picks up on the last shift with the emergence of epigenetics, and provides a closer look at IOM reports that index key changes before and after epigenetics. The main changes that I identify between the 1990 and 2009 IOM reports resulted from the production and dissemination of epigenetic science. Between 1990 and 2009 David Barker's work on fetal programming in the uterine environment emphasized the importance of maternal nutrition and weight. The intense way in which Barker disseminated his work brought about influential changes in maternal nutrition and obesity during pregnancy (Almond and Curie 2001).

Another point to underline in parts one and two is the role of causality in linking health risks with gestational weight gain. The problems with establishing standards or recommendations that imply causal correlations between maternal weight gain and health risks is that this relationship is not causal. Throughout all the phases that I outlined, the relationship between gestational weight gain and different health risks are not linearly correlated. For instance, women who gained too much weight, based on the IOM recommendations, can still deliver a small baby. The high instances of low birth weight among African American women, complicate simple

correlations between gestational weight gain and birth weight. Moreover, instances of gestational diabetes also confound the risks of gestational weight gain. A woman who is classified as normal weight, underweight, overweight, or obese at the beginning of pregnancy can still develop gestational diabetes regardless of her weight gain.

The current emphasis on causality is also indexed by the 1990 and 2009 IOM diagrams that focus on the "causality of obesity during pregnancy." As I show in part two, finding causation between gestational weight gain and future risk reflects the ways in the epigenetic changed the framing of relationality, environment, and risk. Although the 2009 diagram emphasizes the emergence of epigenetics, both the 1990 and 2009 diagrams maintain a focus on causal relationships between gestational weight gain and future health outcomes.

Furthermore, epigenetic science inherently underlines the unpredictable, and non-linear forms of epigenetic modifications and risk that can be trans-generationally inherited. Despite the non-linear relationship between gestational weight gain and birth weight or delayed development, and despite the emergence of the epigenetic paradigm, approaches to maternal nutrition and weight still focus on controlling maternal nutrition (in the UK) and gestational weight (in the US). The causal correlations of the past decades haunt our present understandings of epigenetics and the inheritable risks of obesity during pregnancy. The selective application of epigenetic theories and the over dependence on causal connections are explored in the next two chapters.

## Chapter 3:

## Scales and Contexts of Epigenetics: Which Environments Matter?

On June 18, 2014, Dr. Elizabeth, the principle investigator at the StandUp trial, gave a presentation at a conference organized by the King's Health Partners (KHP). The KHP, which is part of the Academic Health Science Center for London, is funded by the National Health System (NHS) in the UK and is associated with King's College London, the Guy's and St. Thomas' Trust, and the King's College Hospital. Dr. Elizabeth invited me to see what kind of research the KHP was organizing.

Dr. Elizabeth is tall and around her late forties. If she were participating on the clinical trial that she leads, she would be classified as European, or white British. This demographic "code" is one out of the total ten that are used to classify participants in terms of ethnicity. The codes change frequently for each study and most recently the code changed from British to European. Like Dr. Elizabeth, most of the people in attendance at the conference appeared to be "European."

While Dr. Elizabeth, the only woman at her table, sat chatting with her colleagues, I found a place to sit at a different table. I looked through all the materials, noticing the high quality graphics and paper. The picture on the front of what? was a colorful illustration of a genetic sequence at the top and a phrase under the KHP logo that said "pioneering better health for all." This conference was well funded with an elite attendance to match. The keynote speaker of the conference was the CEO of the National Health System, Simon Stevens, and an Oxford Graduate. When Stevens finally came on stage, he announced to everyone that he was late because he had taken the bus to the conference from his office in Westminster, central London. This gave the impression that he did not use a private car service, which is significant

considering the recent budget cuts to the National Health System. Every month a group of organized nurses and doctors would stand outside the hospital protesting the NHS budget cuts.

The conference presentations were organized around the themes about expanding clinical trial recruitment, improving translational research, and bridging the divide between the research prevention efforts and underserved communities. For instance, the Chair of the KHP discussed how important it is for research and prevention efforts to focus on anti-smoking campaigns, alcohol prevention, and interventions on obesity and type II diabetes. The second speaker focused on the importance of bridging the gap between the "bench" and "bedside," or better translating clinical laboratory research to standard care practices. A common theme across the different presentations was that many used the clinical trial method on human patients to test theories in transplantation, cancer treatment, and fetal deformations in utero. In addition, presenters emphasized the need to continue recruiting people into clinical trials from within the NHS. The high number of clinical trials and cohort studies in the UK is directly related to the standardized National Health System. As Dr. Elizabeth explained to me later, it is very challenging to implement large, longitudinal cohort studies in the US medical health system because the system is not standardized.

One presenter discussed spinal deformities that can be detected earlier with the use of 3-D ultrasound images. He showed a series of images of a fetus at twenty weeks gestation and he carefully identifies which are "normal" or "abnormal" for spinal development. He emphasized that the technology of fetal MRI will compile all the pictures and decide whether they are normal or not. The machine can "learn" how to discern a normal picture from an abnormal picture. Therefore, as he stated "technology = easy you don't have to be an expert." This rhetoric of research and medical integration for the sake of patient care is characteristic of the 20$^{th}$ century

medicine. The focus on high-tech quality images of a fetus is also not novel mission. Rayna Rapp's work on amniocentesis and sonograms exposes how technologies are used to probe the intrauterine environment for information about the fetus (1997). The uses of 3-D ultrasounds images of the fetus along with technoscientific pathologies of the "normal" (Canguillem 1991) are both a part of the biomedicalization during pregnancy.

The KHP conference provides a snapshot of the elite and well-funded research projects that are taking place in the UK. Dr. Elizabeth is part of this elite scientific community and is highly regarded in many different scientific and academic circles. The conference also illustrates the economic and political climates that influence the production of scientific knowledge in the UK. Inspired by this snapshot, this chapter aims at illustrating the different scales of context that influence the production of the StandUp trial.

As I discussed in the Introduction, one of the logics of epigenetics that I examine is the concept of the environment. Epigenetic environments exist and interact at multiple scales. A scalar approach to epigenetics brings awareness to how epigenetic science also exists and is practiced within larger environmental scales. Using the image of the matryoshka doll, or a Russian nesting doll, the StandUp trial represents a scale of the environment, and it is influenced by the methods and expertise of the staff and principle investigator. The StandUp trial is also immersed in a larger teaching hospital and National Health System that is influenced by particular economic and political factors that shape research funding, ethical permission, and prenatal standard care. This chapter applies a scalar approach to examine how scientific methods and experiments are situated within larger contexts. In addition, I will show how these different contexts shape the prioritization of trial objectives and interventions.

The primary objective in this chapter is to examine how the scientific methods and design of the StandUp trial target only certain scales of the environment for intervention. In my research at the StandUp trial, I found that the main scales, for intervention are the molecular and uterine "environments." This narrow approach to epigenetic environments is directly related to the design and methods of the trial, as well as the expertise of the Principle Investigator. For instance, Dr. Elizabeth's training as a physiologist invariably influences how she plans and carries out the StandUp trial. Whereas, Dr. Sally, the principal investigator at the SmartStart trial in the US is a psychologist by training, which resulted in a completely different approach to epigenetics and prenatal interventions. Additionally, other significant scales of the environment marked by social and gendered experiences are obscured or exempted from consideration within design. To develop my argument, I focus on how the design and implementation of the StandUp trial, as well as the expert knowledge and background of the PI, influence what I argue are narrow approaches to epigenetic environments within trials purportedly expert at measuring epigenetic factors.

The secondary objective in this chapter is to elucidate some of the scientific and social contexts that influence the production of the StandUp trial. Both objectives require analytically zooming in and out across different scales. For instance, examining the methods, designs, and expertise that influence the StandUp trial will illuminate forces at individual and local scales; at the same time, situating the StandUp trial within economic and political environments will incorporate a broader scale. Near the end of the chapter I zoom out and explore the economic and political conditions of possibility that influence the StandUp trial. Overall, the emphasis of certain scales of the environment in scientific experimentation exposes a central tension related to examining epigenetics. Despite scientific agreement that there are multiple scales of

epigenetic environments that are interrelated, various scales of these environment are not equally prioritized in the everyday implementation of the clinical trials. The focus on certain scales of the environment begs questions, such as: Whose environment matters? Whose environment is important to protect?

**Methodological Scales: UK StandUp trial**

The context that shapes the StandUp trial includes many different factors. For instance, the StandUp trial uses the randomized clinical trial method to test an intervention of glycemic control on diverse obese pregnant women. Glycemic control aims at getting women to eat foods lower on the glycemic index. They are taught how to identify foods that are high on the glycemic index and then how to swap them out with foods that are lower on the glycemic index.[46] In theory, the randomized clinical trial method requires the control group to be exactly the same as the experimental group. The only difference with the groups is that the experimental group receives the intervention on glycemic control. In the context of nutritional interventions during pregnancy, maintaining pure control and experimental groups is hard to realize, if possible at all.

The intervention itself required constant communication and contact with the pregnant participants. The health trainer, or the interventionist on the StandUp trial, spent most of her time trying to scheduling meetings with each participant. The participants were required to do at least eight intervention sessions between twenty and twenty-eight weeks gestation.[47] At the intervention sessions the health trainer would review key lessons related to glycemic control and produce goals for making healthy swaps. The guiding hypothesis for the trial was that women in the intervention group would have lower rates of gestational diabetes, pregnancy complications,

[46] I explore glycemic index and glycemic control further in Chapter Four.
[47] Most women did not meet with the health trainer a total of eight times.

and cesarean sections. The longer-term hypothesis was aimed at following the progress of the children. The study will collect data from the children at six months and at three years of age to evaluate whether children born to women in the intervention group have any significant metabolic differences compared to the children born to women in the control group. In other words, tests on children will look for differences in neonatal fat and hormones associated with metabolic syndromes like cytokines.

Between 2011 and 2014 the StandUp trial recruited 1,558 women, half of whom received the intervention and half whom received standard care. During recruitment phase of the trial, the staff approached all women who had a Body Mass Index (BMI) of thirty or more at twelve weeks gestation. The trial also aimed at including ethnically diverse women including Southeast Asian, African, Afro-Caribbean, White British, and European. However, the trial had a very difficult time recruiting participants in general, and non-European women in particular, for different reasons. One main challenge was recruiting women from Muslim communities. The trial did not anticipate the cultural tensions related to implementing a nutritional intervention during the months of Ramadan. In addition, they did not customize the interventions for different ethnic foods, which may have impacted their ability to attract and retain women from immigrant and racially/ethnically diverse communities. Other challenges in recruitment emerged in the pilot study of the StandUp trial. In an unpublished report, they noted that seventy percent of women declined to participant in the trial because they did not want to be told how to eat or how to manage their weight. The social scientists in charge of analyzing the interviews with the women who decided not to participate in the trial concluded that obesity and fat stigma had a significant role in the women's decision to not participate.

There were five participating sites across the UK, however I focused the majority of my fieldwork at one main site called Rosalind, a teaching hospital in central England.[48] The Rosalind site had the highest recruitment numbers each month and it was also the main office for Dr. Elizabeth, the project coordinator, and key collaborators. The teaching hospital facilitated the centralization of all recruitment, data collection, and intervention delivery. In addition, the Rosalind hospital handled over 2000 births every year, so there was a large pool to recruit from. For these reasons, the Rosalind hospital was the ideal site to observe the structure of the clinical trial.

All activity pertaining to the clinical trial took place within the same building across three different floors. The staff had offices on the 10th floor within the women's health department. All bio-samples, including blood and urine, and analyses were stored and took place on the 10th floor as well. The fetal medicine unit, where recruitment took place, was located on the 8th floor. Data collection visits along with intervention delivery took place on the 4th floor in the clinical research facility (CRF) at Rosalind. The CRF was a large area in which many different research studies were taking place. The StandUp trial was just one of many ongoing randomized control trials at CRF. Distinctly, the StandUp trial was the only trial at the CRF that experimented with nutritional interventions during pregnancy.

The CRF was a clinical laboratory environment. It was hard to find and hard to navigate. The floor looked like a maze of grey rooms mixing wet labs with MRI, X-ray, and other technological equipment. The data collection visits with the research midwives took place in exam rooms one or two, which included various medical props like sterilizing equipment, needles, blood pressure cuff, a bed, a scale, and a stadiometer to measure height. The StandUp trial personnel had to label and bring their own scales to each room because the rooms did not

[48] This is a pseudonym

have any scales in them. There was also a computer for the midwives and participants to enter data. The intervention sessions also took place in similar exam rooms.

The four other participating sites around the UK did not have the same resources as Rosalind. Midwife researchers at smaller sites had to write everything by hand and then input it into an available computer after their meetings with the participants. During my conversations with other research midwives, there was a sentiment that the Rosalind staff had it "easier" or was spoiled because of the resources at the teaching hospital. In addition, the Rosalind site had a lab technician in charge of processing all the bio-samples, something that did not exist at other sites. At other sites the midwife researchers would have to process bio-samples themselves, and at two sites, the midwives would travel to local community centers to meet women far from urban areas.

The diverse experiences of data collection and intervention delivery at each location index the different kinds of structures, contexts, and resources that shaped the individual sites in the consortium. The distinct resources and distribution of labor at other sites complicated the ability to standardize all data collection. However, the StandUp trial consortium needed five participating sites in order to reach a recruitment population goal of over a thousand women. Therefore, although more sites make it difficult to standardize data collection and resources, each site will contribute to the larger recruitment goal.[49] The key to a "strong" RCT is a large population. Statistically, it is assumed that if the trial has a large population size then it can have

[49] One example of the challenges to standardizing across different sites had to do with the data collection. For instance, since some of the sites did not have a lab technician to process bloods, there were always problems with identifying and organizing the blood samples. With such a large sample size there are hundreds of thousands of samples to organize, classify, and store. In addition, some sites did not collection fasting blood glucose for their research bloods at the twenty-four week visit. Another instance occurred when Lucasade, the soda that they used for the glucose tolerance test, changed the amount of sugar they put in the soda, this effected the amount of sugar the women needed to drink for the test.

more "power" to control for random effects that may slant the data as well as to better elicit evidence of causality.

Not only were the individual sites different in terms of data collection and resources, but there was also a hierarchical division of labor within each site. The division of labor at the StandUp trial was organized by the National Health System (NHS) through levels or "bands." The bands refer to the amount of skill and pay required for each job within the NHS.[50] For instance, the Principle Investigator was band or rank 9, which includes all senior professionals with a doctoral degree and advanced research experience. The project coordinator, who was also a midwife, was band 7 and the research midwives were band 6 or 5 depending on experience. The research midwives handled the bio-sample and survey data collection visits with the pregnant women. In general, research assistants are usually band 4 and have the potential of getting promoted, depending on work experience and earned degrees or certificates. In the StandUp trial the research assistants recruited the pregnant women. The health trainers were strictly titled as band 3 and had no option to move up in band level or pay. Band 3 did not include any midwives and they were paid the least amount compared to the other staff members on the trial. The labor structure was similar across other trials housed in the women's health department. Describing the division of labor is important for understanding the context and economic incentives regarding the trial implementation. The health trainers often felt unappreciated and wanted more guidance from superior staff. The hierarchical labor structure of the trial is directly related to the NHS. The standardized health care system in the UK provided the structure and resources for the StandUp trial.

[50] In the U.S., the SmartStart trial took place mostly in public clinics and some private clinics. However, both trials were funded by government health research funds.

The StandUp trial is one of many emerging studies that are drawing from epigenetics to prevent future epidemics. Zooming out helps situate the StandUp trial in reference to other similar trials. For instance, in 2012, a study entitled "LIMIT: Limiting weight gain in overweight and obese women during pregnancy to improve health outcomes," lead by Jodie Dodd, concluded one of the largest RCTs testing behavioral interventions on pregnant women. The LIMIT study recruited 2,212 women with a body mass index over twenty-five. Half of the women received a behavioral intervention of diet and exercise and half received standard prenatal care. The study found no clinically significant difference between the control and intervention group (Dodd et al., 2014). The LIMIT study is currently following up with the participating mothers and the three year old children. They are collecting anthropometric and genetic data from the mothers and children. The follow up study aims to contribute to the epigenetic literature on non-Mendelian inheritance. The StandUp trial staff and PI worked closely with the LIMIT study. In 2013, the project coordinator and Dr. Elizabeth of the StandUp trial traveled to Australia to observe the execution of the LIMIT follow up study with the mothers and children. The StandUp trial will do a similar follow up on their respective 1,558 participants in the UK.

The StandUp trial is one of many trials testing nutritional interventions on pregnant women. For instance, the low glycemic index diet in pregnancy to prevent macrosomia (ROLO) study, in Dublin, Ireland, is another RCT that tested dietary interventions on pregnant women. The ROLO study recruited 800 women and randomized them into a control group and experimental group that received a dietary intervention. The aim of the study was to find a link between women in the intervention group and decreased rates of macrosomia. Macrosomia, or infants that are large for their gestational age, is associated with metabolic syndromes like

obesity and diabetes later in life. Therefore, macrosomia is a target outcome that indexes future risk of chronic disease in early development. The ROLO study also justified their intervention using epigenetic theories related to increased risk of obesity among children born to obese women. The trial did not find any significant difference between the intervention and control groups (Walsh et al., 2012).

The ROLO, LIMIT, and StandUp trials are similar in that they all use evidence-based medicine to test epigenetics in and through prenatal interventions. The outcomes are focused on rates of improved pregnancy outcomes and fetal outcomes. The nutritional and behavioral interventions tested in the LIMIT and ROLO did not reflect any significant effect in the pregnancy outcomes. In addition, a recent publication on the results of the StandUp trial concluded that consistent with other nutritional interventions during pregnancy, they did not find a significant difference between the control and experimental groups. The findings at the StandUp trial, ROLO, and LIMIT, reflect the limits of testing nutritional interventions during pregnancy. Clinical trials in the US have also found similar inconclusive results.[51] However, both the LIMIT and StandUp trials are still ongoing and collecting data from the children of the pregnant participants. By following the development of the children over time it is possible that they find some significant difference between the children of the women in the intervention group compared to control group. Another common trend across all the different large-scale trials testing nutritional interventions on pregnant women is that none of the trials measure or account for the cultural, historical, and gendered experiences in the women's environment. The study designs, data collection, and target outcomes focused on particular scales of the environment that excluded many other scales. Next I explore the disciplinary background and expertise of Dr. Elizabeth, which centers around her work on animal models.

[51] see the Diabetes longitudinal clinical trial in the US, collaborator named Rena Wing.

**Disciplinary Scales: Animal models**

One aspect about the StandUp trial that sets it apart from other trials is the fact that it based its design on animal models. Dr. Elizabeth's background in physiology and expertise in animal models provided the lens and resources to inform the StandUp trial. Dr. Elizabeth is a director of a lab that specializes in animal models. She also supervises many different doctoral and postdoctoral projects on animal models that test different factors related to obesity and diabetes. Dr. Elizabeth used her expertise in animal models to support the epigenetic theory that the uterine environment can, as she explained, "insidiously" affect fetal development. However, animal models or obese, pregnant mice are only one environmental context that reflects epigenetic modification. In her speech from the KHP conference, Dr. Elizabeth explains her work on animal models.

> We do a lot of our work in animal models. We go backward and forwards between animal models and the clinic. And these models are incredibly important to us. […] Since 1998, I've been working on [the Barker] hypothesis, in relation to obesity in pregnancy. […] For our animal models we give rats and mice absolutely delicious things to eat, and then they get fat and then we make them pregnant, and this is actually a quite good model of obstetric obesity. And we've been looking at the children when they grow up, or the offspring of these rats and mice, and it's extraordinary they have very high blood pressure, they become fatter, they have abnormal glucose control […].[W]e believe that the fetal [development] is very susceptible to the maternal environment and that it predisposes children to disease as they are exposed to these metabolic [conditions] in utero.

This excerpt of Dr. Elizabeth's speech reflects how her research is guided by animal models. In various conversations I had with Dr. Elizabeth she mentioned how important it is to have scientifically valid animal models before designing trials on women. It is also from mice models that scientists learn which biomarkers to examine in trials on human pregnant women.

Intervening and experimenting on mice or rats provides the groundwork for understanding epigenetic mechanisms.[52] In a 2011 article Dr. Elizabeth cites her own research on animal models to argue that animal models provide evidence for the prolonged and adverse effects of maternal obesity on the offspring (Dr. Elizabeth et al. 2011).

The slippage between calling the offspring of the mice "children" reflects the ways in which Dr. Elizabeth states that she "goes back and forth" between animal models and humans. This conceptual movement draws from her disciplinary expertise and has implications for the design of the intervention on human pregnant bodies. Blurring the boundaries between mice models and human models maintains implications for understanding epigenetic environments. For instance, the differences between mice that are overfed and impregnated in a laboratory and human pregnant bodies that are not force fed or impregnated against their will might seem obvious to the lay population. However the differences in the scientific discourse between human pregnant bodies and pregnant mice are often minimized to one aspect– the placenta.[53] Mice do not have the same kinds of placentas as humans and this challenges scientific understanding of how the uterine environment and food transfer effects fetal development. Some argue that the difference between mice and human placentas is significant while others argue that it is not. For instance, when I asked Dr. Elizabeth about this, she explained that the uterine environment does effect human? fetal development, knowing the biochemical processes of nutritional transfer across the placenta is not necessary for understanding epigenetic mechanisms.

[52] Landecker and Panofsky write, "much of the supporting evidence in [epigenetics of early -life exposure] comes from experimental rodents" (2013:341). These models are often the precursors of randomized clinical trial design. See also Haraway's work on OncoMouse

[53] At the obesity conference in Atlanta, Georgia, I saw many panels on animal models linking obesity during pregnancy to future risk of obesity in children. The conference had over 9,000 experts, physicians, scientists, surgeons, and academics who all worked on obesity. There was also an entire expo dedicated to a variety of weight loss strategies, including surgery, liquid diet plants, and exercise equipment.

However, the significant difference between mice and moms is not just their placentas. For instance, mice are not subject to social environs in the same way as humans. The laboratory environments of mice have completely different historical, political, and cultural factors that influence their behavior. Human pregnant bodies are also socialized differently primarily in reference to gender and race. Human pregnant bodies live in social environments that expose them to a variety of local biologies (Lock 1993), which influence their behavior and bodies.[54] As Niewohner explains in his work on the "molecularisation of biography and milieu," epigenetics are trying to link gene expression to local cultural practices (2011). For instance, pregnant bodies are often correlated with heteronormative relationships and sexual orientations. In addition, pregnant bodies are associated with gendered notions of mothering and motherhood. However, as queer scholars have argued, having a body that biologically reproduces does not map onto different kinds of kinship and families. Pregnant bodies are gendered, and therefore, pregnant bodies are gendered environments.

Scientists have already fallen into the gap of assuming that mice are just like humans. Before the mass-production and distribution of the thalidomide drug, it had been tested on mice (Wolf 2010). However, it turns out mice are not harmed by thalidomide the way that pregnant humans are. The mass consumption of thalidomide to prevent nausea during pregnancy resulted in thousands of children born with small or non-formed limbs (Timmermans and Berg 2003). The thalidomide trials were not the first or last assumption that contaminated scientific trial designs. In another example, scientists using mice models found spurious associations in their cancer research because they assumed that the plastic boxes that mice live in are not a significant environmental factor (Haraway 2008). In cancer research the assumption of a "controlled"

[54] See Lock, Margaret. 1993. Cultivating the body: anthropology and epistemologies of bodily practice and knowledge. Annual Review Anthropology.

environment confused the rates of cancer among the control and experimental mice groups. As it turns out, certain plastic boxes leak cancerous chemicals over time.

Therefore, it is not just animal models that are seamlessly translated into human trials, but the same taken for granted assumptions about environments are also translated. Plastic boxes are social and material forms of an environmental scale. Like a matryoshka doll, or a Russian nesting doll, a plastic box is an environment within a laboratory environment within a private or publicly funded building in an urban or rural environment, etc. Similarly, uterine or fetal environments are contained within the bodies of women, who too are exposed to various other environmental factors, like air quality, physical stressors from waged labor, or issues of legalization and migration across national borders.

The collapsing of the scales of the environment or the narrow focus on one scale of the environment misses the large network of relations that influence significant differences in mice models and human models.[55] Focusing on the placental difference between humans and mice reflects implicit assumptions that are not accounted for in the translation between human and animal models. Ignoring other differences between humans and mice effaces the cultural and gendered experiences of pregnant participants, but it also ignores the importance of environmental scale. Put another way, trials on humans that are translated from animal models are using theoretical frameworks that narrowly conceive the epigenetic environment and apply the same narrow conceptions of the environment in human models. In this practice of epigenetics the scales of the environment are not just hierarchical (Van Speybroeck 2000), but rather non-existent. By applying narrow conceptions of the environment, scientists are missing significant scales of the environment that are important to understanding epigenetics.

[55] See also, Carol Adams on the Cultural Politics of Meat.

**Molecular Scales: Data Collection and Analysis**

Although the short term goal of trial was to test the effectiveness of nutritional interventions on obese pregnant women, the longer-term goal aimed at examining whether nutritional changes during pregnancy would affect fetal development and development in early childhood. The longer-term goal focused on tracing biomarkers from the mother to the child, which could potentially expose epigenetic mechanisms. The systematic collection of bio-samples and prioritization of biomarker analysis reflects the trial's focus on tracing the molecular scale.

To understand the mechanistic aspects of epigenetics, the trial is collecting large amounts of bio-samples from the pregnant participants and then their children after delivery. As I mentioned above the women have to provide six samples of blood, which includes five tubes each visit, and at delivery, the women have the option to give a sample of their placenta and cord blood. Within twenty-four hours after birth, the staff will schedule a time to measure the infant's height, weight, and skin fat. At six months, the women return to do another data collection visit with their babies. Then, at three years, both the women and their babies get measured and provide DNA samples. The focus on bio-samples is structurally incorporated into the design and funding of the trial. Unlike the StandUp trial, the SmartStart trial did not maintain such an emphasis on collection bio-samples. The SmartStart only collected blood three times during pregnancy and did not have resources or infrastructure in place to collect mass amounts of placenta samples. In contrast, the UK the research midwives and research assistants were on call each day checking the online hospital records to see if participants were admitted and whether they had delivered. The focus on collecting certain kinds of data are reflected in the trial design and also reflect the various environments that clinical trial research is executed within.

Despite the recent results that found no significant difference in the health outcomes between the control and experimental groups in the StandUp trial, the data collected during pregnancy is valuable for exploring epigenetic mechanisms using biomarkers. As some of the collaborators of the StandUp trial told me, scientists really do not know that much about pregnant bodies and nutritional transfer across the placenta. The opportunity to collect blood, urine, cord blood, and placenta samples is a scientific gold mine for any scientists interested in the organ transplantation, immunology, regenerative medicine, and stem cell research.

In the second half of the twentieth century the systematic collection and examination of bio-samples during pregnancy were not easily accessible at such a large scale. The effects on the children from the Thalidomide trials in the 60s and 70s lead to the increased protection and ethical regulation of including pregnant women into trials. These regulations around participation were put in place to protect the developing fetus, not necessarily the women. However, with the emergence of epigenetics, the importance of maternal nutrition, and the growth of ethical regulations around randomized clinical trials, scientists can now receive funding and permission to collect blood, urine, cord blood, and placenta samples from pregnant women. Moreover, the collection of pregnant bio-samples has expanded exponentially as a result of the growing number of nutritional interventions during pregnancy. For instance, the principle investigators at both trials told me how different scientists would approach them to use some of the samples for separate analyses, other samples included feces and breast milk. At the SmartStart trial, Dr. Sally decided to collect feces samples from the pregnant women to collaborate with another scientists on a project related to the microbiome.

All of the biomaterials collected from pregnant bodies can be used to trace biomarkers. Biomarkers can illustrate a biological process that is are said to be objective and measurable

(The Biomarkers Definition Working Group in Rose and Singh 2009). Epigenetics draws a direct relationship between molecular changes to DNA sequence and environmental stimuli, which can occur at different scales of the environment. The prioritization of biomarkers relates to the ways in which they can trace biological processes across time, space, and bodies. The premise underlying the collection of biomarkers is that they can provide a way "to study the outside and the inside of the body together, along chains of causation that contain both social and biological elements" (Landecker and Panofsky, 2013:243).

To study the biomarkers and DNA of the pregnant participants and children, the StandUp trial has a small team that is dedicated to bio-sample data analysis.[56] One of the key people on this team is Connie. Connie, a four year veteran on Dr. Elizabeth's research team, is in charge of monitoring and organizing all the bio-sample data. She is one of the lead lab technicians and completed her doctorate in proteomics—a specialized field within genetics--a few years before becoming a staff member on the StandUp trial. She is an expert in managing labs, data, and bio-sample.[57] Connie was present at the first visit I made to the trial in 2012 and she was still working on the trial during my longer data fieldwork phase in 2014. On one of the days I shadowed Connie, I asked her about the kinds of analyses the trial can do with the bio-specimen data. Connie responds, "they could do infinite analyses on these samples." By "infinite" Connie

[56] The trial found it difficult to recruit men into their study for DNA collection.

[57] The protocol of the StandUp trial requires the participants to provide blood and urine samples at their baseline/randomization visit, 28 week visit, which is when they have to do a glucose tolerance test, 36 week visit, and 6 month follow-up. In addition at around 28 weeks the participants are given another consent form for the extraction of placenta samples and cord blood, which all needs to be collected on just after the women have delivered. During the four visits outside the delivery room, 5 tubes of blood are collected. Except for the glucose tolerance visit. At that 28 week visit a few more tubes of blood are collected for the GTT. At the 28 week visit the "research bloods" should be collected at fasting (but different participating sites do it at different times) the are then labeled, recorded, and prepped for different kinds of analysis. During the four visits outside the delivery room, five tubes of blood are collected. Except for the glucose tolerance visit. At that 28 week visit a few more tubes of blood are collected for the GTT. At the 28 week visit the "research bloods" should be collected at fasting (but different participating sites do it at different times) the are then labeled, recorded, and prepped for different kinds of analysis.

implied that if money was not an obstacle, Dr. Elizabeth and her colleagues could do an infinite amount of analyses.[58]

At the KHP conference discussed at the beginning of the chapter, Dr. Elizabeth explained why examining biomarkers is important:

> In July we will start the first studies in the first children [from the StandUp trial] and we will be looking at them as they grow up.[59] […]. We have been funding very well by MRC and European Union consortium […]. And actually I'm going back to my love of biomarkers now and we have a lot of money to look at the biomarkers in the mother which might be influencing the developing fetus and therefore we'll get a handle on those associations, and we are using lots of fancy techniques [and] technology to look at that [...]

As she suggests, the scientific practice of collecting and analyzing biomarkers is vital for understanding the molecular modifications in fetal development. Bio-sample collection and funding towards biomarker analyses reflects a narrow approach to the environment. Biomarkers are used to trace an exposure in the uterine environment to an effect or genetic expression in the offspring. As Dr. Elizabeth states, she is very interested in following up the children of the pregnant participants. Through epigenetic logics the children could potentially inherit the biochemical changes that may occur as a result of being exposed to a nutritional intervention in utero.

Biomarkers represent the belief in prediction and prevention by targeting the individual and focusing on the molecular scale to trace mechanisms (Abu El Haj 2012). However, they do

[58] For every set of research bloods there are different ways to prep them and store them for analysis. Each participant has four sets (four data collection visits) of research bloods and each set requires four different kinds of preparations with different serums and DNA extractions. The blood processing results in 50 vials that need to be frozen and stored until they are sent to another site for analysis. A key problem for Connie is making sure that the 5 different participating sites are all collecting, prepping, recording, and freezing the DNA samples in the same way. Since different sites were doing it differently, Dr. Elizabeth decided to have all the sites send their bio-specimen/ DNA and urine samples to Connie so that she could organize them and store them. With 1500 participants and at 50 vials each for 4 sets of research bloods there are over 300,000 DNA and urine vials that Connie needs to record and properly store, on top of the daily bloods that come in for processing.

[59] In a previous interview I had with Dr. Elizabeth she told me that only 30 percent of women with a BMI over 30 have pregnancy complications, and that 70% have no complications at all.

not necessarily illustrate or capture an individual's, or in this case, a pregnant person's cultural or personal experiences (Strimbu and Tavel 2010).[60] Moreover, biomarkers do not reveal experiences of gender or social environments, which also influence individual behavior. Scholars caution against the belief that biomarkers will be the solution to predicting or diagnosing disease because there is still a degree of uncertainty in tracing gene expression across the life span (Rose and Singh, 2009). For instance, Lock uses the concept of somatic reductionism to argue that despite the known limits of molecular and physiological determinism of disease outcomes, current research on Alzheimer's proposes to focus solely on identifying molecular biomarkers in individuals (2013)[61]. The focus on biomarkers is based on the idea that Alzheimer's can be detected, at the molecular level, in individuals twenty years prior to experiencing any symptoms. Lock argues that the prioritization of biomarkers reflects a reductive approach in epigenetic science. I further argue that it is not only a reductive approach, but specifically a narrow framing of epigenetic environments.

In early May 2014, I attended a "data conference call" with Dr. Elizabeth, Connie, and a few other collaborators to discuss plans for the data analysis. The trial was awarded around one million pounds for analyzing the bio-sample data, which does not include the behavioral data from the intervention delivery. At the beginning of the meeting Dr. Elizabeth requested to have all base-line DNA and urine analysis by June 2014, since they finally reached their recruitment

[60] Strimbu, Kyle and Jorge Tavel. 2010. What are Biomarkers? Current Opinion HIV AIDS. 5(6): 463-466.

[61] Although Lock acknowledges that research on epigenetics (or the epigenome, which includes the chemical changes at the molecular level that produce modifications to genetic sequence), is influencing the ways in which we approach disease, health, and illness throughout the life cycle, she warns that the age of epigenetics can also produce new kinds of reductionism. In her article titled "The Epigenome and Nature/Nurture reunification: A Challenge for Anthropology", Lock writes that the "[epigenome] has the potential to incite new forms of reductionism that may well result in inappropriate moral attributions, stigmatization, and discrimination, largely because social and economic variables external to the body are, from the outset, set to one side" (2011: 296). Lock brings attention to the fact that although epigenetics offers a way to understand how social and historical inequalities can leave material traces on the body, the research does not necessarily focus on the political, structural, and historical systems that perpetuate inequality.
See also: Lock, M. 2013. The Alzheimer Conundrum:entanglements of dementia and aging. Princeton University Press. Princeton, New Jersey.

goal of 1500 just that week. After the group discussed some of the key logistical challenges to meeting this goal (e.g., freezer space and staff to process the bio-samples), they began making a list of key biomarkers that they wanted to examine.[62]

At the meeting, Dr. Elizabeth's briefly described a list of biochemical substances to examine in the pregnant participants' blood and urine samples: feraline, vitamin D, and cytokines. Feraline is linked to liver disease in non-pregnant adults but nothing is known about the effects of feraline in pregnant adults. The group decides to examine feraline in the pregnant women's DNA because of its publication value, or impact on the scientific community in addressing pregnant adults. Vitamin D is linked to deficiencies in obese population. However, it is cut because it is not related to the trial's main hypothesis. Cytokines are linked to neo-natal adiposity, or how much a fat an infant is born with, and is approved because of its potential to indicate fetal programming during gestation.[63]

The purpose of testing the blood for cytokines is that if they are present in the pregnant body or uterine environment they can influence fetal development in the uterus, resulting in an infant with high fat deposits, or adiposity, which is linked to obesity in adulthood. Cytokines are associated with high processed foods and foods high in sugar, salt and saturated fats. In this way, biochemical materials are associated with food and nutrients, which is associated with the metabolism or ways of processing these foods. Obesity and diabetes are often regarded as metabolic syndromes, because they deal with how foods are processed in the body and the

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[62] Most midwives do not have experiences processing bloods and as Dr. Elizabeth says they are not qualified to do this kind of technical labor. Therefore, she wants Connie and one other graduate student to handle the processing and organizing (an issue that Connie had alluded to before). Connie assures Dr. Elizabeth that she will aim to have all samples organized within the next month, a task that she later told me seems impossible but she is willing to work overtime without any extra pay to get it done.

[63] In the same meeting, Dr. Elizabeth states, "[we will publish a] paper on metabolics of hyperglycemia in pregnancy. It will be ahead of the game, addressing the issue of the chicken or egg. Is insulin resistance a predictor for diabetes?"—this is the key point she is interested . See also, 9/11/15 version of chapter, which has the paragraph on how Dr. E claims she is not interested in the behavior stuff.

effects they may have on blood pressure, cholesterol, and hypertension. Research shows that nutrition can affect cell metabolism, which then influences the kinds of epigenetic changes that occur. In this way epigenetics and metabolism are entangled and this entanglement is projected onto the diets of pregnant women.

Dr. Elizabeth comments that she is interested in examining cytokines in the blood of the pregnant women because the animal models suggest that cytokines may cause extra fat deposits in the new infants. Since the ROLO trial is also testing for cytokines, she decides to include it but specifies that the test should be done on the bloods collected at twenty-eight weeks gestation to see the influence on fetal development.

Once the group finalized the list of biomarkers they are interested in examining across the pregnant women and their children, Dr. Elizabeth exclaimed, "This is such a fascinating time to be doing this. I am so excited!" She goes on to say that the more tests they do, the more reliable their results will be. At the end of the meeting I spoke with Dr. Elizabeth, who stated: "I am more interested in the [DNA samples]. That is why I did this. The behavior part is not my thing, the science mechanistic is vital to clinical outcome related to metabolics, diabetes, and care." Dr. Elizabeth's focus on biomarkers emphasizes the molecular environment in an attempt to map out causal mechanisms of epigenetic modification. Although all scientists are trained and have particular passions for particular kinds of scientific knowledge production, epigenetics as a concept and object of science knowledge production requires an explicit interest in the relationship between and among multiple coexisting scales of environments.

While focusing entirely on biomarkers, the role of multiple scales of environments fades away into the background. Epigenetics tells us that there are multiple scales of the environment and they are all interrelated. However, the various scales of the environment are not equally

prioritized in the everyday implementation of the clinical trials. The intense focus on collecting samples from obese pregnant women during gestation, at delivery, 6 months after, and their children highlights a systematic definition of which environmental scales should be targeted for intervention and how environments can be quantified. The process of collecting, prepping, and organizing biomarkers is an instantiating practice of belief around what environments mean and which scales matter. The next section explores the how the trial discursively frames the uterine environment or maternal metabolic states as the key target for intervention.

**Uterine Scales: Targeting the Pregnant Body**

In the presentation Dr. Elizabeth gave at the KHP conference, she outlined her research and approach to epigenetics in the context of obesity during pregnancy. Similar to the other presentations at the conference, Dr. Elizabeth aligned herself with the goals of bridging "gaps" in the community and "translational" medicine or using findings from the laboratory to improve patient care. As a physiologist by training, she is also greatly invested in advancing our understanding of epigenetic mechanisms to predict disease before it manifests. In her own words she explained:

> So [I'd like to talk to you] about the area of research that I am most involved in, which is a hypothesis called the developmental origins theory, it's been around for 20 years now. And it suggests that the maternal metabolic state plays a very important role in the future risk of disease in the developing child. So if the mother is diabetic for example or if a mother has or is fairly obese then the suggestion is that the fetus is insidiously affected by the metabolic state of the mother in the way that that child is at risk for disease in their future lives. 20 years ago, David Barker who developed the theory, nobody really believed him and least of all me actually, that's when I started working on whether it's true, now I am completely convinced. The suggestion, which is not totally unusual, is that the intrauterine environment has a prolonged affect on the health of the child. *So what we are thinking is,*

> *importantly, that pregnancy is the window into the health of our next generation* (emphasis added).

This excerpt is reflective of the ways in which the epigenetic paradigm is guiding scientific research. Although Dr. Elizabeth does not use the term epigenetics in her speech, she indexes the logics of epigenetics in different ways. She refers to the fetal origins hypothesis, David Barker, and the development origins of disease, which is formally called the developmental origins of health and disease (DOHaD). Terms like "maternal metabolic state" and "intrauterine environment" are also connected to notions of DOHaD, which claims that exposures in the intrauterine environment can potentially (and unpredictably) cause latent effects for the development fetus, child, and adult.

In Dr. Elizabeth's own words the developmental origins theory "suggests that the maternal metabolic state plays a very important role in the future risk of disease in the developing child." The "maternal metabolic" state refers to, for instance, glucose metabolism, excess fat, cholesterol levels, and blood pressure, all of which are related to diabetes and obesity. The term maternal metabolic state does not aim to illustrate the figure of a pregnant body, instead it foregrounds the metabolic metabolism, a process, or a state of being. Although attaching the word "maternal" to "metabolic" implies something else. The root word of maternal, is associated with "mother," but it is merely a gendered placeholder for a metabolic state of being that can "insidiously affect" fetal development.

At the end of Dr. Elizabeth's speech at the KHP conference, she used the phrase "intrauterine environment" instead of the "maternal metabolic state." By doing so, she drew a direct connection between the maternal metabolic state and the intrauterine environment to imply a relationship between the pregnant body, uterus, fetus, and child. By focusing on the intrauterine

environment, the future risk, and "insidious affects" of the maternal metabolic state Dr. Elizabeth diverged from the traditional representations of gender in science. Are intrauterine environments women? Are metabolic states gendered? Are environments also gendered? Gender does not seem to be a category of its own but also one that cannot escape from different scales of the environment. The intersection of gender, race, and sexuality are all aspects of environments that are not accounted for in the trials. I emphasize gender because pregnancy and motherhood is a period of time in which gender is naturalized to women's behaviors and lifestyles.

There are different ways of referring to the same space-time of gestation, such as EG and EG (Rapp 1987, Rapp and Ginsburg 1994, Martin 1987). Dr. Elizabeth uses the uterine environment and maternal metabolic state, which draw directly from epigenetics, and others use different terms. In their book *Mismatched: Why Our World No Longer Fits Our Bodies,* Peter Gluckman and Mark Hanson claim that a mismatch occurs between the uterine environment and the environment that surrounds the developing offspring (2006). The environment outside the uterine environment is referred to as the "modern world," and this world is not conducive to the anticipated fetal environment in pregnancy (2006: 253). Gluckman and Hanson argue that the cause for increased rates of obesity and diabetes is related to the "mismatch" between the fetal environment and the environment in which a child develops. For instance, if a fetus in utero is nutritionally deprived or stressed then the "adaptive response" is to prepare for a nutritionally poor environment outside of the uterus. The mismatch occurs when the offspring ends up developing in a very nutrient rich environment.

"Nutrient rich" refers to the amount of food not the quality, access, or affordability of food. The emergence of the term "food desert" is symptomatic of the structural problems related to equal access to fresh and affordable food, an issue that has plagued American cities for

decades (Font 2012)**.** Gluckman and Hanson are bringing attention to the issue of distribution and access in the "modern world" that effect epigenetic modifications during pregnancy and throughout adulthood.

In the co-authored book called *Mismatched: Why Our World No Longer Fits Our Bodies,* Peter Gluckman and Mark Hanson claim that a mismatch occurs between the uterine environment and the environment that surrounds the developing offspring (2006). The environment outside the uterine environment is referred to as the "modern world", and this world is not conducive to the anticipated fetal environment in pregnancy (2006). Gluckman and Hanson argue that the cause for increased rates of obesity and diabetes is related to the "mismatch" between the fetal environment and the environment in which a child develops. For instance, if a fetus is nutritionally deprived or stressed then the "adaptive response" is to prepare for a nutritionally poor environment outside of the uterus. The mismatch occurs when the offspring ends up developing in a very nutrient rich environment (2006).Nutrient rich refers to the amount of food not the quality, access, and affordability of food. The emergence of the term "food desert" is symptomatic of the structural problems related to equal access to fresh and affordable food, an issue that has plagued American cities for decades**.**

Gluckman and Hanson also discuss "the environment of the fetus [which] is created by its mother" (Gluckman et al., 2005: 530). This description of the environment breaks down the fetal environment to include the individual pregnant body, which is automatically assumed to be the "mother." In a social context where queer families and assisted reproductive technologies are expanding (Mamo 2007), older Platonic notions of biological reproduction still pervade contemporary scientific discourse. However, it also implies that uterine environment is the fetus's environment in the possessive form. The possessive form prioritizes the fetal environment

over the pregnant body. In another diagram produced by Gluckman and Hanson they use the term "Fetal Matrix", which Richardson critically examines as a representation that foregrounds the fetus and places the pregnant or maternal body as an "obligatory point of passage" (Richardson 2015: 223). The current maternal representations in epigenetic theories like Gluckman and Hanson's, illustrate narrow conceptions of the environment. Both the fetal or uterine environments are referring to the same space-time of gestation, but the terms carry different meanings. Further the focus on the fetus draws from social and political framings that undergird contemporary debates around fetal/maternal rights. Likewise, the term maternal metabolic state, backgrounds the fetus, and implies a state of being rather than a discrete space like the uterus or pregnant body.

Feminist scholars highlight how scientific descriptions of bodies are not created in a vacuum but reflect social and cultural meanings (Haraway 1991, Martin 2001, Jordanova 1986). The language used to describe the critical periods or environments that are vulnerable to epigenetic changes emphasize particular gendered environmental scales of context. As Haraway claims, bodies are not born but made (1991). In the same vein, I would argue that scales and hierarchies of environments do not exist naturally, but are understood and conceptualized in particular socio-biological ways. For example, in the epigenetic paradigm, the increased dependence of framing women's bodies as uterine environments foregrounds a space-time that elides other environments from the scope of consideration. Pregnant bodies are also environments, which are enveloped in and nested within multiple scales of the environment.

Emphasizing uterine or fetal environments over cultural, historical, and gendered environments limits the possibility of exploring multiple interrelated environments.[64]

Moreover, targeting uterine environments or women's behaviors are still emphasizing particular scales of the environments at particular times. Targeting pregnant bodies for intervention assumes that pregnant bodies are mothers, and that mothers are individually responsible for the health of the nation. The essentialization of biological reproduction limits the scope and potential of epigenetics as a framework and object of analysis. Epigenetic science aims to explain how all these scales are related to each other. However, in the implementation of the clinical trials I examined, the target scale is the individual, ethnically diverse, pregnant and gendered body.

**Economic and Political Scales: Conditions of possibility**

In an anticipatory regime, epigenetics links women's eating behaviors during pregnancy with the risk of (re)producing obesity and diabetes in future generations. As Dr. Elizabeth stated, "pregnancy is the window into the health of our next generation." In this way, epigenetics as a new paradigm could further justify the control of women's bodies and behaviors across the lifespan. Alternatively, epigenetics may also offer an avenue for shifting the focus on individual responsibility, to environmental, structural, historical and political inequalities. To analyze the epigenetic environments using a scalar approach I will outline a few different environmental scales and factors that influence the production of epigenetic knowledge. By doing so, I aim to highlight the nested scales of the environment that surround the lab or clinical trial, or the spaces

[64] Landecker and Panofsky write about how social factors like socioeconomic status and poverty are included in the macro-notions of the environment (2013). For social scientists and scientists alike epigenetics offers a way to trace the inequalities of social and historical conditions to biochemical processes that can cause disease.

that apply epigenetics. I will broadly examine the scientific, political, and economic factors that influence the StandUp trial.

One dimension focuses on the economic justifications that are central to healthcare. Another similar aspect is the speculative framing of cost benefit analysis that characterizes the justification for funding preventative interventions. Both factors are part of neoliberal approaches in techno-scientific regimes. The following example reflects the economic and speculative approach to prenatal interventions. Illustrating this context highlights the milieu that epigenetics is emergent in. For instance, Nadia Abu El Haj notes that neoliberal epistemologies are persistent in the postgenomic era. El Haj argues that epistemologies focused on risk, self-care, individual responsibility, and speculation are all characteristic of neoliberalism (2007). Further she explains that neoliberal approaches promote the "reentrenchment of the welfare state, the deregulation of industry, and […] the privatization of biological research" (El Haj 2007:290). El Haj emphasizes that postgenomic science operates within a "neoliberal field" (200:291). Similarly, I argue that it is important to recognize the economic and political environment that epigenetics operates within. Distinctly, I argue that if we take epigenetics seriously, the social, economic, and political environments that influence the production of epigenetics and postgenomic science must be acknowledged as vital environmental factors. If we do not critically examine the neoliberal environment that epigenetics operates within, we will continue to produce epigenetic knowledge that reinforces neoliberal epistemologies of risk and individual responsibility.

To justify nutritional interventions during pregnancy, Dr. Elizabeth describes the future risk, and more importantly the future cost related to obesity during pregnancy (2011). Dr. Elizabeth and her colleagues write that obese pregnant women have higher rates of pregnancy complications and that pregnancy complications are expensive and contribute to higher

healthcare costs. Since the National Health System budget has a growing financial deficit, the authors suggest that interventions during pregnancy can save money. In addition, as a result of epigenetic inheritance interventions during pregnancy, may also have long-term impacts on the children. Therefore, the prevailing scientific suspicion is that controlling pre-pregnancy weight and preventing obesity among women of reproductive ages may also reduce the risk of obesity in future generations. Further, interventions during pregnancy could reduce the healthcare costs from pregnancy complications and future healthcare costs in the next generation.

Claiming that particular research interventions may save money in the future is characteristic of the current healthcare environment in the US and UK The National Health System in the UK experienced major restructuring in 2010 and the effects of that restructuring are currently manifesting across hospitals and healthcare centers in the U.K (Ham and Murray 2015). In the current economic climate healthcare costs are still increasing and research that aims at reducing cost is deemed extremely valuable. Saving money in the future through disease prediction, speculation, and anticipation, characterizes the economic and ideological shift in the life sciences, which focuses on maximizing individual responsibility of health and surplus profit (Cooper and Waldby 2014). Dumit (2012), Cooper (2008), and Sunder-Rajan (2006) Peterson (2014) show how the speculative movement in capitalism(s) and the life sciences focuses on the need to control future risk and future cost. Sunder-Rajan describes the future oriented projects in genomics as a "shift in grammar" that increasingly conceives of life in the future tense (2006:14). Justifying interventions in the present by speculating on future cost savings is characteristic of the neoliberal healthcare environment.

Clinical trials that test dietary interventions on pregnant women are guided by epigenetic understandings, *and* they are practiced in particular economic, political, healthcare environments.

The production of epigenetic science is occurring within a healthcare environment that is increasingly focused on redistributing state responsibility onto individuals and rationing healthcare (Wailoo and Anand 2000). Therefore, attending to how scientific practices emphasize certain environments at certain scales and times is important to consider when exploring the limits and possibilities of epigenetics.

**Conclusion**

Overall, I have argued that the methodologies, disciplinary expertise, and data collection priorities of the StandUp trial all target narrow scales of the environment. The focus on particular scales of the environment has implications for the knowledge and practice of epigenetics. Despite the growing epigenetic research reflecting the complex, unpredictable entanglements between epigenetic modifications and environmental stimuli at different scales, there are more clinical trials that target the eating behaviors of individual pregnant women now than ever before. These trials often have inconclusive results and more research is emerging that interventions should occur before conceptions, rather than during pregnancy (Sohni et al., 2014). The conditions that make it possible to justify research on dietary interventions during pregnancy are directly related to neoliberal healthcare and research climates. The question that emerges then is what specific scientific, economic, and political environments may enable the production of epigenetic knowledge that evenly examines different environments at different scales?

Although the number of trials on diets and lifestyles before, during, and after pregnancy are growing, it is not known whether the interventions are clinically significant.[65] Further, the individual bodies that have traditionally been targeted – not just for the sake of reducing risk and

[65] Other trials in the U.K are testing the intervention of giving obese pregnant women metformin, a drug for diabetes, during their pregnancy instead of a behavioral intervention.

cost in the future, but also for maintaining a strong labor force for nations and militaries--are diverse pregnant bodies. As with older strategies of intervening and monitoring – in the name of future citizens – developmental origins echoes the same focus on the future with the added attention to scales of the environment. However, in the case of obesity during pregnancy, the older methods play out in new and nuanced ways. In the paradigm of epigenetics, the target becomes the uterine and molecular scales of the environment. The justification of intervening on bodies or environments remains the same. Focusing on the uterine environment and the eating behaviors of pregnant women obscures the larger environmental scales that epigenetics is supposed to help us expose. An emerging literature, which I expand on in the next chapter, cautions against using epigenetics or DOHaD to blame mothers for adverse outcomes in their children (Richardson et al., 2014, 2015), or reifying gendered notions of motherhood (Muller and Kenny forthcoming).

Paying attention to scale is also important for understanding how different forms and applications of epigenetics produce different epigenetic adaptations or versions of epigenetic. This chapter and the following one support the idea that different versions of epigenetics are informed by the methods and philosophical approaches that principle investigators use in their clinical trial design. In addition, this chapter emphasizes the contradiction inherent to the knowledge and practice of epigenetics, and reflects the different versions of epigenetics that exist in practice. In the next chapter I highlight the different approach and expertise that influenced the design and implementation of the SmartStart trial. Together, Chapters Two and Three gesture at a common theme: epigenetics is not a homogenous paradigm, and the different disciplinary approaches to epigenetic logics result in different adaptations of epigenetics. Moreover, by

illustrating the different epigenetic adaptations that exist, I am underscoring how epigenetics itself is multiple and emergent, or still in the process of becoming.

## Chapter 4:

## Relationality: Nature, Nurture, Nutrition, and "Good" Mothering

Social scientists and scientists alike are approaching epigenetics from many different disciplines. Throughout the ethnography I argue that epigenetics is not a monolithic or homogenous paradigm, but rather a collection of *epigenetic adaptations*. These adaptations of epigenetics are derived from different disciplinary approaches and expertise. In the last chapter, I explored how Dr. Elizabeth's expertise influenced her approach to epigenetics. For instance, Dr. Elizabeth used animal models to design the StandUp trial and focused primarily on the molecular and uterine scales of the environment. Epigenetics has facilitated a common space of engagement for both PIs, but their distinct disciplines and expertise influences their interpretations of epigenetic concepts such that their designs and research questions are very different.

In what follows, I show how Dr. Sally's interpretation of epigenetics is guided by her focus on behavioral weight management during pregnancy. A key focus in Dr. Sally's approach to designing the SmartStart trial is the responsibility of the "mother" as a nutritional gatekeeper whose behaviors, she believes, greatly influence the social and biological development of the fetus, and future child. Dr. Sally draws from psycho-social theories and non-Mendelian forms of inheritance that are inherent to epigenetics. Individualizing nutritional responsibility solely on mothers emphasizes but one relationship—the maternal-fetal relationship—while denying a much larger relational system of food production, consumption, and economy. One similarity across both trials I examined is the focus on targeting individual women. What results is an examination of only certain scales of the environment, and not how these different contexts and scales of the environment are entangled.

Furthermore, both trials draw from the subfield of nutritional epigenetics to justify nutritional interventions during pregnancy. Nutritional epigenetics examines how nutrition or food can act as environmental exposure and modify genetic expression (Landecker 2011). By studying the development of honeybees molecular biologists have found a model case for nutritional epigenetics (Kucharski et al., 2008). All honeybees, whether they are sterile worker bees or fertile queen bees, develop from genetically identical larvae (Kucharksi et al., 2008). So at the larvae stage all bees have the same DNA. However, a modification occurs in early development that causes some larvae to become queen bees, and some to become worker bees (Kucharksi et al., 2008). Two phenotypically different insects emerge from the same DNA. The queen bee is much larger in comparison to the worker bee, and more significantly the queen bee is fertile whereas, the worker bee is sterile. Epigenetic scientists claim that the developmental change between worker bees and queen bees is a result of nutrition (Kucharski et al., 2008). The larvae that are fed royal jelly develop into fertile queen bees, and the larvae that are not fed royal jelly develop into sterile worker bees.

At the molecular scale, this process is not well understood. However, what is known is that exposure to royal jelly causes genetic differentiation between worker bees and queen bees. The case of the honeybees reflects nutritional epigenetics because royal jelly, or nutrition, acts as an environmental factor that epigenetically modifies DNA regulation and expression. In addition, there is the potential to inherit this modification across different generations. Although honeybees never have more than one queen bee in a colony, there is always the potential for other larvae to become honeybees if the environment, or royal jelly, stimulates epigenetic changes. Moreover, the process of "making" queen bees throws into relief the roles that nature, nurture, nutrition and the environment play in epigenetics.

Although each PI draws from nutritional epigenetics to design and justify their interventions their interpretations are distinct. For instance, at the StandUp trial if a pregnant woman experiences a spike in sugar in her blood due to eating a donut, the assumption is that the developing fetus might be exposed to the cascade of reactions related to glucose metabolism. This process is framed as an environmental stimulus that can influence fetal programming (Barker 1986). However, that same interpretation of high blood sugar is not a targeted environmental factor in the SmarStart trial. The SmartStart trial targets calorie control rather than glycemic control. The different interventions reflect the distinct epigenetic interpretations and applications. Nevertheless, regardless of their differences, both trials engineer the diets of pregnant women based on presumptions that such efforts will prevent obesity and diabetes in the children born.

Prenatal nutritional interventions are a site of ethnographic inquiry because they reveal how different scientists are using particular assumptions about gender, mothering, and nutrition to design nutritional interventions for pregnant women. I argue that through prenatal interventions older and gendered notions of mothering and nurturing behaviors are instantiated within contemporary explorations of epigenetics. To do so, I focus on how the significance of nutritional epigenetics interacts with existing psychological approaches to nutritional interventions and weight management. In addition, I underscore the point that although dietary interventions draw from epigenetics to test interventions of diet and exercise on pregnant women, only particular aspects of nutrition and nurturing behaviors are targeted. For instance, the SmartStart trial does not distinguish between different types of nutritional substance, but rather focuses specifically on caloric intake only. Next, I examine a taken for granted concept of motherhood that is used to frame and structure prenatal interventions.

*Motherhood and Epigenetic Science*

Feminist scholars that critically examine epigenetics highlight how women's bodies and behaviors are framed as risky environments in epigenetic science. For instance, Sarah Richardson and her colleagues warn scientists against framing pregnant women as the sole culprits of their children's health or future disease. In an article in the journal *Nature* Richardson et al. write, "Society: Don't blame the mothers" (2014: 1). Her warnings echo similar concerns from past feminist scholars who trace mother-blaming discourses to well before epigenetics arose.[66] In particular interpretations and applications of epigenetics the focus on individual women draws on older ideas of motherhood, or nurturing behaviors, as environmental factors that can influence nature, or biological/genetic development, through nutrition or what pregnant women eat during pregnancy. Therefore, I explore how the relationship between nature, nurture, and nutrition is reinscribed into epigenetic interpretations that focus solely on targeting women's bodies and behaviors.

Nature, nurture, and nutrition index different kinds of relationships. For instance, the way "nature" and "nurture" are put into relationship stretches into a long debate in social, scientific, and philosophical scholarship between the divisions or entanglements of natural facts and social forces. The idea that biological development is influenced more by nature (which comes increasingly under the sign of genetics) than by nurture (such as environmental conditions, including social, historical, economical, legal, cultural forces) has roots in the classic theories proposed by Lamarck and Darwin. In another vein, "nurture" is often evokes concepts of motherhood and mothering, or maternal behaviors. While treated as a biological given the notion

[66] For instance, feminist historians and anthropologists note that the emergence of population health established a state endorsed project of targeting pregnant bodies and mothers as a way to maintain future citizens and soldiers (Davin 1978).

of motherhood and associated "instincts" of nurturance is a socially and historically constructed concept that connects biological ability to reproduce with social expectations of kinship, responsibility, and labor. What it means to be a "good" mother is not stable cross-culturally or historically (Scheper-Hughes 1992; Zelizer 1985). "Bad" mothers take various forms, depending on cultural and historical contexts, and are often illustrated as having cold and neglectful behaviors. The popular media portrayal of "tiger-moms," or moms who are demanding, strict, and disciplinary, are also linked to overachieving, anxious children and adults (Peck 2015). What counts as "good" or "bad" mothering is also shaped by race and class. For instance, as Dorothy Roberts argues, Black women in the United States have been historically framed as the "bad" mother type (1991).

Producers of scientific knowledge draw from cultural discourses about mothers that perpetuate similar associations between "good" mothers and nurturing mothers. For instance, Haraway provides a rich analysis of the Harlow studies, which examined the connection between "mother love" and "good" primate mothers in an experimental behavioral context (1989). The studies looked at the effects of child development among primates if they substituted the "mother" figure with wire structures, or warm cuddly figures. Harlow's studies found that attachment and "mother love" was emotional and cultivated during the "critical period" of early childhood, and was not "natural" or instinctual (1959). The results of these studies were used to construct standards and psychological conceptualizations on how an individual's relationship with her mother can influence social and emotional development. His work was also used to inform adoption theory and attachment parenting.

Moreover, epigenetic science examines mothering styles and behaviors from a slightly different perspective. Like the Harlow studies, studies in epigenetics draw from animal models to

understand how "maternal behavior" influences not just social and emotional development, but also biological and genetic expression. For instance, one study, titled "Epigenetic Programming by Maternal Behavior," examines the relationship between maternal behaviors in rats and epigenetic modifications (Weaver et al., 2004). The results of this research presume and articulate a relationship between a mother's postnatal behavior and long-term biochemical changes in the offspring. The story goes like this: a "mother rat", which is the term that Weaver and his colleagues use, licks her offspring during the first week of life. The act of licking sends a signal to the brain of the offspring and causes methylation, acetylation, and histone modification to the genetic sequence. Therefore, the offspring that were licked more, during early development, had a different biochemical response to stress than rats that were not licked. The authors found that this modification could be reversed and that it had a lasting impact up through adulthood (Weaver et al., 2004, Weaver et al. 2006).

The study shows how a stress response is "inherited" by the offspring, after birth and outside of the uterine environment, through the female rat's behavior. This model focuses not on the uterine environment but on behaviors between rats and offspring immediately following birth. Studies such as this suggest that the relationship between rats, behaviors, and stress responses outside the body collapses the divisions between nature and nurture. They also redefine the relationships purportedly implicit to inheritance. By doing so, epigenetic science emphasizes the interconnectedness of inheritance across bodies and behaviors, or what I am exploring through the concept of "relationality."

However, the role and potential influence of the mother's behavior on the offspring's development maintains significant consequences for the gendered responsibilities placed on mothers to behave in ways that produce healthy and well-adapted offspring. The important

aspect that the rat licking study also highlights is the reversibility of inherited behaviors through pharmacotherapy or cognitive behavioral therapy. If the inheritance of adverse stress reactions can be reversed, then perhaps other inherited aspects can be reversed by intervening on maternal behaviors. In the trials I studied, nurturing behaviors are explicitly linked to nutrition, or the ability to provide health nutritional environments in utero and in early childhood. For example, the role of a mother as both nurturer, and individually responsible for providing nutrition is the main target for intervention at the SmartStart trial. It is through the collapsing of nature, nurture, and nutrition that individual eating habits during pregnancy are targeted for intervention.

In response to research that analogically relates human maternal behavior with laboratory animal models, social scientists bring attention to the fact that women's behaviors and bodies have become the key targets in the epigenetic paradigm (Richardson 2014, 2015). Martha Kenny and Ruther Muller critically analyze the ways in which epigenetic science has taken up gender roles of mothering as forms of nurturing and ways of influencing nature. Upon reflecting on the Weaver studies, the authors write:

> The mother increasingly comes to stand in for the whole environment of the infant rat. Her actions determine what kind of rat her pup will become [….] mother nurture programs her children's epigenome and determines much of their fate (Kenny and Muller forthcoming).

Instead of using the phrase "mother nature," Kenney and Muller use "mother nurture" to show how epigenetics emphasizes a mother's nurturing behaviors as significant factors of biological development. In addition, Muller and Kenny bring attention to the idea that within an epigenetic paradigm, maternal behaviors are the environment for the offspring. However, a limitation of using epigenetics and mice models is the universal assumption underlying the concept of maternal behavior. In practice, epigenetic logics that reconstitute forms of inheritance and

relationality are used to target maternal behaviors rather than the entangled scales of epigenetic environments. The only relationship and environmental scale that matters in the development of the offspring are pregnant bodies and maternal behaviors. All of which, imply particular understandings of fetal-maternal and child-maternal relationships. A narrow approach to epigenetics misses the complex relationality that exits in families and societies. In addition, animal models typically completely exclude paternal roles and responsibility. Studies that examine paternal relationships in mice models are limited, which parallels the limited examination of parental roles in human models. Moreover, the focus on maternal behaviors assumes that mothers are completely independent from other forms of relationality that also influence maternal behavior.

By using animal models, the gendered and social-constructed aspects of motherhood are completely missing. Are maternal behaviors universal? Can maternal behaviors be compared across rats and humans? Are maternal behaviors linked to women's bodies and behaviors only? How would queer families fit into the Weaver study? By leaving these assumptions unexamined, epigenetic science is simultaneously harnessing persistent naturalizations of gender, and framing motherhood as a biological and environmental factor for development. Further, using concepts like motherhood and maternal behaviors in an uncritical way facilitates the (re)production of scientific knowledge that is gendered and socially biased. The question still remains, who decides what motherhood is?

Another implication of uncritically translating naturalized assumptions about maternal behavior into studies involving epigenetic is the increased surveillance on what counts as "good mothering." As I outlined in the Introduction, the biopolitical justification of intervening on women's bodies in the name of future citizens is a well-established legacy in public health and

child-maternal health (Rapp and Ginsburg 1991; Foucault 1978). However, in the case of obesity during pregnancy, older forms of state surveillance are manifesting in new contexts. Through epigenetics, intervening on pregnant bodies may impact biological and social development in the present and in the future. In what follows, I explore how Dr. Sally draws from her expertise as a health psychologist, to approach epigenetics, and how that influences the design of the Smart Start intervention.

*Epigenetic Adaptations: how psychosocial approaches to obesity during pregnancy became a part of the epigenetic paradigm*

The PI of the SmartStart trial is a psychologist by training and has placed a huge emphasis on the design of the behavioral intervention. Her expertise draws from multiple fields including statistics, nutrition, epidemiology, and health counseling. She worked extensively in weight management clinics to improve the health and quality of life of individuals. Dr.Sally is also a co-investigator on three other clinical trials in addition to the SmartStart trial. Within the epigenetic context Dr. Sally's long-term work on behavioral interventions and weight management is relevant for new reasons. For instance, through epigenetics, Dr. Sally's focus on obesity during pregnancy has come to matter in the prevention of future epidemics. Similarly, the role of behavior-- specifically dietary behavior--assumes new roles in understandings about what nurture and nutrition mean during pregnancy.

Twenty years ago, when Dr. Sally began her work on behavior, diets, weight loss, and pregnancy, the research was situated primarily in the field of health psychology. With the emergence of epigenetics, her work on maternal nutrition and behavior is spotlighted as the site to examine how nutrition during pregnancy changes genetic expression in fetal development; *and*

how maternal behavior in early development may also change genetic expression. Both maternal nutrition and behavior are framed as epigenetic environments that influence fetal and child development.[67] Further, Brenda, a consultant on the SmartStart trial, explained that maternal nutrition used to be seen as trivial in the public health community up until the 1990s. However, once the obesity epidemic became a public health concern in the late 1990s, people started to pay attention to women's weight and nutrition during pregnancy. By 2009 the Institute of Medicine (IOM) published an updated report that reevaluated the 1990 gestational weight gain recommendations. Brenda says that the 2009 report reflects the influence of epigenetic theories because it specifically highlights the role of maternal nutrition in the future risk of obesity, not only for the mother, but also for the developing child.

In an interview with Dr. Sally, I asked her how she came to research obesity during pregnancy. She knew she wanted to work in psychology because doing so would give her the opportunities to work with patients, do research, and analyze data. She came into examining obesity somewhat by accident. She states that she became "frustrated with self-reported outcomes" in psychology research because they focused primarily on asking people "how they feel." In Dr. Sally's perspective, self-reporting leads to bias because people can lie and have their own biases. Therefore, she was attracted to obesity "because you have this objective measure of weight, which is reflective of behavior." Weight, as Dr. Sally claims, is an object measurement of behavior. Therefore, her focus on weight became a key theme throughout her research.

[67] During my observations of the bio-specimen steering committee, in which key PIs of the consortium discuss plans for how to collect various bio-samples, Dr. Sally told me that sometimes there is a division between what is framed as more important or more complicated to collect the bio-specimen or the behavioral data. Dr. Sally argues that both kinds of data are complicated to collect. Her focus is on collecting the behavioral data in a systematic way. Due to her training in psychology, she believes that the behavioral data is just as important, and her interested in studying obesity during pregnancy stems from the fact that obesity lies at the intersection of biology and behavior.

The first clinical trial that Dr.Sally led was an intervention on normal and overweight pregnant women aimed at preventing excessive weight gain. The study began in 2004 and, at that time, there were just a few other studies on obesity and weight during pregnancy. Since then the number of studies that examine behavioral interventions of diet and exercise have exploded in the US. However, as Dr. Sally notes, they all have "small sample sizes."Therefore, no matter the quantity of studies, there is a limit to the clinical significance of the studies. In the study that she designed in 2004, she assumed that "just telling women what to eat and what not to eat" would be enough of an intervention to prevent excessive gestational weight gain. However, the study was not successful and she concluded that the intervention was not rigid enough to have any significance. As a result of her previous experiences, the SmartStart trial included a highly structured behavioral and environmental intervention.

To better understand how Dr. Sally approaches the design of behavioral interventions I asked her about her research philosophy. Dr. Sally stated that she focuses on behavioral and environmental influences on obesity, but since she started her work on pregnancy she started to learn more about the biological side. In her words, obesity during pregnancy reflects the interaction of biology and psychology:

> In psychology there is lot of work showing that personality traits are mimicked across a generation, or they skip a generation, on the maternal side, so a lot of the behaviors in a grandma can be found in her grandchildren, and seeing the same thing in biology, is just really interesting, I see the parallel between the biology and the behavior.

Dr. Sally's reference to the parallels between psychological personality traits skipping a generation on the maternal side, and biological modifications that also skip generations, is reflective of non-Mendelian inheritance in epigenetics. The fact that personality traits and metabolic syndromes, for instance, can both be inherited across generations is the entanglement

of the psycho-social and biological that Dr. Sally finds in her work on obesity during pregnancy. To illustrate a different example of the interaction of the biological and behavioral, Dr. Sally explained:

> GDM (gestational diabetes mellitus) is prevalent if your maternal grandma had GDM. So your chances increase if your grandma had GDM, and you see these things in cancer as well this skipping the generation. We don't know why, and I see the parallel between the two as very close and very interesting. So I think my approach is bio-behavioral, psychosocial, and as comprehensive as possible; seeing the individual within it, and trying to study all these different interactions, and influences. To do this, we try and see what we can and can't change, and the effect of those changes.

Dr. Sally's bio-behavioral or psychosocial approach fits well in the epigenetic paradigm. Her understandings of the trans-generational inheritance of behaviors or personalities, and gestational diabetes are aligned within emergent epigenetic science. Moreover, her reference to GDM is another example of how she is drawing from both epigenetic science and psych-social approaches in health psychology to understand diabetes and obesity. Dr. Sally's research is particularly relevant now because she focuses on prenatal and post-natal interventions of diet and exercise on women. The target population of new epigenetic understandings of obesity and diabetes are women of reproductive ages (Richardson 2014).

Although, some literature suggests that paternal diets are important to consider. From my observations and interviews, it is clear that the fathers are much harder to recruit to clinical trials. Both the StandUp trial and the SmartStart trial intended to recruit the pregnant women's partners, but the success rate was minimal. When I left in 2014, they were still trying to find ways of getting the men to participate. There is a dearth of research related to recruiting partners into trials during pregnancy. Unlike women, men are not systematically and medically surveyed

or monitored during their lifetimes.[68] It is through these longstanding infrastructures of medicalization and surveillance that make it possible to recruit and intervene on women's bodies.[69] However, if we take epigenetics seriously the relationality across generations on both the maternal and paternal side are significant for epigenetic modifications. Next, I explore an epigenetic framing of nutrition as exposure and Dr. Sally's approach to nutrition as exposure to illustrate the new overlaps and adaptations that exist in Dr. Sally's research and engagement with epigenetics.

*Food as Exposure and Exposure to Food*

To design dietary interventions on pregnant women, Dr. Sally takes both behavior and environment into account. In her words "the intervention is an intensive environmental manipulation because we are reducing exposures to food, and so it is a pretty intensive approach to reduce intake of food – we tell them exactly what to eat, when to eat, and what not to eat, and give them something to use instead of eating." Dr. Sally's application of the phrase "exposure to food" has a different meaning than Landecker's "food as exposure."

In Landecker's article "Food as exposure: nutritional epigenetics and the new metabolism", she asks the question, how does food become an environmental factor (2011)? For Landecker, the reconceptualization of food as environmental exposure indexes a fundamental shift in how epigenetics has changed our understandings of the environment – not necessarily of food. In the same article, Landecker claims that the conceptualization of "food as exposure" is nothing novel, but rather that epigenetic science is a site of a particular social and historical

[68] Moreover, all the "families" were understood to as heter-normative and did not include queer families.
[69] One may wonder whether women's partners are also not considered significant parts of the women's environment. The literature on women as "nutritional gatekeepers" justifies the inclusion of men into the trial, because the assumption is that if you change the women's diet, there will be an effect on the partner's eating habits, not the other way around.

understanding of food as exposure. She supports this claim by providing different approaches to food and bodies throughout history. She draws from work in the early nineteenth century that connected certain foods with nutritional value for brain development. In the twentieth century the emergence of nutrigenetics focused on the ways in which bodies absorb nutrition and the molecular interactions involved in metabolism. It was also in the twentieth century that folic acid became mandatory as part of prenatal nutrition. However in an epigenetic age, nutrition is framed as changing gene expression in critical periods, which can affect future generations.[70] Thus, Landecker argues that food as exposure has existed in one way or another throughout history; epigenetics is just one particular moment in our social and cultural understanding that frames food as exposure in a slightly different way.

Similarly, for Dr. Sally, food as exposure existed as a concept prior to the emergence of epigenetics. When I asked what exactly she meant by "reducing exposure to food" she responded by saying that "by having a meal replacement shake [which is part of the SmartStart intervention] you're not having ham and cheese and bread, and mayonnaise, and mustard, and everything else that you could put on a sandwich, you just have this one thing, and you don't have all the excess food in the house […]."In Dr. Sally's perspective reducing exposure to food functions as a way to limit excessive eating behavior in pregnant women. Limiting access to food also reduces the risk of obesity and diabetes in the mothers and children. Food as exposure and exposure to food seem to be two sides of the same coin. Limiting the food pregnant women are exposed to both decreases their weight, and therefore in the SmartStart trial, is assumed to decrease the risk of obesity and diabetes for the pregnant women and their children. Through

[70] Critical periods are defined in the epigenetic literature as a time and space that is most vulnerable for genetic modification. For instance, the previous chapter explained how critical periods have come to frame pregnancy as an important time for interventions because it is a vulnerable time/space for epigenetic modifications.

epigenetic logic, if food acts as an environmental factor that can expose genetic material to manipulation and change, then no-food or limited food is another way of doing the same.

The significance in detailing the parallels in food as exposure and exposure to food relates to the idea that the role of food during pregnancy has both changed as result of epigenetics as a growing discipline, and not changed in other disciplines such as health psychology, public health, and child maternal nutrition. The dialectical interaction between the old and new iterations of nature, nurture, and nutrition are similar to the forms of surveillance and the assumptions of gender that are enveloped into the production of emergent epigenetic knowledge.

For instance, Landecker's analysis of food engages directly with epigenetics to show how nutrition and exposure has endured and taken different forms prior to epigenetics. For Dr. Sally, nutrition as a kind of psychosocial or bio-behavioral factor has also been an object of study for decades in health psychology research and obesity research writ large. Both of these forms of food as exposure are coming out of distinct and different disciplinary perspectives; however, they are applied in the same context of RCTs aimed at changing pregnant women's diets. Limiting food intake has traditionally been the advice for decreasing obesity and diabetes in adults, but targeting pregnant women to reduce their caloric intake and their exposure to food draws on older concepts functions from traditional psychosocial approaches to obesity; and also draws from epigenetic science. Ultimately, the recent justification for dietary interventions during pregnancy is directly related to nutritional epigenetics. It just so happens, that Dr. Sally's approach aligns itself well with nutritional epigenetics.

The other reason for explaining the overlaps in psychosocial approaches and nutritional epigenetics is that, as Muller and Kenny write, mother's bodies and behaviors take the place of

the environment in epigenetics. In the SmartStart trial, the design of the intervention aims at manipulating pregnant women's behaviors and home environment, which is supposed to change women's weight during pregnancy and fetal development. The home environment is both a psychological factor in changing behavior, and a scale of epigenetic environments that can also influence biological and social development. In the same interview with Dr. Sally, I asked her how the SmartStart trial intervenes on women's behaviors and environments. She responded by stating:

> I think we can change their home environment and I think that is exciting because of the babies who are going to come and play in that environment and growing and developing in that home environment. I think the women in the intervention will or I hope, or we hypothesize that they will have a more healthy home, food, and exercise environment […].

The SmartStart intervention aims to intervene on the home environment to create a healthy and active environment. She expanded on this more in the following excerpt.

> On a basic level, we are giving the women scales, measuring cups, pedometers. We give them some [behavioral/environmental] cues, give them handouts, folders, bags, all those kinds of cues. We are asking them to exercise, so that usually leads to more sneakers around the house, or gym clothes around the house and yoga tapes and exercise tapes. We talk about, I guess, in the newsletters about having an active environment, for family and kids, having basketballs and toys like that, more active toys. We can manipulate it there, but I think in this study it is more exercise cues, and some dietary cues, meal plans and shakes. That is manipulating their home environment. We tell them to do a cabinet clean out; to take out all the junk from their home […]. [We change] how they interact with each other, how they serve meals, and not doing a buffet type meal, but having serving yourself portions, not having all the food in the middle of the table, so that is kind of a manipulation.

Dr. Sally's behavioral and environmental approach to changing women's environment is relevant to the relationality of nature, nurture, and nutrition because it depends on certain gendered assumptions of motherhood and class. Assuming that women are individually responsible for

creating the "home environment" does not consider the other significant relationships that shape the living conditions of people in the "home environment." In addition, the focus on the "home environment" assumes that there is a stable home to begin with, which is becoming less and less equitable in precarious housing markets. Notions of mothering and nurturing are gendered and socially constructed, yet they are inextricable bound to very premise of nutritional epigenetics. If mothering is associated with creating an environment that is "fit" for healthy behaviors and bodies, then access to housing and affordable produce should also be considered a factor associated with good mothering/parenting.

*Introduction to the Start Smart Trial*

Dr. Sally personally designed and wrote the intervention-training manuals for staff, and the lessons for each intervention visit with the participants. Every week she meets with the interventionists or nutritional counselors to review the progress of the participants in the intervention group. Drawing form the 2009 Institute of Medicine (IOM) guidelines, the SmartStart trial hypothesizes that the intervention will help women gain the recommended amount of weight each week. The weekly gestational weight gain goal is a half a pound or less. If the women reached the IOM recommended weight gain goal, then the women in the intervention group would have lower rates of pregnancy complications and their children would be healthier. Overall, the purpose of the trial was to examine epigenetic effects of nutritional interventions during pregnancy with regards to prevention of gestational diabetes through measures of maternal obesity.

Dr. Sally intends to show that diet and exercise interventions, specifically ones that focus on weight and calorie control, are effective in diverse pregnant populations. Consequently, the

trial aims at recruiting "50% Caucasian women and 50% Hispanic women" (SmartStart protocol 2012). Most of the women that I observed were Mexican-American and Latina. All but three members of the staff were Mexican-American and spoke Spanish. In addition, the intervention was delivered in Spanish and English. To be inclusive the trial included information and examples using "ethnic foods" like tortillas, mole, and tacos.

The women in the experimental group in the U.S are required to: weigh themselves everyday; write down every meal that they have; count their calories; adhere to a strict calorie plan which includes, replacing breakfast and lunch with meal replacement beverages that are provided to them; meet with an interventionist counselor every two weeks to discuss self-monitoring strategies; do 30 minutes of physical exercise everyday; and, work towards a goal of 10,000 steps each day. The intervention is rigid by anyone's account, however, the participants are not forced to do anything, and compliance is a key issue in the implementation of this intervention.

When I worked as a nutritional counselor for the US trial, we were trained to calculate a specific calorie plans for each woman, which we used to create a customized meal plan.[71] For example, a meal plan consisted of two meal replacement shakes for breakfast and lunch, which could be eaten with a piece of fruit. The Start Smart Trial uses meal replacements as a form of calorie and portion controlled meals. These liquid beverages are intended to contain all the necessary or "healthy" nutrients required for pregnant women. Plans for dinner were based on what the woman cooked or made for herself. There was also a hand out for "free" foods, which

[71] The calculation was based on a woman's weight during her first visit as an enrolled participant and you multiplied her weight in kilograms by eighteen, the calculation was that every woman needed 18kcal of energy per kilogram of weight. For instance, if a woman weighed between 165-189 pounds at the beginning of her pregnancy she was prescribed a 1400kcal meal plan.

meant that some foods did not have calories; those consisted of lettuce and some other vegetables.

In addition to creating a meal plan, as a nutritional counselor, I would go through the pregnant participants' food journals, in which they wrote down everything they ate and their daily weight reports, and I would weigh them at each visit. I monitored the pregnant participants' weight by using a graph that was taken from the maternal weight gain recommendations published by the (IOM). The X-axis on the graph represented each week of pregnancy, and the Y-axis was the weight gained. The graph included a line, which represented the IOM weight gain goal of a half of a pound per week throughout their entire pregnancy. If they were gaining too much I was told to send the women a letter with a card that said "Slow down: your rate of weight gain is above ½lb each week."[72]

In addition, the training manual stated that we should inform the participants about the benefits of prepackaged foods. The training manual told us to reinforce the use of prepackaged meals such as Lean Cuisine as a strategy for making self-monitoring easy. This is the script we were encouraged to tell the participants:

> If you aren't a big fan of math, what's the best way to make sure you are meeting your calorie goals? Follow the meal plan. The Meal plan makes counting calories MUCH easier. These are calorie and portioned controlled and make meeting calorie goal much easier because you don't have to keep looking up very many foods each day! Other benefits to using pre packed foods are:
> No food preparation required
> Easy and convenient, you can shop for groceries less often. Prepackaged products can be stored for a long time. You can shop every other week and have time to do other things.
> Less exposure to food that might tempt you
> Easier keeping track: there is no need to look up calories of many different types of food

[72] We were reminded at each intervention meeting with the PI to send these "reminder cards" to the pregnant participants but most of the interventionists would forget to send them.

The emphasis on controlling calories and portions limits exposure to food. Dr. Sally, specifically claims that meal replacement shakes and pre-packaged foods can limit the exposure women have to food. This aspect of her design is further explored below. Also, the underlying focus on controlling calories and portions reflects a calorie in/out framing of nutrition.

The meal plan, weight gain goals, and daily self- monitoring all emphasized food and nutrition as calories. For instance, if a woman ate ramen noodles and fruit loops for the day but maintained her calorie goal, it was considered a success. The goal of the intervention was to make sure each participant gained within 1/2 pound per week. If other issues came up about food insecurity or access to affordable or fresh produce, I was told to brainstorm ideas on how they could keep within their calorie goal. I was also told to explain to the participants that they should have fruits, vegetables, protein, and a certain amount of carbs, but usually each visit was spent on evaluating what challenges the pregnant participant had experienced since our last visit.

From my experiences as a nutritional counselor, along with the interviews with other nutritional counselors, the intervention visit was rarely about the food that they ate. That is, the intervention visit itself was entangled with the pregnant women's families, work schedules, transitions, unemployment, evictions, and childcare. *The nutritional interventions were less about the food itself and more about managing life in general*. These particular aspects of social, economic, and environmental factors that influence maternal nutrition are listed as factors on the IOM's schematic of what affects gestational weight gain; however, the diet intervention is not intended or structured to address any of these issues.

*Destiny's Story*

Destiny ‘s story represents a composite of different women´s experiences with whom I worked. Most of the pregnant women I spoke to had one or another issues that are represented by Destiny’s story. Creating an ethnographic character, like Destiny, allows me to illustrate the challenges of a narrowly conceived epigenetic trial, while also honoring the anonymity of all the participants I worked with. Her story highlights how the trial prioritizes her individual relationship to food and calories, rather than the relationality across the social, environmental, and economic scales of context.

Destiny identified as “Hispanic” for the purposes of the trial. However, her ethnic identity was much more complicated than the “Hispanic” category entails.[73] Her parents were Puerto Rican and Mexican and she was the first generation born in the United States. I met with Destiny every two weeks, and although our sessions were supposed to focus on her diet and exercise, I ended up learning a lot about her life. Destiny had three kids and was pregnant with a fourth. She was active in her church and ate at the weekly potluck, which was one of the only cooked meals her family had each week. Money was tight since Destiny and her husband were unemployed. They lost their jobs around the 2008 economic crisis. Destiny also suffered from depression, insomnia, and, at twenty-four weeks gestation, was diagnosed with gestational diabetes (or GDM). After she was diagnosed with GDM, she also met with a dietician every other week and a diabetes specialist. All together she had about eight health appointments each month including her participation in the clinical trial. Near the end of the pregnancy she was also struggling with eviction and eventually she and her family moved out of their apartment to live with some in-laws.

[73] The term “Hispanic” was first used in the U.S. census under Nixon’s presidency. One reason for creating the new racial/ethnic category had to do with the political motivation to assess and target more voters.

As social scientists have argued, stress, like the stress of unemployment and unreliable housing can impact health (Gravlee 2009; Mullings and Wali 2001; Sapolsky 2004). In addition, the fields of health science, medicine, genetics, and epidemiology all make a case for why stress is an important health factor (Marmot et al., 1991). In a 2014 report by the Institute of Medicine they examined what significant factors should be included in electronic medical records for patients. The IOM committee concluded that stress should be included in the comprehensive evaluation of health. However, stress was not a significant factor in the SmartStart trial, nor was it assessed in anyway during the implementation of the trial.[74]

At the weekly staff meetings with the principal investigator, I presented Destiny's case and the significance of her mental, emotional, and physical living conditions. However, I was reminded that I had to focus on delivering the intervention as it is delivered to everyone in a standardized manner. This moment reflects what is measured and what is not, or rather what environmental factors are important to target and what are not. For instance, food may be an environment, using the meal replacement shakes may reduce Destiny's exposure to food, and the intervention might try and change her home environment in various ways, but the stability of her income or housing are not considered measurable environmental factors in the RCT. More importantly, the ways in which these different scales of environment are entangled or related to Destiny's living conditions is left unexamined.

Sometimes I felt silly asking Destiny about her food journal, calorie goals, and steps she walked that week. She usually preferred to talk about the immediate and material challenges she faced every day and I would listen. Despite all her stressful days, Destiny kept coming to meet

[74] Mental health and depression were evaluated before participants were randomized into the trial. The trial used a standard questionnaire to assess depression. If the women scored high on the test, then protocol required that they be referred to a health professional, and not enrolled into the study. However, Destiny's case reflects, stress and depression can emerge throughout pregnancy.

with me every two weeks throughout the duration of her pregnancy. In the end she delivered a healthy baby, and she met the trials weight gain goals. The study followed up with her in six months and they weighed her and her baby and took bio-samples for testing. The biomarkers that they test will indicate whether or not the nutritional intervention changed any genetic expression in the babies. Similar to the biomarkers analyzed at the StandUp trial, Destiny's metabolic levels in relation to sugar, fat, and insulin will be assessed through the blood samples taken during pregnancy. Then after six months, Destiny's child will be tested for similar metabolic markers that should tell the investigators if Destiny's metabolic status influenced the development of the child. However, what remains unchanged are Destiny's precarious, unstable living conditions.

The most salient parts of Destiny's context and behaviors were not the parts addressed by the trial. As the literature on the social determinants of health affirms, Destiny's housing, income, and employment influence her health and that of her baby. Destiny commented that fast foods were often cheaper than fresh produce. When I asked her about her steps and how she could fit in a few walks during the day she told me that she does not feel safe walking around her neighborhood. In addition, meeting the physical activity requirements were difficult because Destiny suffered from insomnia and depression, which made it difficult to feel motivated to exercise. The intersection of epigenetic understandings of the environment coupled with the priorities and practical realities of the RCT, make it difficult for poor, minority, overweight pregnant women like Destiny to comply and adhere to the intervention. Although, it was difficult Destiny still kept attending, and even though she did not completely comply, she still made an effort to participate in the trial. She came to meet with me and sometimes she would count her steps, or count her calories, and sometimes she would use the meal replacements shakes.

As an interventionist on the trial, I felt that two different understanding of the environment competed for my attention and care. One the one hand I saw the multiple and interconnected scales of environments that influenced Destiny's living conditions and her health, and on the other hand as an interventionist on the SmarStart trial, I had to focus on Destiny's calories as the key environmental factor. The SmartStart trial focused on food as exposure and the women's behavior as significant factors to intervene upon, not the relationship between Destiny's eating behaviors and her living conditions. The focus on nutrition in the form of calories individualizes responsibility and elides the entanglement of other factors that influence Destiny's diet and health.

The intense effort to use nutritional epigenetics as a way to target maternal behaviors and eating habits, maintains implications for the treatment of pregnant women and for the production of epigenetic knowledge. Through epigenetic inheritance we can understand that pregnancy is a critical period that shapes genetic expression and regulation that can be inherited by future generations. We also can understand food as a key environmental factor. However, what we miss when we focus entirely on food or calories, is the entangled relationship between nature, nurture, and nutrition across epigenetic environments. Furthermore, focusing on changing what a pregnant woman eats during pregnancy in an attempt to reduce the risk of obesity and diabetes in the future ignores the existing probabilities of survival and quality of life that are unequally distributed among marginalized people of color. The justification for prenatal nutritional interventions is an expansion of older models of surveillance and management that control and direct individual bodies and behaviors, in the name of obesity prevention.

***Conclusion***

To conclude I will return to the honeybee story. When I introduced this story at the beginning of the chapter, I framed the story through my own words. This time I will tell you the story using the language of the scientific authors. Drawing from feminist anthropologists and science and technology scholars, I will analyze the scientific discourse of the passage.

> Fertile queens and sterile workers are alternative forms of the adult female honeybee that develop from genetically identical larvae following differential feeding with royal jelly […]. Young nurse bees in the hive produce and feed a largely biochemically uncharacterized substance named royal jelly to larvae *destined* to become queens, whereas the other larvae are fed with less sophisticated food (Kucharski et al., 2008: 1827 my emphasis).

One aspect that stands out from this description is that the authors characterize the nurse bee as the only individual responsible for producing and providing nutrients to the developing larvae. By focusing on the individual role of nurse bees, attention is displaced from other entangled relationships between honeybee behavior and environmental scales.

First, honeybees live and behave collectively, in relation to each other. The process of nurturing and providing nutrients does not depend solely on the nurse bee. There is a whole food production process that is missing from the scientists' description. The only way that nurse bees can produce royal jelly is if they have pollen. Worker bees are responsible for collecting pollen and feeding it to the nurse bees that then feed it to larva. There is a relational chain of food production and labor across nurse bees, worker bees, and queen bees. In addition, honeybees around the globe are experiencing significant environmental stress, which is causing colony collapse disorder. Colony collapse disorder occurs when worker bees randomly abandon their hives, leaving the rest of the colony to starve to death.

The relationship between nurse bees that produce royal jelly and human bodies that produce milk and gestate are similar in that both for bees and humans their behaviors are personified and naturalized by humans in relation to socially constructed notions of gender and

narrow framings of nutrition as environment. Further more, the scientific portrayal of nurse bees as the only nutritional providers for the colony is a narrow conception of epigenetics. The missing link is the relationality between nature, nurture, and nutrition, all of which also affect the development and "destiny" of honeybees – a link that also holds true for understanding maternal nutrition and infant health too.

For both honeybees and pregnant women, epigenetic science is used to explain only a fraction of the story between genes and environmental interactions. In the honeybees, the emphasis is on the food- royal jelly, and likewise in the clinical trials food, or nutrition in the form of calories becomes the main focus, but how the collective life of the hive effects the larval feeding process and how Destiny accesses quality food or expends her energy disappear from view.__

This chapter critically examined the assumptions implicit to applying epigenetics in prenatal dietary interventions, and the unexamined forms of relationality that are inherent to epigenetic logics. My analysis highlights how taken for granted assumptions about nature, nurture, and nutrition are folded into their questions and methods in the process of producing epigenetic knowledge. Using the concept of maternal behaviors in designing mice models assumes a universality and stability in how maternal behaviors are defined. The assumptions about motherhood are imposed onto mice models, and then the results from mice models, are then translated into human models, which reflects an epistemological conduit model. There is a (re)production of older notions of gender incorporated into new scientific understandings of epigenetics. Similar to mice models, my examination of the SmartStart design and intervention delivery reflects how the gendered and socially constructed roles of nurturing, and mothering are also left unquestioned in the human models.

In addition, this chapter and the previous one have illustrated how particular expertise and disciplinary approaches to epigenetics influence the ways in which epigenetics is deployed. Each expert focuses on different aspects of epigenetics that is most relevant to their background and methodology. In the case of the SmartStart trial, Dr. Sally's psychosocial and bio-behavioral approach to weight management engages with environmental and behavioral components of epigenetics, but not completely. The design of the trial stops short of engaging with the complex relational aspects of environmental scale. In the last chapter the different epigenetic scales were narrowed down to the uterine and molecular scales. In this chapter I show how the relationality of scales becomes individualized to maternal behavior, which in an epigenetic paradigm can affect inheritance outside the pregnant body

The selective and narrow conception of epigenetics applied in each trial is also a result of the methods that principle investigators are encouraged to use. My findings suggest that the scientific design of testing prenatal interventions using the randomized clinical trial method does not align itself with the kinds of questions that are inherent to epigenetics. If we take epigenetics seriously, then how are RCTs the right tool for the job? The "gold standard" of evidence-based medicine cannot capture the institutionalization of racism, oppressive histories of motherhood, trauma, and the potential for trans-generational inheritance across time-space, and bodies, all of which are significant and relational aspects of epigenetic environments. The trial protocol selectively targets individual variables like eating habits during pregnancy, but does not account for the social, gendered, and racialized milieu that influences "good" maternal behavior. In light of the racial disparities in health and the role that gender and the institutionalization of racism plays in the development of people's lives and opportunity, how can RCTs adequately capture the epigenetic inheritance of trauma and inequality?

In highlighting the taken for granted assumptions and implications that characterize different applications of epigenetics, this ethnography elucidates how the epistemological practice of epigenetics and the politics of practicing epigenetics ought to be called into question in prenatal interventions of diet and exercise. The application of narrow conceptions of epigenetics can help justify the individualization of eating habits onto pregnant women in standard prenatal care practices, which will also expand the surveillance and monitoring of pregnant bodies. However, if we take the epigenetic logic of inheritance and relationality seriously, it could be used to develop new forms of child maternal health that do not prioritize, universalize, hetero-normative, and gendered aspects of "good" mothering. Using epigenetic logics, how might we imagine parenting as a collective social practice that does not prioritize gender and nuclear families?

The next chapter draws on the thread of individuality that I have woven into this chapter to expose an inherent paradox related to the prioritization of individual responsibility, risk, and control. By doing so I challenge the assumption that individual pregnant bodies have full control over what they eat. Food choices are intimate and entangled with larger economic, political, and agricultural processes. Barbara Prainsack argues that individual choice is not realistic in a relational context. Prainsack's work focuses on the ethics of individual consent in personalized medicine and big data (forthcoming). She challenges the possibility of achieving individual consent through the reasoning that significant choices in life, death, and health are made in relation to others, not individually. Similarly, in the next chapter I argue that individual pregnant bodies do not have full control over what they eat or their environmental conditions, as we saw in this chapter through Destiny's story. Consequently, how can prenatal interventions justify the

individualization of responsibility onto pregnant bodies if these bodies are at once not quite individual, and not quite in control of their environmental conditions?

## Chapter 5:

## Risk, Responsibility and Control: The Pregnant Paradox

This chapter focuses on the narratives of the pregnant participants at the StandUp trial and highlights the risk, responsibility, and control that centers around their diets and weight gain. The common theme that brings all the actors together is the assumption that pregnant persons are responsible and in control of their diets and related risk to fetal development. The clinical trial reifies the focus on changing individual eating behaviors during pregnancy in attempt to protect and reduce risk for the developing fetus. In Richardson's examination of epigenetic theories, she draws from literature within the Developmental Origins of Health and Disease (DOHaD) to highlight a key contradiction in epigenetic models that focus on the maternal body.

> [Epigenetic] changes manifest at the level of the intergenerational lineage rather than the individual female. The significance is that DOHaD research advances a shifting and mixed message regarding maternal agency and responsibility: it exhorts mothers to make lifestyle changes in the service of their genetic lineage, while maintaining that these changes are unlikely to bring them or their offspring any benefit. At the same time, it produces a model of the maternal body that suggests that maternal experiences, exposures, and behaviors may have very significant, amplified consequences for her offspring, her descendants, and society at large (2015:224-225).

In this excerpt, Richardson is highlighting a key contradiction related to the responsibility and control implied and imposed on pregnant bodies. One interpretation of epigenetics frames then pregnant bodies as environments and entangled within different scales of the environment. Further, the trans-generational inheritance implied by epigenetics claims that risk of epigenetic changes can occur transversally across time and space. Or as Richardson describes, changes can manifest at the intergenerational scale, not at the individual scale. The message implicit to interventions during pregnancy is that individual women have the control or power to change

fetal development, which is not an accurate message according to epigenetic science. In fact, Richardson further states that individual women do not have "conscious control" over the unpredictable changes that can manifest in utero (2015:224).

Building on Richardson's work on epigenetics and the maternal body, I argue distinctly that it is not only epigenetic science that assumes or imposes individual responsibility on women. Rather, through the exploration of pregnant participant narratives I show that individual women also assume responsibility for their diets, and for how their diets affect the developing fetus. Exploring the narratives of the pregnant participants at the StandUp trial reflects complex issues of control and responsibility that converge at the site of the intervention. The models and justifications of DOHaD in nutritional interventions during pregnancy are not applied onto a blank slate. Rather, the focus on controlling and managing pregnant bodies in effort to protect future citizens is embedded into structures of medicine and research.

This chapter highlights the ways in which pregnant participants subject themselves to systems of management and control. By doing so, pregnant women find some comfort and guidance in managing all the risk and responsibility that comes with the ability to grow a fetus. Put another way, Richardson's work emphasizes the message that we should not make pregnant women solely responsible for the future health of their children (2014), but what I find is that the mothers willingly take on the responsibility as if they were in total control of their children's health and biological development. Participants who decide to enroll in nutritional interventions during pregnancy represent a part of the population that are willing to subject themselves to surveillance and management in order to deal with the social and biological risks and responsibilities that are associated with being an overweight pregnant person in society. For

pregnant participants in the StandUp trial, it could be epigenetics or any other theory, the assumption is the same: "mothers" are solely responsible for the health of their children.

Additionally, this chapter sets itself apart from current critiques and examinations of epigenetics because it provides empirical data from the perspective of pregnant participants that participate in epigenetic studies testing nutritional interventions. The literature on epigenetics and DOHaD has a dearth of information that explicitly engages with pregnant persons. This is evident in the trials themselves, which do not communicate the significance and stakes of epigenetics to the pregnant participants. The explicit discussion of epigenetics in the StandUp trial is limited to higher levels of elite scientific networks. The term epigenetic or the DOHaD concept is not mentioned or referred to by the staff or pregnant participants at either the SmartStart or StandUp trials. The selective communication of epigenetics is reflective of the dynamic that epigenetics is theorized in elite spaces, tested upon pregnant bodies, but not necessarily communicated with pregnant persons in a formal medical manner. One reason for this might be that trials would not benefit from disclosing the stakes, risks, and contradictions inherent to DOHaD.

Associating risk with what women eat during pregnancy is also an older framing – one that existed before the inception of epigenetic paradigm. For example, insufficient nutrition affects fetal weight and brain development. Eating too much, and gaining too much weight during pregnancy can lead to child obesity and macrosomia, or infants that are deemed overweight (Fowles 2004). Public health campaigns and messages claim that women who are overweight or obese during pregnancy are at higher risk of pregnancy complications (Cedergren 2004). Studies also claim that children born to women who are obese are at higher risk for diabetes and obesity (Catalano et al., 2009). Associating risk with BMI is a common practice and

a well-established idea in public health and medical literature (Janssen et al., 2002). Moreover, eating certain fish, cheeses, and deli meats is also associated with certain health risks for the mother and child (Athearn et al., 2004).

All of the different risks associated with eating and weight can be interpreted in a variety of ways, which results in distinct health polices between the US and UK. The US focuses on weight control while the UK focuses on glycemic control. Further, the national policy recommendations in the UK explicitly state that there is not enough evidence-based medicine to prove that monitoring weight during pregnancy improves health outcomes. In Chapter One, I argued that the differences between counting calories and counting grams of sugar and saturated fat is not just an epistemological difference, but a historically and culturally situated difference. In this chapter I expose complex relationships to food, diet, and risk that emerged for the pregnant participants in the StandUp intervention.

From the perspective of epigenetic logics, risk is transversal. The risk of epigenetic modifications occurs across time and space, in the past, present and future. The transversality of epigenetic risk is often ignored in the application and examination of epigenetics that target pregnant bodies. Barker's work on latency emphasizes that epigenetic modifications in utero may or may not be triggered later in life. The potential is there, but the manifestation is always unpredictable and indeterminate. Further, changes inherited trans-generationally can be triggered at one point or may not be triggered in one's lifetime. My theoretical framing of epigenetic risk emphasizes transversality, however, in what follows I show how the understanding of risk in prenatal interventions remains speculative and future oriented. The behaviors of individual pregnant women now are risky for future generations. The framing of risk in the trials that I observed do not acknowledge the transversal component of risk. The focus is not on how past

behaviors may or may not have already affected present and future health outcomes. Instead the trials, which use the RCT method, are focused on understanding whether nutritional interventions during pregnancy can reduce the risk of (re)producing obesity and diabetes in the future.

In what follows I examine the design and intervention delivery of the StandUp trial, and contextualize the concept of "diets" within broader understandings of weight loss programs. In addition, I explore why pregnant participants decide to enroll in the StandUp trial, and highlight how their motivations and desires are wrapped in larger discourses of responsibility and control related to their diets. Finally, I carefully review the narratives of pregnant participants to spotlight their experiences, complex motivations, fears and anxieties related to the risk and responsibility of being pregnancy.

## The StandUp Intervention

### *Intervention Design*

The National Institute of Clinical Excellence and the National Health System in the UK claim that there is not enough evidence to justify routinely weighing women during pregnancy; therefore, the StandUp trial does not give any advice or recommendations on how much weight pregnant women should gain or how much weight is healthy for women to gain. One caveat is that the trial does record weight measurements at each data collection visit, however, this information is not disclosed to the participants. Sometimes, the women want to know their weight, sometimes they do not look at the scale. If women ask for guidance on how much weight they should be gaining, the trial staff and general medical practitioners do not offer any

recommendations. Instead of focusing on weight control during pregnancy, the nutritional intervention at the StandUp trial focused on glycemic control and glycemic index.

The American Diabetes Association defines *glycemic index*, or GI, as a measure for how a food high in carbohydrates affects blood glucose levels. "Foods are ranked based on how they compare to a reference food – either glucose or white bread. A food with a high GI raises blood glucose more than a food with a medium or low GI" (American Diabetes Association 2015).[75] The national diabetes organization in the UK states that "the glycemic index is a good way of making food choices, [and] glycemic load helps to work out how different sized portions of different foods compare with each other in terms of their blood glucose raising effect" (Diabetes.co.uk. 2015).[76] The UK site explains that the glycemic index offers a list of foods that are low, medium, or high, which indicate how the specific foods will affect blood glucose levels. Foods that are high on the GI are supposed to be avoided or controlled.

The protocol manual for the StandUp randomized clinical trial, which was published for a wider audience in 2014, refers to the intervention as a "behavioral intervention designed to improve glycemic control" (2014). In a protocol draft from 2012 the stated aim of the trial was to test the effectiveness of using nutritional interventions of glycemic control to reduce the rages of gestational diabetes mellitus (GDM) and risks of macrosomia, or large for gestational age infants (StandUp protocol 2012). Overall, the intervention aims at "educating" women to make changes that can help "control" or manage their blood glucose levels.

When I interviewed one of the dieticians who helped design the StandUp intervention she emphasized that the intervention was designed to be simple and achievable for all women, regardless of ethnicity. She also noted that because they wanted to include a wide diversity of

[75] http://www.diabetes.org/food-and-fitness/food/what-can-i-eat/understanding-carbohydrates/glycemic-index-and-diabetes.html?referrer=https://www.google.com/
[76] http://www.diabetes.co.uk/diet/glycemic-load.html

women into the trial they did not add food diaries to the intervention, in case English was not someone's native tongue. The dietician was a white British woman in her mid-30s and she was notably thin. She had just had a baby and had recently returned to work from her maternity leave. She spoke highly and in defense of the intervention design. The dietician was also the only one to explicitly describe the intervention as being based on "control theory and social cognitive theory."

However, what the dietician did not explicitly state, and what is also not stated in the publications, is the general assumption that the intervention is also aimed at protecting the fetal environment. The focus on glucose control is directly related to epigenetic theories, which state that the uterine environment can affect fetal development. Only the PI of the trial discussed this specific aspect of the trial to me and in speeches to her scientific community and colleagues. For instance, in the conference I attended and commented on in Chapter Two, the PI explicitly states that the intervention draws from epigenetics and the DOHaD theories to claim that the "uterine environment can insidiously affect the risk of future disease" in children. The PI was the only one on the trial that used epigenetics to explain the risk of high blood glucose levels during pregnancy for fetal development.

As I explore further below the health trainers responsible for delivering the intervention did not use epigenetics to explain how exactly blood sugar levels could influence fetal development and early childhood development. Nevertheless, the pregnant participants as well as the trainers all assumed the same fetal-maternal relationship namely that what a pregnant woman eats during pregnancy will affect her child. Although the PI used epigenetics to justify the importance of nutritional interventions during pregnancy, the staff and participants did not explicitly refer to epigenetics, but everyone still came to the same conclusion. The pregnant

participants, staff, and PI all assumed that the pregnant women were responsible for changing eating habits and protecting fetal development. The key underlying point is that the assumption of maternal responsibility precedes the epigenetic paradigm. The intervention reflects the convergence of epigenetic theories, older assumptions of fetal-maternal responsibility, and older strategies of biopolitical control of women's bodies. The convergence of these factors produce prenatal interventions that are an iteration of contemporary biopolitics.

In the medical literature, sharp spikes in blood sugar can lead to short and long-term health consequences. For non-pregnant adults, spikes in blood sugar after eating foods high on the glycemic index can lead to fatigue and blurred vision. Long-term consequences of sugar spikes can lead to elevated A1C levels, which is a clinical measure associated with heart disease. For pregnant women, the medical literature states that spikes in blood sugar can result in large for gestational age babies (or macrosomia), gestational diabetes, or other pregnancy complications. By monitoring, controlling, and making swaps from foods high on the GI to low on the GI the women can produce a slow secretion of sugar into their blood, avoiding violent spikes and adverse health outcomes for pregnant body and fetus.

In the first lesson of the intervention, the participants receive a graph of sugar metabolism. It is a curved graph and it reflects how foods that are high on the glycemic index will release sugar quickly into the blood stream and will spike the pregnant woman's blood sugar. In the intervention delivery the health trainer would explain that these spikes in blood sugar are not healthy for "mum or babe" but she did not give a detailed explanation and from my observations, the pregnant participants never asked for further explanation. The pregnant participants also came to the intervention session with the assumption that whatever "mum" did would affect "babe."

However, like I examined in Chapter One, the fetal maternal relationship is not "uni-directional." What a woman eats and how that affects the risk of fetal development is precisely what is examined and at stake in the trial intervention. Through the logics of epigenetics, what a woman eats is risky because it directly affects epigenetic fetal programming. At the same time epigenetics implies that changes occur trans-generationally and unpredictably, therefore women do not necessarily have the control to influence epigenetic changes. Regardless, the intervention is designed to target pregnant body blood glucose levels, through diet and exercise behaviors to protect the fetal development from exposure to high blood sugar. In this way the pregnant body acts the "epigenetic vector" (Richardson 2015), to prevent future health risks of diabetes and obesity.

*Intervention Delivery*

In total, I observed twenty-eight intervention sessions at the StandUp trial. These were sessions with a health trainer and a pregnant participant. Twice I observed a session with two participants and one health trainer, and I monitored two phone intervention sessions. The sessions lasted about one hour during which the participants were given diet goals and physical activity goals to work on in between the sessions. All intervention sessions took place in exam rooms located in the Clinical Research Facility on the 4$^{th}$ floor of the Rosalind Hospital. From the intervention sessions, as I explore further below, the conversations involved intimate discussions about their bodies, health, families, work, anxieties, and motivations. The personal and intimate conversations that took place during the intervention appeared at odds with the sterile clinical laboratory setting of the exam rooms.

During my first visit to Rosalind Hospital in 2012, there were two staff members dedicated to handling the intervention delivery. When I returned in 2014, the main health trainer had moved to another hospital. The one remaining health trainer, Diana, had a background in holistic medicine and acupuncture. Diana was in her late forties and she was the first generation of her family to be born in England after her parents migrated from Jamaica. She is well educated with different certifications in health promotion. She was also only one of two Afro-Caribbean women working on the StandUp trial site at Rosalind Hospital. I did most of my intervention observations with Diana.

The protocol manual of the StandUp trial states that the intervention must be delivered between twenty weeks gestation and twenty-eight weeks gestation. In this window of time the StandUp intervention required participants to meet with health trainers for eight visits. However, in my observations it was rare for any one participant to meet eight times. In an early publication of the study the Principal Investigator and co-authors wrote that any participant who attended at least one session with the health trainer was included in the data analysis as a participating member of the intervention group. The biggest challenge to the intervention delivery was confirming and scheduling visits. Most women had more than one child, full time jobs, and it was difficult to take time off during work. However, one significant difference between the SmartStart intervention delivery and the StandUp intervention was that in the UK the National Health System recognized the StandUp trial as a form of prenatal care. The trial had to give the participants a letter to show their employers in order to justify the time off during work. In the US the participants had to squeeze in time to do the intervention before, after, or during lunch hours if they were employed.

The trial also had a separate hand book dedicated to the health trainers. The handbook provided specific guidelines and justifications for how to deliver the intervention. At the beginning of the handbook the aims of the intervention are clearly defined:

> The [StandUp] programme combines advice on nutrition and physical activity with 'behavior change techniques' to help develop habits that encourage a healthier lifestyle with the ultimate aim of improving pregnancy outcome. The aims of the programme are for participants to:
>
> - Make positive steps to improve their health and the health of their baby during pregnancy
> - Improve their blood sugar control during pregnancy
> - Learn how food and drink affects their blood sugar levels
> - Learn how to be physically active during pregnancy

The handbook was written for the health trainers and by "a team of health professionals and experts in the area of pregnancy, child health, nutrition and physical activity" (StandUp handbook). The actual team consisted of the PI, collaborating midwife social scientists, a dietician, and a group of postdoctoral students who specialize in nutrition, psychology, and behavioral interventions. The key message for the participants is that the intervention focuses on controlling sugar levels.

The handbook also explains that the health trainers should not include any other information from "other programmes" such as other dietary programs or individual understanding of food and diets. The purpose of explicitly instructing the health trainers to stick to the program of this is twofold. First, the randomized clinical trial is intended to test the effectiveness of this particular intervention on obese, ethnically diverse pregnant women. "It's therefore really important that you follow the content and structure so we know the information given to all the women is the same – otherwise we won't know what has, and hasn't, worked" (StandUp handbook). The emphasis on standardizing the content and structure of the

intervention is important for the trial's results, not necessarily for any other medical or health reason.

The other purpose of sticking to the "programme," or the StandUp intervention, is that other dietary advice or programs are not necessarily geared towards pregnant women. The handbook explains that this particular intervention may differ from other dietary advice because it is "designed especially for pregnant women with a BMI of 30 or over." The women in the clinical trial are encouraged to comply with the intervention because if they do not comply their deviant behavior might disrupt the intended effect of the intervention. If the women do not enact the "programme" as they are told then the scientists will not know whether any changes between the control and intervention are a direct result of the intervention. From this perspective, the aim of the trial is as much about enacting and implementing the randomized clinical trial method as it is about improving healthy pregnancies through dietary interventions.

In general, the clinical trial setting used the RCT method to test a nutritional intervention on a new population – pregnant women. In this way, the RCT represents a method of bodily surveillance and control during pregnancy. In addition, nutritional interventions, or dietary interventions during pregnancy imply self-discipline and control as well. The focus on control is also an existing theme in the dieting industry. Together, the nutritional interventions and the previous experience that women already have with dieting reify the focus on control and individual responsibility in the intervention delivery.

Telling women what to eat and what not to eat during pregnancy is based on the assumption that women have control over what they eat, and that they are individually responsibility for their eating behaviors, and health of the fetus. However, food studies literatures emphasizes the issues of neighborhood effects and food deserts that significantly impact a

person's ability (or control) to access affordable fresh produce (Athearn 2004; Axelson 1986). The underlying premise and assumptions of control, risk, and individual responsibility that frame the RCT and the intervention during pregnancy do not align with the epigenetic theories that are used to justify these interventions. Epigenetic logics emphasize the relationality and porous boundaries between bodies and scales of the environment. Moreover, epigenetic modifications are unpredictable and indeterminate. Even if women change their behaviors during pregnancy that may not prevent epigenetic modifications in the fetus and future child that have already been inherited from the past and that could still be potentially triggered in the future. Drawing from epigenetic science and the DOHaD Kuzawa explains that the nutritional affects on fetal development are also influenced by intergenerational inheritance (2010; Kuzawa et al., 2012). The key point is that individual pregnant bodies do not have full control over the fetal and child development. Next I examine the concept of the "diet" both at the StandUp trial and in the weight loss industry. Contextualizing the concept of diet is necessary for understanding how and why pregnant participants assume control and responsibility for their diets.

**What is a Diet? Parallels in Weight Loss Programs and Nutritional interventions during pregnancy**

One of the most common and curious statements that I heard during my observations of intervention implementation at the StandUp trial was: "This is not a diet." In my analysis of the interventions, I paid attention to practices or language related to diets, dieting, and weight loss/gain. Through this process I found that clinicians and participants used the term *diet* in either of the two dictionary definitions: 1) a food regimen (specifically an intervention) or 2) a weight loss program involving food restriction. I also coded for any relation or reference to sugar or

calories. What I found and describe below is that these themes interacted in complex ways. For instance, calories were often referenced in relation to dieting and some participants conflated counting teaspoons of sugar with counting calories. Counting calories then implied "dieting" in the weight loss sense of the word, and this association linked the intervention in participants' minds to being like a diet for weight loss. The idea that the intervention is "not a diet" and that counting or controlling sugar and counting or controlling calories are fundamentally different was a key aspect that the health trainer had to consistently remind the participants of during the sugar reduction Start Up intervention program.

The StandUp intervention appears to be a diet because it explored "the kinds of food that a person, animal, or community habitually eats" (Oxford English Dictionary 2015). Diana would sometimes use the term "diet" in her discussions. She might ask, what about your "diet" as in what are you eating, how are you eating? However, when the pregnant women would use the term *diet* in relation to restricting food or losing weight, Diana would correct them by saying "this is not a diet."

The handbook for the intervention, which guides the health trainers, states that the health trainers must "explain that [StandUp] is *not a diet and will not involve calorie counting* – and emphasize that we have suggested healthier alternatives rather than just telling people to avoid certain foods. It will also involve increasing your daily activity levels" (StandUp handbook, my emphasis). This statement implies a kind of presumption about how diets "just tell people to avoid certain foods" and emphasize denial, restriction, and calorie counting, whereas, the intervention sets itself apart by providing "healthier alternatives." From the perspective of the StandUp staff and principle investigator the intervention is "not a diet" and therefore it is a better option for pregnant women. Although literature in weight loss programs like Weight Watchers

state similar goals as the StandUp trial, it is important for the StandUp trial to distinguish itself distinction from other weight loss programs. The distinction is important because the aim of the trial is to scientifically test the effectiveness of nutritional interventions, through evidence-based medicine. Evidence-based medicine influences health policy and is highly regarded as scientific truth. The weight loss industry does not carry the scientific clout in the healthy policy world. A closer analysis of the overlaps between nutritional interventions tested in clinical trials and dietary programs sold in private industry reveals the common theme of individual responsibility and control.[77]

In ethnographic work on diet strategies in the Netherlands Anne Marie Mol argues that different diet programs or techniques represent different foods and bodies (Mol 2012). Epistemological approaches to food as fuel, nutrients, or a health risk "enact" food and bodies in entirely different ways both materially and conceptually. Mol's notion of enactment is a methodological and conceptual way of understanding how science and medicine are practiced (2005). Therefore, the enactment of food and bodies refers to the "various and varied practices" that shape bodies and food (Mol, 2012:vii). Mol's observations in a dietary clinic examined the different underlying norms that make people enact food in particular ways (2012). Drawing from Mol's analytic approach to dietary practices as kinds of epistemologies that enact different bodies, I show how the participants' dietary practices aimed at weight loss and the trial's dietary intervention aimed at glycemic control are intended to shape fetal development, and not only pregnant bodies. For instance, The StandUp trial is intending to change the physical metabolism of sugar in pregnant participants, and the participant's desire to change their bodies in terms of size and shape, are all intended to shape and protect fetal development. Further, the enactment of

diets and desires in the prenatal interventions also shape notions of risk and health for developing fetuses.

The cultural sentiments around monitoring weight during pregnancy are different in the private sector compared to the scientific community. For instance, the private company called Slimming World, a weight loss program for adults, has recently included pregnant women into their groups in the UK. In one of my observations of a visit with a research midwife and a woman who was randomized into the control group, the participant explained that she was upset that she was not in the intervention group. She said that she really wanted to work with the health trainer. She had gained so much weight in her last pregnancy that she wanted to do things differently during this pregnancy. When she found out she was not going to be in the intervention group she decided to "do her own thing" and joined Slimming World.[78] She said that they do not promote "losing weight" during pregnancy, but rather they do weekly "weigh-ins" every Saturday.[79] In terms of her diet, she said that Slimming World encourages eating fresh fruit and vegetables. She also started exercising during this pregnancy, which she had not done in her two previous pregnancies. In this case the woman's desires did not align with the aims of the trial because she was randomized into the control group, and did not receive the intervention. Nevertheless, the participant took matters into her own hands and joined Slimming world to monitor her weight and diet each week. She selected to participate and pay for a program that monitors and surveys her eating and exercise during her pregnancy.

[78] (6/4/14 H, 35wk visit)

[79] I am not sure what Slimming World recommends for pregnant women to gain each week since there are no accepted guidelines in the UK. However, it is possible that they draw from the US IOM recommendations like Weight Watchers does. On the Weight Watchers website they state that they do not work with pregnant women in the United States but that they do give a summary of the IOM guidelines that make recommendations for gestational weight gain.

Like other weight loss programs the StandUp trial promoted specific dietary changes, and although the investigators and collaborators deny any association between their intervention and other diet programs, the participants do not see a clear distinction. In another visit, a woman in the intervention group commented that she had been doing Weight Watchers before her pregnancy. Weight Watchers is the competitor program to Slimming World. A woman named Jean Nidetch from Queens, New York, started the Weight Watchers program. According to the website, in the early 1960s Jean began organizing a group of women to meet at her home to "talk about how best to lose weight […] today, that group of friends has grown to millions of women and men around the word" (Weight Watchers 2015)[1]. This "group of friends" now includes women from both the United States and the UK and eighteen other countries. In contrast to Slimming World, Weight Watchers does not allow pregnant women to join their program. The participant at StandUp said that the intervention was similar to the Weight Watchers program, except Weight Watchers focuses on "how much you're taking in through calorie counting and they monitor weight."[80] For this participant in the StandUp trial who also participated in a private weight loss program, the dietary programs were not that different. The pregnant women do not see a significant difference between calorie control and glycemic control. Both approaches to nutrition are about controlling, managing, monitoring, and surveying diet. The woman who had previous experience at Weight Watchers represents the desire to manage and control diets and weight that existed for her before getting pregnancy.

In a separate intervention observation a woman asked Diana about whether rice cakes were a healthy swap. To the woman's surprise she finds out that, in fact, rice cakes are not a "healthy" swap. Diana explains that rice cases are high on the glycemic index because they can release sugar quickly. The woman claims "Oh no! I didn't know this; I ate like ten this week!

[80] (5/12/14 H, GTT visit)

Rice cakes, really? Rice cakes are in every weight watchers diet, but I guess they aren't paying attention to the sugar […]." This was a key distinction between the StandUp intervention and weight watchers: counting grams of sugar vs. counting calories.

The people who helped design the StandUp trial defend the intervention as a form of evidence-based medicine and a test of an experimental hypothesis. The legitimacy of the intervention as a scientific experiment depends on maintaining the difference between the intervention as a lifestyle/educational program and a "diet aimed at weight loss." The intervention cannot be associated with just any kind of mainstream diet program, especially in the context of pregnancy. Despite some private weight loss programs opening doors to pregnant women, the idea that pregnant women should intentionally try to "lose weight" during pregnancy is still not an acceptable public health message in the UK.

Moreover, when I explained the SmartStart intervention from the US site to the dietician at the StandUp trial she exclaimed that a trial like that would be "unacceptable in the UK."[81] She went on further to say that people in the UK do not emphasize weight and do not recommend weight gain options or monitoring. However, controlling sugar is acceptable in the StandUp context. It is possible that in employing glycemic control during pregnancy, women may not gain as much weight because they are eating less sugar and saturated fat. Additionally, the nutritional literature often conflates both calories and glycemic control as two sides of the same coin. Studies will often use low calorie interventions to improve glycemic control among adults with diabetes (Wing et al., 1991).

The StandUp trial is following the national health policy and does not provide any weight recommendations for pregnant women and is not measuring weight as an indicator for healthy pregnancies. Therefore, the need to distinguish between glycemic diets aimed at controlling

[81] (4/9/14 HC, interview)

blood sugar and diets aimed at weight control is not necessarily a medical issue, but an issue of national policies. The key difference is that the StandUp trial emphasizes glycemic control, not weight control, despite the desires or motivations of the pregnant participants. The complexity in the message of sugar control rather than weight control is that either approach is based on the ability and responsibility for women to control their weight and diets.[82] Regardless of whether interventions focus on glycemic control or weight control, the main theme is control.

**Why do Women Participate in the StandUp Trial**?

During our first interview Diana, the main health trainer at the StandUp trial, told me that the intervention is not a diet for the purposes of weight loss. However, when I asked her why she thinks women participated in the trial she told me that the women were motivated to participate because of family history, medical problems, and weight concerns. She said, "young people don't want to put on too much weight." In Diana's own description of why the women participate she notes that women are worried about gaining too much weight. In this way the women view the program as a kind of diet that will help them manage their weight, and at the same time the intervention the protocol states that it is not a weight loss program.

The behavioral intervention aimed at changing pregnant women's diets was difficult to distinguish from a diet intended for weight loss. Diana, the health trainer at Rosalind Hospital, succinctly describes the overlap between the two notions of diets by relating the objective of the trial and the motivations of the pregnant women: "trying to reduce sugar intake is a diabetes

[82] The fundamental difference between sugar and calories is also reflected in the interview I did with LP, she talks about why GWG is not the key measure. Also, the public health literature on nutrition and diet emphasize that not all calories are equal, which means that a calorie of cheese is going to be processed different than a calorie of chocolate. A concept that supports the inequality of calories is the idea of "empty calories" (Palmer et al., 2008). Empty calories are ones that can be consumed but do not satisfy hunger or nutritional requirements. It is the metabolic process that distinguishes nutrition from calories. In the distinction between calories and sugar, some argue that glycemic control attends to the nutritional substance more than calorie control.

thing, but they realize, if I stop eating the biscuit I'll lose some weight too" (5/3/14). In this way, the intervention is both a diet promoting sugar reduction and better health and a diet that may have an effect on women's weight. On the one hand Diana is sensitive to the goals of the trial and on the other she is also sensitive to the participants' own goals and desires. For instance, she could not offer any recommended weight standards for pregnancy even if the women asked. She had to remind the participants that the main goal of the intervention was not to lose weight, but she would also say that weight loss was an added benefit to the intervention.

The trial approached women in their first two prenatal visits at the Rosalind Hospital for recruitment. The trial had access to all prenatal visits scheduled at the hospital. The database included medical history information as well as their weight and height. If they satisfied the BMI requirement, the research assistants would approach them at their prenatal appointment on the 8th floor of the hospital and ask for permission to contact them later. If the women agreed to give their contact information to the midwife researchers would call them and recruit them into the study. Out of one hundred women approached to participate in the StandUp trial only thirty women enrolled into the trial. A social scientists who was working on publishing material from the focus group phase of the StandUp trial explained to me that one of the reasons women did not participate also had to do with their concern about weight.

For instance, when interviewing women about why they decided not to participate, she found that women were resistant to engaging with more medical attention during pregnancy especially since they were overweight. The women claimed that they did not want to join a trial that told them to change their diets, they knew what they had to do to be healthy and lose weight; it was a matter of just doing it.[83] The sentiment of "just doing it" prioritizes a notion of will, choice, and control. Not wanting to be told what to eat during pregnancy reflected the increased

[83] This data was written up and prepared for publication, but then never submitted to a journal.

attention and stigma around being overweight while pregnant. The responses from the women who did not participate in the trial also illustrated feelings of shame and fat stigma for being overweight while pregnant. One comment that I remember from the report was that overweight women felt upset that no one could tell they were pregnant. These feelings around weight are sensitive and in the conclusion of the report the social scientist recommended that all physicians reflect on how they approach women who are overweight or obese during pregnancy.

To understand why thirty percent of women decided to enroll into the trial I interviewed various staff members.[84] I found three key motivations that influenced women to participate in the clinical trial. First, the pregnant participants, for the most part, liked seeing the same midwife researcher (MR) during each visit. The MRs handled all the data collection visits, during which the women could ask questions about their birth plan or discuss any concerns they were having about their pregnancy.[85] The midwife researchers were trained as midwives and all had experience working as midwives for the NHS. The title "midwife researcher" has been included in the NHS for the past two decades. This relatively new job title requires midwives to have some experience doing research in a wide variety of settings. In some of my interviews the research midwives commented that the growth in studies on pregnant women may be related with the growth in the MR profession.

A second reason for participating in the trial had to do with gestational diabetes. Some women were familiar with the different kinds of risks associated with having a high BMI during

[84] One note on methods: I did not have access to interview the pregnant participants directly, so I relied heavily on my observations of each visit interviews with staff members. I had access to interview all the staff members who were willing to meet with me. The staff members I interviewed worked at different sites of the RCT consortium (5 sites in total). However, I was only able to do ethnographic observations at one main site, Rosalind Hospital, where Dr. Elizabeth, the PI, was working.

[85] In addition, the two main MRs in charge of working with the participants at Rosalind Hospital, where I was observing, organized each visit so that they would see the same women consistently. This was important to the pregnant women because they were not typically able to see the same midwife during their routine pregnancy visits. Due to budget cuts, increase in demand, and decreased staff pregnant women often do not see a given midwife more than once thorough out their entire pregnancy. Also, allowable prenatal and postnatal visits have decreased recently.

pregnancy, although others did not know about any risks. Some women had a family member who had diabetes, which increased their awareness of gestational diabetes. Finally, the one reason that stood out to me for why women decided to participate had to do with weight. Pregnant women reported to the midwife researchers and the health trainers that they were worried about gaining too much weight. It is the last reason that I focus on in this section.

Even though the trial does not focus on weight as a key indicator for outcomes, they do weigh all the participants during the data collection visits. The bodily measurements associated with the trial instigated different responses about their bodies. Many women would get on the scale and react in a surprised or concerned manner. Some would gasps and say "oh my god!"[86] Others would ask for their weight from the previous data collection visit and the midwife would always share that information. In one case, a woman calculated that she had gained two stones (or 28lbs) between her first visit at fifteen weeks gestation and her second visits at twenty-six weeks gestation. This calculation worried her and she commented that two stones was more than she had gained during her last two pregnancies. This woman asked the midwife "[I]s this okay? Should I be gaining this much? You would tell me if I am gaining too much right?" The midwife responded to the concerned participant like she responded to many others who were worried about their weight gain. She said: "There are no recommendations for how much weight you should gain. The baby is growing, you are alright." The response soothes the woman for the time being, but the concern over weight gain persists for women throughout their pregnancy no matter the national health recommendations.

During my observations it was clear that some women, not necessarily all, wanted to know about their weight. I interviewed two women that reflected perspectives as staff members and participants. Sheryl and Candice were staff members on the StandUp trial, they enrolled in

[86] (5/12/14 H, GTT visit)

the trial as participants, and were randomized into the intervention group. Candice was a lead research midwife at the beginning of the StandUp trial, and became pregnant during the course of the trial. Candice recruited the first participant, worked on articles for publications about the trial, and trained other staff members. Sheryl joined StandUp trial first as a pregnant participant, and after she finished the intervention she decided to work on the trial as a health interventionist.

Candice's had significant experience with dieting and losing weight. In an interview with Candice she told me about her struggles and victories with weight loss before getting pregnant. She even showed me a "before and after" picture, which is documented on a national UK television program. She was selected to participate in a weight loss program on TV that applied hypnotherapy to subjects. She lost over 100 kilos in the process.[87] Candice stated that losing weight was not a key motivation for her to join. Instead, Candice said she joined because of her it was her family's medical history with diabetes and heart disease that made her feel like she was a high-risk pregnancy. She also commented that she wanted to "give back" and help future women by volunteering in science. However, Candice made it clear that her past experiences with different weight loss methods influenced her perspective and experience in the intervention. She said that she already knew most of the nutritional material covered by the intervention.

In an interview I did with Sheryl[88], she told me that she decided to join the program like most women do, in order to watch her weight. Sheryl has three small kids and is trained as a nurse, but decided to work as a health trainer on the study.[89] She was recruited into StandUp during her last pregnancy and after her delivery she decided to apply for a job on the trial as a health trainer. Sheryl lives and works at one of the northern most sites in the UK trial. Her

[87] Other staff members on the trial would talk about Candice as an exemplary weight loss story. See interview with IVF advocate, OBGYN doctor and with LP/AN
[88] SH
[89] Health trainers are a lower "band" or level than nurses, so they make much less than nurses, it is significant that she wanted to work as health trainer because she liked doing it, not necessarily for the money.

explanation for why she decided to participate in the trial reveals how she perceived the motivations of most trial participants:

> Probably for like most people, [in the trial], not to gain too much weight. […] I made the mistake during my first pregnancy to 'eat for two' and I gained 2 stones[90] and it was difficult to loose the weight because of returning to work, and balancing the kids, and it's not easy when you have a wee one at home. […] 80% of the girls don't want to gain too much weight during their pregnancy, some will be conscious about GDM if they have a family member with it, […] but 9 times out of 10 its usually just about the weight gain, -- not to repeat the same mistakes.(Interview w SH 4/22/14)

Sheryl echoes the same concern and anxiety about weight that many of the pregnant participants had. The concern with gaining too much weight during pregnancy is a real concern that physically and emotionally preoccupies women's minds before, during, and after pregnancy. An awareness of weight among staff and pregnant women reflects an existing issue with weight outside of the context of the trial. Regardless of how the trial frames the intervention, weight plays a significant role in its implementation. For Sheryl the focus is on weight, but the broader aim of the trial is to reduce gestational diabetes during pregnancy.[91]

The key point to highlight in the motivations of the women who enrolled in the trial is the desire to monitor and control weight and diet. One main difference between the thirty percent of women that decided to participate and the seventy percent that did not, has to do with the desire to have their diets managed, surveyed and monitored. That is the key difference is that thirty percent of women wanted to subject themselves to the surveillance and the other seventy percent did not necessarily want to be told how to eat.

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[90] 1 stone = 14 lbs.

[91] Not only are the pregnant participants aware of their weight but the health trainers like Sheryl, Candice, and other staff members are also conscious of their own weight. During my first visit to this same site I spoke to another health trainer and to fellow staff members and they all talked about how working on the StandUp trial made them more of aware of their own weight and diets. The staff members had internalized this concern with weight even though they were trained to tell the participants that the intervention was not a diet for weight loss.

In Thompson's work *Making Parents: The Ontological Choreography of Reproductive Technologies,* she highlights how women willingly subject themselves to assisted reproductive technologies (2005). In doing so, Thompson is exposing a complex relationship between technology, objectification, subjectivity, and agency in the context of infertility and assisted reproductive technologies. Foucault used the term subjectification to explain the ways in which individuals participated in biopolitical regimes of bodily discipline, and in turn how biopolitical strategies produced new subjects in the process (1975). From a feminist perspective Thompson focuses on how processes of engaging with ARTs can influence the subjectivity of women who submit to objectifying medical processes in order to meet their fertility aims. In this way women are both objectified and enact a kind of reproductive agency.

In a different ethnographic example, Bridges shows how poor women of color have to submit to a bureaucratic system that surveys and monitors their behaviors and lives in order to receive healthcare during pregnancy. In Bridges assessment of race, reproduction and surveillance in Medicare clinics, she found that women have to submit themselves to being classified as "nutritionally at risk" in order to receive WIC benefits for food stamps and subsidies for baby formula (2011:55). However, the cost of submitting to the label of "nutritional risk" comes with other social stigmas and heighted surveillance. Bridges analysis emphasizes how the submission to classificatory regimes produces "unruly" bodies that are seen perpetually seen as at risk (2011).

Similarly, the participation in clinical trials that monitor, survey, and manage diets during pregnancy include forms of objectification; however women submit themselves to this process and reflect some agency in the process. The complexity in the process of enrolling is that the motivation is based around weight control. The submission to disciplinary strategies in both the

SmartStart and StandUp trial comes out of an individual desire to be disciplined and surveyed. From one perspective the thirty percent of women who did participate self-selected into the trial because they wanted to be intervened upon. This motivation and desire to participate reflects a larger systematic and cultural focus on individual responsibility, choice, and control around eating and weight before, during and after pregnancy. In the next section I explore in detail the ways in which pregnant persons participate in dietary interventions.

**Pregnant Narratives: risk, responsibility, and control**

The examples below illustrate the complex relationships that women have with food, diets, and their weight prior to and during their pregnancy. Examining the pregnant narratives closely is important for understanding the perspective of pregnant persons who are targeted for nutritional interventions. There is a lack of information that reflects the perspective of pregnant participants in ongoing clinical trials (in part because the inclusion of pregnant women is new after decades of exclusion), particularly trials that draw from epigenetics to justify prenatal interventions. My emphasis on the perspective of the pregnant participants is framed by the intersection of feminist science and technology studies and medical anthropology. In feminist STS, the focus is on understanding methods, tools, and theories of scientific knowledge production to examine the taken for granted aspects of race, gender, power (Haraway 1991; Traweek 1988). Medical anthropological approaches prioritize the narratives of patients in medical encounters (Kleinman 1988; Martin 1987; Rapp 1987). The following is a combination of both approaches. Exploring the following narratives elucidates the ways in which individual women take on responsibility for their diets in effort to control their weight, and reduce any health risks to their developing fetus.

*Shaina: not a diet but education*

During a first visit with Shaina, a trial participant, Diana asks her to explain her regular food habits. Shaina is in her late-20s and identifies as Afro-Caribbean. Much of her family still lives in Jamaica while Shaina is studying accounting at a university in the UK. She is also parenting a three year old as a single mother. Shaina describes her diet through her daily activities, such as drinking "fizzy drinks" or soda, eating too many chips, and "nibbling fruit at Uni [university]."[92] Diana responds by stating the benefits Shaina might receive from the intervention, including physical fitness for labor and a healthy pregnancy. Diana also adds that the intervention can help manage her weight not by dieting, but focusing instead on maintaining healthy sugar levels." Diana's last statements may sound contradictory, but in the setting of the trial both not gaining weight and not dieting can co-exist. Diana avoids mentioning weight loss by redirecting the conversation to managing sugar levels with the added benefit of "managing" weight, or assisting woman to not gain "too much." Like the handbook stated, Diana needs to make sure the pregnant participants understand that the intervention is not a diet. She says that the intervention focuses on "maintaining sugar levels" as a way to ensure a health pregnancy and womb environment for the fetus. The weight management is an "added" or secondary benefit, not the primary goal of the intervention. Diana makes this clear throughout the intervention sessions.

In the same meeting with Shaina, Diana goes over the amount of sugar that is in a regular soda and again she reminds Shaina that the intervention is "not a diet."

D: 1 coke = 7 tsp of sugar! Visually it's quite a lot.
D: Once you become aware you can make swaps.

[92] " Diana and 1A, Shaina 5/3/14

Shaina: Is that the same as calories?
D: Well, we aren't counting calories, this isn't a diet.
Shaina: Well, after baby I'm planning on going on a diet.
D: Rather than diets this is making educated choices that can help long term.

The participant's question about whether seven teaspoons of sugar are the same as calories reflects a gap between her understanding and the scientific reality that the trial is presenting. The participant does not have a history or prior experience with counting calories or evaluating foods based on grams or teaspoons of sugar. During another part of the same meeting, Shaina reacts with surprise when she learns that white sugar and brown sugar are the same, or that one does not have more or less sugar content. In another instance, Shaina was surprised to learn that the first ingredient on a label represents the ingredient that is used in larger proportion than the rest of the ingredients. Through these realizations, Shaina is being taught a particular way of reading and examining food labels. She is learning how to focus on controlling sugar or becoming aware of sugar content for the purposes of monitoring her blood glucose levels.

Since Shaina is interacting with this approach to food through glycemic control for the first time, her experience in processing and incorporating the intervention information is different than for someone who has more experience with "counting" or reading labels. This is echoed in remarks of some of the other participants. Diana indicated that on average the women of "African descent" do not have as much experience with dieting or counting calories as the white British women do. As a result she said that the Afro-Caribbean or African women were more open to the intervention and less resistant because the information was new to them.

In response to whether counting teaspoons of sugar is the same as counting calories Diana reminds the participant that the intervention is not a diet, but a way to make "educated choices" in the long term. This distinction may not resonate with the participant since she is still

planning on "going on a diet" after the baby is born. From this particular session with Shaina, it is not clear whether she views this program as an educational intervention in the way Diana believes it to be or a kind of diet aimed at weight management. Whether Shaina desires to go on a diet to lose weight for cosmetic reasons and also reduce sugar for health reasons, the distinction between the two in the intervention does not matter as much because by participating and enacting the intervention she is also satisfying her own desires to control her weight and diet.

Although Shaina doesn't explicitly state why she would go on a diet after pregnancy, it is implied that for the participant, counting calories or monitoring sugar shape bodies in similar ways. By participating in the trial the participant enacts both her idea of a "fit" body that is thinner and can metabolize sugar in a "healthy" manner. For Shaina the relationship between calories, sugar, and weight loss is one of similarity. However, for the aims of the trial, which Diana is trying to implement, there is a clear difference between counting calories versus being aware or "educated" about the amount of sugar and saturated fat in food. To maintain the legitimacy of the trial, the distinction is important because the trial is science not a fad diet. The distinction that is labored on behalf of the staff seems to fade in importance for the participant. for the participant counting sugar or counting calories are two sides of the same coin, which is still based on controlling diets.

*Donna: liquid food diets, IVF, risk, and responsibility*

In other intervention sessions participants are explicit about their motivations to lose weight prior to getting pregnant and their experiences with other forms of dieting programs. In a first visit a pregnant participant shared her experiences with other weight loss programs and

related these to her desire to be "healthy" and lose weight for her pregnancy. Donna[93] is a white British woman in her mid to late 30s. She is a school teacher and has experienced five miscarriages before this pregnancy. She has a rare genetic disorder that results in aborting all male sexed fetuses. Prior to this pregnancy she was told by her doctor that if she wanted to try in-vitro fertilization (IVF) she would need to lose weight. Donna explains how she lost the weight prior to getting pregnant.

> Donna: […] last year I lost 2.5 stones (around 37 pounds)
> D: what did you do?
> Donna: I did something controversial- I did liquid beverages, liquid life-replaced all food with liquid it was hard at first but I had to do it I was pushing 20 stones (280 pounds).
> Donna: [my] BMI was too high for IVF, so I had to get my weight down to conceive, to have a baby

In this exchange Donna reflects on her motivations to do a "controversial" form of dieting. Interestingly, she uses the term *controversial* to describe her perception of what is appropriate or not in the UK setting. This controversial method in the UK is similar to the intervention that is tested in the United States (Smart Start). Similar to Thompson's work on IVF and assisted reproductive technologies, Donna submits herself to the process of weight control and surveillance in an effort to reach her desire for conception. It is clear that Donna is familiar with dieting, and in the next exchange Diana asks Donna about her diet as in what she normally eats.

> D: so how is [your diet] now?
> Donna: I get up, have cereal milk, snack on some melon grape roll, and a fizzy drink.
> [Donna whispers when she says fizzy drink, as if it is a bad secret]
> Donna: I don't like to eat sweet stuff although I am heavier. I don't eat sweets, I like savory, my husband and I, we write down our foods, menu plan--I tend to

[93] 12A DB, teacher

worry when I get hungry I get worried because I think I'm not just eating for myself. I don't want to get lightheaded […].
Donna: to be totally honest I need a sugar fix [she giggles].
D: no judgment--So per 100ml of fizzy =2 teaspoons. So those beverages will have around seven teaspoons of sugar. What you experience you baby also experiences, [so you and your baby] will have a spike in sugar […].
Donna: I think with me this is my 5th pregnancy I really want this, I don't want to do anything that would risk it.

In describing her own diet Donna tries to explain that even though she is "heavier" she does not necessarily eat tons of sweet foods.[94] Donna also notes that she and her husband have tried different dieting strategies like writing their food and menu down together. Her relationship with her weight and dieting has a longer history beyond this first intervention session. Towards the end of the exchange it is revealed that Donna had to lose weight, in order to be eligible for IVF treatment. Her doctors told her to manage her weight in order to ensure the viability of her pregnancy and ward off any potential risks that her eating habits might incur. Donna's epistemological approach to food and dieting is with her body's ability to become pregnant.

For Donna there is a lot at stake in maintaining a healthy diet for her and her baby. Both Diana and Donna draw explicit connections between Donna's diet and the baby's development. As Diana notes, "what you experience the baby also experiences." In Diana's framing, there is no separation of the maternal/fetal subjectivity; the fetus is described as being able to "experience" spikes in sugar metabolism.[95] Diana approaches the fetal/maternal relationship from a glycemic control perspective and Donna references the fetal/maternal relationship in relation to risk and responsibility. Donna does not want to risk her pregnancy and would do anything to try and ensure the safety of her fetus. The complexities around weight, diets, sugar

[94] This is a judgment she receives that is related to the fat stigma around seeing heavier bodies and making assumptions about their life, habits, tastes, and lack of will power.
[95] It is unclear whether the use of the term "experience" refers to a subjective experience or physiological experience for the fetus.

control, and pregnancy are magnified in Donna's experience. Both Donna and Diane come to similar conclusions about the effect that eating and food have on the risk and health of the fetus. In the clinical trial setting and in Donna's personal and familial life, controlling her weight and diet is directly related to the health and risk of the developing fetus.

*Ashley: chocolate, stress, and baby*

For other participants the intervention interacts with existing approaches to diet, food, and behavior. For instance, in Ashley's first visit with Diana, Ashley commented on how she does not "eat badly."[96] Ashley knows what foods are "healthy" and which foods are "bad." So she felt like she didn't have much to work on during the intervention sessions. She continued explaining at her first visit with Diana that "I have a problem with chocolate and crisps, which I'm trying to compensate [for] with more exercise, but my biggest challenge is that I work from home." Ashley is a computer programmer, and she lives with one other flat mate, and primarily works from home. Ashley is a woman in her mid-thirties, identifies as white British, and she was pregnant for the first time. In the first visit Diana listened and tried to assess Ashley's diet or regular eating habits. It is from Ashley's own experiences with other programs that she identifies chocolate and crisps as "bad" foods, and it is also from other experiences of "avoiding" certain foods in other diets that she reflects on how these "bad" foods are problems for her.

During Ashley's second visit she shares her physical activity goals and comments on how it was hard to walk after forty minutes. They also went over her goals of not eating out and limiting dessert, which again seems similar to dieting goals. Ashley explains how she ate since the last visit with Diana.

[96] 5/16/14, 11A 16

> Ashley: I had chocolate today I have to be honest. My flat mate is a bastard. She brings in rubbish and I eat it.
> D: What can you swap for these things? Let's look at lesson 2
> Ashley: I drink water, I don't do rice I don't do white bread, I do noodles, (whole wheat egg) I do couscous, brown pasta, Breakfast I do porridge […]. To be honest it is just chocolate, just the odd chocolate. I have fruit and yogurt. I'm not into biscuits, cakes, or dried fruit. I am getting into frozen yogurt
> D: So for this week, on average how many (chocolate bars) will you have per week?
> Ashley: Three times per week. I had one bad day where I progressively had the whole package of chocolate. It was so good. The baby kicked the whole time!
> D: oh because of the caffeine, obviously baby kicking, here are the measurements of caffeine, If you buy it you'll eat it, […] The reality is that you are home a lot--it's good to stop you from buying it and bringing it in the house. Talk to your flat mate.

During this second session, Ashley seemed to talk more about what she does and does not eat, which did not allow Diana to get through the lesson. Ashley's past experiences with restricting or avoiding certain foods is apparent in her list of what foods she "does." Her moments of "honesty" reflect an internalization of "good and bad" behaviors or "good and bad" choices related to certain foods. Discerning between foods that are "good/bad" is a practice that Ashley was familiar with before enrolling in the intervention.

Ashley's moralization of good and bad behaviors or foods existed before she started participating in the trial, which reflects her experience with dieting in the active and restrictive sense of the word. Her moralization of food is related to literature (Greenhalgh 2012, Carney and Greenhalgh 2014, and Chavez forthcoming) that discusses a neoliberal regime that frames peoples eating decisions as "good or bad." Mol (2012) also identifies this kind of framing in her words as the "control vs. pleasure paradox." The moralization of foods as good and bad motivate and frame Ashley's participation and enactment of the trial's intervention.

In considering both "control vs. pleasure paradox" and the "good/bad" framing of diets and food, I find that being in control is associated with being good and that indulging in the

pleasure of eating is bad. For instance, Ashley's "problem with chocolate" is an issue of controlling how much chocolate she eats. Ashley claims that she had a "bad" day when she ate an excessive amount of chocolate. At a different visit Diana asks Ashley "how is the chocolate?" Ashley replies, "I hadn't had chocolate all week, until yesterday, and for me that's a bloody miracle![…] I still feel like allowing myself that one blowout per week on chocolate, but this is not a diet it is a lifestyle."

Ashley reflects her understanding of the intervention's aim of making lifestyle changes rather than a program that restricts foods. Although, she did not eat chocolate most of the week, which counted as a "miracle" for her, she still feels a desire to "blowout" or consume lots of chocolate in one sitting. Her desire, motivation, and feelings about chocolate are not necessarily addressed in the intervention. Instead, Diana can only make suggestions about "healthy swaps" to replace chocolate. Diana emphasizes different alternatives and also incorporating small snacks throughout the day so that Ashley is not tempted to overeat in one sitting. The tension around chocolate as "bad" but also "so good" reflects the contradictions within the substance of food and the relationships the women have with food.

The other issue underlying the pleasure and control of eating and being "good or bad" relates to fetal risk. In the context of pregnancy Ashley's relationship to food and her desires can be risky for fetal development. In reference to the previous conversation about eating "loads of chocolate" in one sitting, Ashley comments that the baby was kicking in her stomach while she was eating the chocolate. Diana makes the connection that the baby was kicking because Ashley was eating chocolate, which has sugar and caffeine. The exchange also brings an explicit connection between Ashley's eating chocolate and the "baby's" response. Similar to the previous invocations of the maternal/fetal relationship, Ashley's behaviors are directly associated with her

fetus's well-being. Ashley made the connection in an off-hand manner, but it was Diana who brought attention to the immediate effects that Ashley's food choices and desires could have on the health of her baby. In the context of this trial Ashley's "chocolate problem" is related to the potential risks of gestational diabetes mellitus (GDM) and high BMI. Through epigenetic logics, the risk of eating too much chocolate can impact fetal development and future generations. Therefore, Ashley's desire and pleasure from eating chocolate is associated with risking the health of the developing fetus and future child.

*Ashley: The Weight of Labor*

Ashley's was 26 weeks and 5 days pregnant during her last visit with Diana. Her glucose tolerance test (GTT) for the StandUp trial was scheduled in the next two weeks. In the last session her relationship with chocolate emerged again and this time there were more connections made with how she was feeling.

> D: So how are things going?
> Ashley: I can't sleep the heat will kill me, but I'm still swimming […]I focus on swimming more and walking on another day and yoga, I wasn't so good with diet last week, bad day, cake, biscuits, and chocolate. I was emotionally down so I had comfort food. I was cramping couldn't feel baby and I was getting so worried. […] I know it's about pulling myself out of this funk, but my partner is out of the country.

In response to this Diana focuses on encouraging Ashley by reminding her of how well she's done in the intervention and that it is okay to have an "off week." By doing well, Diana is commenting on how Ashley is enacting her body and food choices in line with the intervention's aims. Ashley may be experiencing a wide range of physical and emotional changes that affect her mood, energy levels, and eating behaviors. However, the focus is on how all of these different and complex variables caused her to have a "bad" day and eat "bad foods." The fact

that she could not "feel baby" instigated anxiety and she did not have support. She is aware that this emotional-physiological complex motivated her eating; however, her awareness also comes with a moralization of her behaviors.[97] Diana responds in a kind and empathetic manner, which is not technically described in the health trainer manual. In the end, all of this information is distilled into one goal for Ashley.

> D: Now you have awareness, in term of goals [of] what would you like to work on in the future?
> Ashley: My key goal is not to gain anymore weight, keep exercise going, and keep managing my work load

After Ashley makes the connection between her pregnancy and feelings and her eating, she concludes that her main goal is to "not gain anymore weight." What emerges as a dominant concern from these complex feelings is her weight. Her body, her weight, her behaviors, and the food she eats all converge to represent the future risk and health of her developing fetus. The weight of laboring through these feelings, anxieties, and responsibilities during pregnancy is not accounted for in the epigenetic framing of the inter-generational risk of chronic disease. Whether the language is framed around glycemic control or weight control, the same target comes into focus – pregnant bodies and behaviors are responsible for future risk. And in turn women subject themselves to dietary control and take on the individual responsibility that is mistakenly aimed at their bodies.

## Conclusion

*The Pregnant Paradox: Individual Responsibility, Risk and Control*

[97] In this exchange there is a different fetal/maternal relationship reflected. In previous visits Ashley noted how her eating stimulated a fetal response of "kicking." In this instance the lack or absence of fetal movement stimulated a maternal response. Ashley was worried and feeling emotional, which affected her eating behavior. What emerges from this interaction is that the fetal/maternal relationship is not necessarily uni-directional. It is possible that the behavior or movements of a fetus can also affect a woman's feeling and behavior.

In this chapter, I examine a paradox inherent in the entanglement of individual responsibility, risk, and control. Following the current epigenetic model that focuses entirely on the maternal body as a key site of intervention, the nutritional interventions that I examine target individual eating behaviors and diets of pregnant women. Through the examination of the pregnant narratives I show how both scientists who design the interventions, and the consenting pregnant participants implement the assumption of individual responsibility, risk, and control. By willingly submitting to the idea that a pregnant person is in control and responsible for her dietary choices, participants engage in a troubling paradox.

The issue of individual responsibility is further complicated by the fact that pregnant women have never fit well into the classical liberal notion of individuality. Haraway states that the female body is too 'ambiguously individual' because woman's bodies can make other bodies (1993). She argues that this "special ambiguity of female individuality [explains] why women have had so much trouble counting as individuals in modern western discourse"(1993:405). The existing ambiguity of female individuality also facilitates the justification of interventions on women's bodies. Further, the ambiguous individuality supports the statement that women are not individually responsible or in control of epigenetic changes for two reasons. First, as Haraway states female bodies are not exactly individual in the Lockean or Humean sense.[98] Also, the notion of individuality is a social construct based in classical social theory that prioritizes labor, property and citizenship (Cohen 2009). Women's bodies were considered property of fathers, and husbands until the twentieth century (Beauvoire 1952; Davis 1999); and Black women have an unequal history of being framed as property and denied rights and ownership of their own social and biological labor (Bush 1993; Roberts 1997). Further, the individual rights of female

[98] I use the term "female bodies" but I mean bodies that are deemed "female" by the medical/social assemblage.

bodies are socially treated as if they are at odds with a historically recent notion of "fetal rights" (Berlant 1997; Morgan and Michaels 1999; Solinger 2000). The point I want to highlight is that regardless of the epigenetic context, female bodies have always struggled to receive the social benefits that come with individual rights bearing (white-male) bodies. Scholars have also argued that the notion of personhood founded upon individuality does not map onto the relational connections that are produced through everyday exchanges and experiences across humans and nonhumans, which is referred to as relational autonomy (Mackenzie and Stoljar 2000; MacPherson 1962; Prainsack forthcoming). Paradoxically, female bodies still bear the labor of individual responsibility. [99] Epigenetic studies that target female bodies during pregnancy are just another instantiation of the paradox of "female" individuality and responsibility. Although many scholars have challenged the possibility and stability of individual personhood, in my research population health still gets individualized to the diets of women. [100]

Regardless of the ambiguous social and biological individuality that pregnant persons temporarily assume, they are still responsible for bearing all of the risk that comes with protecting the viability and health of a growing fetus and future child – as if they were in complete control of their context and living conditions. The paradox I highlight is that if we take epigenetic theories seriously, pregnant persons are environments entangled with other scales of the environment. The inter-dependent and inter-connectedness implicit to epigenetics challenges classical liberal notions of individuality, and hence the responsibility and risk that comes with it.

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[99] See Woollard, Fiona (2015) I, me, mine: body-ownership and the generation problem. Pacific Philosophical Quarterly, 1-33. Also work by Elselijn Kingma.

[100] Actually, biological individuality is a fiction propagated by the marriage between western science and social theory (Cohen 2009). No body is biological individual. Bodies are populated with, and co-exist with diverse bacteria that affect our mood, digestion, neurochemical pathways, and immunity, which is recently examined through the concept of the microbiome (Boddy 2015).

In the first half of the chapter I examined the design and conceptual framing of the StandUp intervention, which prioritizes glycemic control. Glycemic control is aimed at reducing instances of GDM for the pregnant participants, and reducing the exposure to blood sugar spikes for the developing fetus. I also explored the motivations that drive pregnant participants to enroll into the trial. Juxtaposing the trial aims and the desires of the pregnant participants reflect the intersection of divergent desires toward a common goal. Although the trial claims it is not a diet intended for weight loss, and despite the lack of formal gestational weight gain recommendations in the UK, the participants are interested in managing their weight gain during pregnancy. Therefore, a significant proportion of women enrolled in the trial desire surveillance and guidance in controlling their weight and diet.

The desire to control weight and diets reflects a deeper paradox within the pregnant narratives that I described in the second part of the chapter. Women who enroll have had a longer history of dealing with weight and diets prior to pregnancy. The common narratives involved in body image and diets are also based in individual responsibility and control. Blaming non-pregnant adults for being overweight as a result of their lack of will or control is an existing social phenomenon (Sutin and Terracciano 2013). The same holds true for pregnant persons, but the blame is compounded by the fact that their lack of personal will and dietary control is deemed risky for the developing fetus. Consequently, the nutritional intervention aimed at glycemic control represents a heighted site of individual responsibility, risk, and control.

The narratives of the pregnant participants are a snapshot of the mental and emotional labor that is required to manage the risk and responsibility involved with eating, exercising, grocery shopping, reading labels, counting grams of sugar, feeding their kids, working, and making time for extra prenatal visits. However, as Annie's story illustrates, the labor involved in

taking full responsibility of fetal health outcomes is at odds with the intense feeling and understanding that she is not in control. Annie cannot consciously control how her body processes food, sugar, and weight, or the changing standards of "healthy" weight gain recommendations, or the wide variety and scales of environmental exposures. Furthermore, epigenetic theories explicitly claim that Annie's individual choices and behaviors cannot influence the unpredictable and indeterminate epigenetic changes that manifested in her grandparents bodies, or could manifest in her body, or in her children's bodies (Richardson 2015). The great schism between control and responsibility is the bases for the paradox that I have developed throughout this chapter.[101]

The implications of the pregnant paradox are evident through the recent prioritization of "fetal rights" and the adversarial conception of personhood in the criminalization of pregnant persons. For instance, during the "crack epidemic" and the war on drugs of the 1980s, South Carolina criminalized (mostly black) pregnant women who tested positive for smoking crack (Roberts 1997). The criminalization of pregnant drug users was based on two key points, the illegal use of narcotics, and risking the health of the fetus. The paradox in this situation is that at once drug addiction is understood as an illness that requires medical care, and individuals are found at fault for their illness and criminalized, as if they had full control and responsibility. The gap between the juridical implications of addiction and medical classifications of addiction are at odds (Garcia 2010). The same contradiction and tension applies to pregnant women, except the judgment is compounded by the legal and social recognition of fetal personhood and rights. As a result of the legal precedent set by the crack cocaine example, laws have expanded and further developed around the idea that fetuses have rights.

[101] The mental, physical, and emotional dissonance between control, responsibility, vulnerability, and helplessness might be a process that is co-constitutive of prenatal and postnatal depression.

Although the supreme court decision on Roe v. Wade (1973), concluded that fetuses are not persons included in the 14th amendment, viable fetuses can still be protected at the state level (Linder 2005). Currently, over twenty states across the US have legal policies that allow the imprisonment of pregnant women who are caught drinking or smoking during pregnancy (Linder 2005). The implications of fetal rights and personhood are further instantiated in an editorial statement made by the *Lancet* journal. From an evaluation of different studies on the risks of home births, the editors of the *Lancet* found that home births reduce co-morbidities for pregnant persons, but may increase the risks of neonatal complications for the newborns (2010). They stated that "women have the right to choose how and where to give birth, but they do not have the right to put their baby at risk" (The Lancet 2010:303). In this example the individual rights of pregnant persons are also undermined by the expectation that pregnant persons are required to undermine their own health in an effort to secure the health of their unborn children. The various examples that undermine pregnant persons rights and individuality begs the question, why should we continue to support the fiction of individual autonomy, responsibility and control during pregnancy?

My approach emphasizes the potential in epigenetics science to promote relational ontologies specifically to processes of gestation. The commitment to assume the temporary space of pregnancy is a relational decision dependent on complex biological, social, economic, and political entanglements. At the same time I understand that it is a very personal and intimate choice that women take on and identify with in complicated ways. However, what exactly do pregnant persons gain in assuming individuality? If being an individual pregnant person comes hand in hand with increased surveillance, contradictory messages of control, and individualized

responsibility, what is the benefit of believing in individuality during pregnancy?[102] What would child/maternal health look like if we took seriously the relationality of epigenetic logics and disentangled pregnancy from the unequal, gendered, and oppressive ideals of individual responsibility?

[102] This question applies only to persons who are willingly pregnant and does not apply to individuals who seek to terminate pregnancy. The undermining of individual rights have different consequences for birth control and safe abortions and therefore the fight for women's rights in accessing reproductive healthcare is an area that I can not address within the scope of the ethnography. The conclusion I illustrate a thought experiment that specifically focuses on situations in which people are willingly committed to becoming pregnant.

## Conclusion:

## Being and Knowing Otherwise

In the summer of 2015 the principle investigators and collaborators published the results of the StandUp intervention. They found that the intervention was not clinically significant. However, there were some health outcomes that varied slightly between the control and intervention groups. For instance, the intervention group had lower gestational weight gain, maternal fat mass, and glycemic load compared to the control group. Each of these factors are associated with high adiposity in the offspring, or fat babies. The authors claim that although there were no significant changes among the pregnant mothers, the children who they continue to follow up with may have a reduced risk of obesity and diabetes through epigenetic pathways (2015).[103] In addition, the article stated that more RCTs are necessary to understand the results from the StandUp trial, and that obesity prevention, in the form of nutritional intervention, should target women of reproductive ages.

Soon after the results were published, I connected with Rita, a key collaborator at the SmartStart trial in the US. Rita and other collaborators on the SmartStart trial were very interested in learning about the results from the StandUp trial. Since the SmartStart trial had initially a much harder time recruiting, their intervention had not yet completed. (As I write this, the SmartStart trial is transitioning into the final phase of data analysis). Knowing I had spent time observing the UK trial, Rita asked me why I thought the StandUp intervention had not been successful. I told her that I thought the variation in gestational diabetes diagnosing, screening, and treatment probably influenced the trial results, which was aligned with what the publication

[103] For confidentiality reasons I am not citing the publication, and only provide the date.

concluded.[104] I also commented on how the findings did not discuss or capture the complex relational factors that emerged in the intervention delivery. In addition, I thought that the women in the intervention group experienced the intervention differently. Some women participated through one phone call while others participated in eight in-person sessions. Regardless of the statistical power to control for the difference in intervention participation and delivery, variations could still emerge if the same intervention was delivered outside the clinical trial setting.

Before the results came out from the StandUp trial, one of the collaborators had proposed the StandUp intervention for a large NHS grant. The collaborator won a few million pounds to implement the same intervention from the StandUp trial into standard prenatal care in a poor Afro-Caribbean community in South London. Although the intervention was not successful in achieving the primary outcomes, other health agencies within the National Health System believed the intervention was still valuable. In the end, it did not actually matter that the StandUp intervention was not technically successful in addressing the intended health outcomes. For the NHS, the StandUp intervention prevailed as a cost-effective and valuable form of nutritional education and surveillance.[105]

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104 The main reason that the authors state for the lack of significant difference between the intervention in control groups is that the fact that they screened for GDM at 28 weeks and they and were given care or intervention for GDM. So that if any woman in either the control or intervention group had been diagnosed with GDM, then she was sent to the diabetes clinic for special care. In addition, the trial used the HAPO regulations for diagnosing GDM, which were more conservative and diagnosed more women than the current diagnostic criteria used by the NHS hospitals. So the trial was diagnosing more women with GDM and treating them in both the intervention and control groups.

105 The story is more complex than I have space to explain here. When I spoke to the collaborator who won the grant, he told me that he wanted to give something back to the community who had contributed to the StandUp trial. He genuinely felt that the intervention could provide some assistance to the poor marginalized community. His priority was to funnel some of the research resources back into the community – resources that the thought would not necessarily be distributed to the community had he not gone through research funding channels. His approach is entangled with the fact that he is the only African physician and professor working within the Women's health department. He prioritizes his research efforts on understanding the health disparities that unequally affect poor African and Afro-Caribbean communities. Currently African British women have the highest rates of premature birth, infant mortality, and maternal mortality, in all of the UK. The complexity emerges when his well-intended efforts to redistribute research funds back into the community come hand in hand with increased surveillance and

During one of my phone meetings with Rita, I voiced my concern regarding the interpretation and implementation of epigenetics in prenatal intervention. One of the main findings that the StandUp trial stated was that obesity prevention efforts should target women of reproductive ages, which includes nutritional interventions *before* pregnancy. Then Rita asked me: "Do you think this is a good thing or a bad thing?" I responded by stating that I do not think there is a clear "good/bad" scenario, but that the ways in which epigenetic theories and DOHaD are interpreted and applied help justify the increased surveillance and regulation of pregnant bodies and behaviors. Rita further responded:

> Right, but Dr. Sally and I agree that we want women to change their behaviors, so that they can optimize their health. This isn't the 1880's where women are trying to eat whatever they can find. In this age, women are in environments that pressure them to eat bad things, and we are telling them to resist that.

Although women are in environments that expose them to "bad" foods, one of the key arguments of this ethnography brings attention to the fact that women's bodies are studied as if they are environments. A holistic interpretation of epigenetics could shift research towards structural interventions, and away from focusing solely on the individual. In my response to Rita, I mentioned that the emergence of population health established elaborate biopolitical strategies to target women's behaviors as the primary caretakers of children. Interventions on women's bodies are not novel, however in the age of epigenetics, new biopolitical strategies are emerging. What I argue is that epigenetics has the potential to reinforce previous models of surveillance through a narrow focus on the molecular and uterine scales. However, if we earnestly consider epigenetic logics, like the ones I outlined in the introduction and throughout the ethnography,

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management in standard prenatal care for the same marginalized communities that are already targeted for interventions.

research questions and methods need to adapt. I encourage an exploration of epigenetics and pregnancy that prioritizes a relational and scalar approach, which includes attending to the political, cultural, gendered, and economic scales of the environments. Rita responded by bringing up a different but related topic:

> Well a public health person asked me what I thought about their plan to implement an intervention to reduce gestational weight gain, and I do not know yet what to do. Perhaps more ethnographic information is needed to understand women's perspective and experience of their weight during pregnancy, [because] you can't tell women while they are pregnant that because they are overweight they are dooming their child.

Public health workers will ask Rita to advise them on prenatal interventions, and her caution to promote more interventions on gestational weight gain is reasonable. Rita represents someone in the field of child maternal health who still questions her own approaches and to a certain extent beliefs about the application of nutritional interventions. Her reflection that more ethnographic work is required to explore different perspectives offers a productive space for collaboration. Moreover, Rita's words about not telling overweight pregnant persons that they are "dooming" their children echoes practices that I had observed in the field. Neither trial ever mentioned the word epigenetics or unpredictable trans-generational inheritance during the recruitment, consent, or intervention phases. However, as Annie's story reminds us, pregnant persons along with scientists will interpret the logics of epigenetics in many different ways.

In what follows, I review my analytical framing and explain how I applied epigenetic logics throughout the chapters in the ethnography. Overall, the ethnography focuses on how epigenetic logics, or rather the selective use or elision of epigenetic logics, emerges in both the

StandUp and SmartStart trials. Moreover, I show how the selective engagement with epigenetics is both changing and reifying older forms of biopolitical intervention on pregnant persons.

The ethnography is guided by three key logics of epigenetics including scale, relationality, and temporality. These logics are derived from Developmental Origins of Health and Disease and epigenetic theories. Through epigenetics, we can understand that the environment includes many different scales. Scale plays a significant role in the conceptual framing of epigenetics and the mechanistic understanding of epigenetics. In addition, the relationality across generations, bodies, and space-time reflects another dynamic to epigenetics. Relationality emphasizes the inter-connectedness of the environmental scales. Through epigenetics we can understand inheritance in a non-Mendelian, non-linear, and non-nuclear way. By non-nuclear, I refer to how trans-generational inheritance expands the boundaries of inheritance to include grandparents and great-grandparents across different generations. Together the logics of relationality and scale underscore how epigenetic environments are compilations of different scales of space and time. Finally, the logic of epigenetic risk also emphasizes time and space but in a different way. The risk of inheriting adverse adaptations from different environmental stimuli is unpredictable and can happen trans-generationally. Therefore, I characterize epigenetic risk as transversal. By transversal, I mean that risk can simultaneously occur across space and time. The three logics that I outlined in the introduction are also woven into the different chapters of the ethnography. My approach emphasizes a key contradiction in the knowledge and practice of epigenetics: although I show how these epigenetic logics exist and could be used to promote a relational ontology, they are often overlooked, elided, or selectively enacted in the design and implementation of clinical trials testing prenatal interventions.

In Chapter Two I highlighted how the emergence of the epigenetic paradigm fundamentally changed the risk associated with maternal nutrition and weight during pregnancy. The comparison between the 1990 and 2009 causal diagrams of gestational weight gain from the Institute of Medicine reflected key changes as a result of epigenetics. For instance, in the 2009 diagram the relational framing between the fetus, future child, and the maternal body was different than the 1990 diagram. For instance, one of the key differences is that in the age of epigenetics the maternal/fetal relationship is not unilateral. Fetuses can change maternal bodies in ways that were not understood twenty years ago, and likewise maternal bodies can affect fetuses through different epigenetic pathways. In the 1990 report the diagram framed the maternal/fetal relationship as unilateral, whereas the 2009 report highlighted the dynamic maternal/fetal relationship. In addition, the 2009 diagram reflects a reconceptualization of the environment that expands the number of influential factors related to obesity during pregnancy and fetal health outcomes. Also, I highlight how the 2009 IOM report associates risk of obesity not just to the mother, but also to the developing fetus, future child, and adult. The different changes to approaching maternal nutrition and weight relate to the epigenetic logics of environmental scale, risk, and relationality.

By using a scalar approach in Chapter Three, I analyzed the ways in which epigenetics emerged in the StandUp trial. My attention to scale reflected the selective focus on the molecular and uterine scales of the environment, which obscured other scales of the environment. For instance, the disciplinary background and expertise of the PI for the StandUp trial influenced her interest in collecting large amounts of bio-samples from pregnant persons and their developing children for the purposes of biomarker analyses. In addition, I highlighted how the translation of animal models into human models also reproduced the selective attention to particular scales of

the environment. I found that animal models hold many unexpected lessons for understanding the translation of social biases across animal and human models. How we study animals can teach us a lot about how we study humans, but not in the way that scientists assume. If we explore the ways in which social and gendered biases frame animal models and therefore also human models, then perhaps future scientific questions can change.

In Chapter Four I also emphasized how the narrow approach to individual bodies and behaviors overlooks other significant epigenetic logics, like the relationality across scales of the environment. Chapter Four explored how the entanglement of environmental scales is difficult to measure and examine using the RCT method in prenatal interventions. Destiny's story reminds us that the way in which her body is entangled with multiple scales of the environment is obscured through the intense focus on her calorie consumption and weight gain. Similar to how scientists study epigenetics in honeybees, the focus on food as exposure in the form of royal jelly, or calories, misses the importance of the collective and inter-dependent forms of food production, labor, and economic climates that also influence the health and behavior of pregnant persons. Together, Chapters Three and Four bring into focus the epigenetic logics of environmental scale and relationality.

Finally, in Chapter Five, I explore how the transversal aspects of epigenetic risk are completely elided through the emphasis on individual responsibility and control. Epigenetic risk is unpredictable and latent, which means that changes could or could not happen at any point in one´s lifetime or across multiple generations (Barker 1992). However, the trials that I examined use RCTs to test prenatal interventions in an effort to reduce future risk of obesity. More importantly, Chapter Five spotlights a key contradiction in the emphasis on individual responsibility, risk, and control. Through epigenetic logics we can understand the

unpredictability and transversality of risk, which also emphasizes that individual bodies cannot control or change the inheritance or occurrence of epigenetic changes. Yet, the models that examine epigenetics in practice target individual pregnant persons, as if they had individual control and full responsibility. The examination of the pregnancy narratives from the StandUp trial reflect the ways in which pregnant participants internalize the individual risk, responsibility, control, and at the same time submit themselves to increased nutritional surveillance.

Critical feminist scholars that examine epigenetics state that targeting a pregnant person for intervention is misguided because individual women have no control over epigenetic modifications (Richardson 2015). I extend this further to argue that in the implementation of nutritional interventions during pregnancy, trials are individualizing responsibility onto pregnant persons, as well as the pregnant participants. The women who participated in the trials submitted to the individual responsibility that was imposed upon them by desiring nutritional surveillance and guidance about their weight gain during pregnancy. The issue of individualized responsibility goes beyond the ways in which epigenetics is targeting the maternal body. Individual women have no control over the epigenetic modifications that can be unpredictably inherited trans-generationally, yet they are targeted as if they have individual control and full responsibility. The key contradiction that the pregnant paradox exposes in epigenetics is that although epigenetic logics claims that there are multiple entangled scales of the environment, studies engaged with epigenetics, like the ones I examined, are targeting individual scales like the uterine, molecular, or maternal environment.

The pregnant paradox coupled with the emergent epigenetic paradigm offers some limits and possibilities for the treatment of pregnant persons. On the one hand, the interpretations of epigenetics in the trials that I examined reflect a reification of older forms of biopolitical

intervention and surveillance. Further, in the new epigenetic paradigm, women's bodies are sliced into different scales of the environment, and interventions are intended to change not only bodily behaviors but also uterine, molecular, and metabolic environments. In the new epigenetic paradigm, it is not only bodily surveillance that is mobilized, but also environmental surveillance at different scales, spaces, and times. I call this the *epigenetic politics* that undergird the justification of nutritional surveillance and management of pregnant persons. Epigenetic politics are characterized by a technoscientific regime that is future oriented (Adams et al., 2009), and focuses on the surveillance of pregnant bodies in the present in an attempt to manage future risk.

On the other hand, a relational approach to epigenetics offers a way to reframe prenatal interventions and interrupt the prioritization of individual responsibility. In a relational approach to epigenetic logics pregnant bodies are one scale of the environment one particular space-time that is connected to different bodies in the past, present, and future. However, in the current approaches to prenatal interventions, individual women are still the key targets. The development of older biopolitical strategies coupled with new epigenetic justifications facilitates the emergence of epigenetic politics. Epigenetic politics affects the treatment of pregnant persons and the comprehensive production of epigenetic knowledge. Overall, I argue that a relational interpretation of epigenetics is aligned with the underlying theoretical concepts of epigenetics.

Furthermore, I argue that the randomized clinical trial method is not the best tool for examining the theoretical concepts underlying epigenetics. Here I will focus on a few main areas that challenge the use of RCT methods in epigenetic studies. The trials that I examined used the randomized clinical trial method to test the effectiveness of nutritional interventions on the health of pregnant persons and future children. However, if the environment can mean anything from toxic chemical exposure, diet and exercise, stress, molecular cell environments, past

experiences, women's bodies, and behaviors, then how can scientists study all of these scales together? RCTs are not designed to test the entangled and relational potential of different scales of the environment.

In addition, RCT methods are designed to find causal linear connections between an intervention and significant differences between the experimental group and control group. The RCTs that currently test nutritional interventions of diet and exercise are overlooking the possibility that children from the pregnant participants may have already inherited epigenetic changes from their grandparents. The unpredictable trans-generational inheritance implicit to epigenetics challenges the viability of using linear causal methods to examine epigenetics. Linear causality does not fit the transversal risk that is implied in epigenetic logics. I find that if we take epigenetics seriously then the concepts underlying the research methods must adapt to the epigenetic concepts.

Finally, the dominance of RCTs to determine health policy, characterizes the contemporary techno-scientific regime. Reflecting on the scientific methods that dominate the current production of scientific knowledge illustrates how epigenetic ideas are applied and tested within an existing scientific environment. The research questions are framed to use RCTs, which produce results that can then influence health policy. Funding agencies prioritize projects that can reduce healthcare costs. Therefore, research projects that engage with epigenetics have to satisfy current funding trends and the methods that are deemed as the "gold-standard." I emphasize that the scientific questions, methods, and political-economic climate are all environmental factors that also influence the production of epigenetic knowledge.

Together, all four chapters incorporate an analysis of epigenetic logics and highlight the contradictions inherent to the knowledge and practice of epigenetics. By showing how certain

aspects of epigenetics are selectively used or not used in particular contexts, I argue that epigenetics is not a homogenous paradigm. Rather, epigenetics in practice is emergent and adapting to various kinds of research agendas, and techno-scientific climates. The illustration of how epigenetic logics are often overlooked in practice aims at emphasizing the limits and possibilities of epigenetics.

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