Title

The Effects of Contemporary Political Economy on Laboratory Labor in the Production of Obesity Knowledge and Environmental Biomedical Subjecthood within the Sciences of Developmental Origins of Health and Disease and Epigenetics

Author

Jeffries Hein, Robbin

Publication Date

2018

Peer reviewed|Thesis/dissertation

UNIVERSITY OF CALIFORNIA

Los Angeles

The Effects of Contemporary Political Economy on Laboratory Labor in the Production of Obesity Knowledge and Environmental Biomedical Subjecthood within the Sciences of Developmental Origins of Health and Disease and Epigenetics

A dissertation submitted in partial satisfaction of the

requirements for the degree Doctor of Philosophy

in Sociology

by

Robbin Marie Jeffries Hein

2018

ABSTRACT OF THE DISSERTATION

The Effects of Contemporary Political Economy on Laboratory Labor
in the Production of Obesity Knowledge and Environmental Biomedical Subjecthood
within the Sciences of Developmental Origins of Health and Disease and Epigenetics

by

Robbin Marie Jeffries Hein
Doctor of Philosophy in Sociology
University of California, Los Angeles, 2018
Professor Hannah Louise Landecker, Chair

Obesity rates have increased over the past few decades in the United States. In response, national research funding has increased, but the field of obesity research is competitive. Therefore, basic science researchers pursue novel explanations that now include environmental determinants of obesity. Those who produce raw experimental data, however, are not principal investigators; they are 'technicians' who are post-doctorates, international medical graduates, and recent immigrant scientists. This dissertation, therefore, analyzes how the political economy of laboratory fact making, in the context of a purported health crisis, shapes obesity knowledge and the biomedical workforce at my field site. I argue that research at my site is structured by

political and economic forces that impact how investigators and technicians conduct scientific practice in a process I call "anticipatory caretaking practices." Drawing on scholarship in science, technology, medicine, and also gender and health, the notion of anticipatory caretaking practices reveals what kinds of obesity knowledge are produced and by whom. I show how political and economic precarity impacts anticipatory practices and how that, in turn, affects laboratory experimental systems. I also illustrate how environmental biomedical subjecthood is produced via model animal organisms of human health. In this context, I also demonstrate how the labor of animal biomass is productive of scientific capital in the form of valuable data. Finally, I describe how grant-based funding impacts investigators as they seek novel yet familiar experimental questions to help make them more competitive in the field of developmental environmental obesity research. Each of these findings reflects changes in laboratory labor and show how, as a consequence of the environmental turn in obesity, an environmental politics of reproduction is produced. This dissertation contributes to scholarship at the intersections of science and technology studies, sociology of health and medicine, gender, and work and occupations.

This dissertation of Robbin Marie Jeffries Hein is approved.

Abigail Cope Saguy

Aaron L. Panofsky

Rachel C. Lee

Hannah Louise Landecker, Committee Chair

University of California, Los Angeles

2018

DEDICATION

This dissertation is dedicated to the memory of my mother, Susan Jo Greer. Like molecular memories that are said to persist epigenetically across generations as marks on our DNA, the marks of love and generosity she left on me continue to endure. You are greatly missed. This dissertation is also dedicated to Wesley Hein, my beloved husband, general merry-maker in the family, and devoted father to our child, Katherine Sophia Hein. My mother, Wesley, and Katy have shown me it is possible to love and to be fully loved without reservation. You are my everything.

TABLE OF CONTENTS

Abstract…… ii
Dedication…… v
Table of Contents……. vi
List of Figures…….. vii
Acknowledgements ……. viii
Curriculum Vita……... xi
Chapter 1: Obesity *Writ* Small: the Molecularization of an Epidemic and the Biomedical Production of Environmental Subjecthood……. 1
Obesity and Developmental Origins of Health and Disease ……... 14
Theoretical Framework – The Environmental Biopolitics of Reproduction and Subjecthood…….. 25
Methodology…… 42
Overview of the Dissertation……... 49
Chapter 2: Laboratory Technicians and Anticipatory Caretaking Practices…… 52
Theoretical Framework: Anticipation as Affective Labor……... 56
Technicians at University Lab……. 62
Anticipatory Caretaking Practices…… 73
Conclusion…… 88
Chapter 3: Biological Labor and the Biomedical Production of Environmental Subjecthood ……. 91
Developmental Origins Models of Appetite and Adiposity…… 94
Implications for the Social Studies of Science: the Meaning of Biological Labor in the Production of Environmental Subjecthood and the Limitations of Biomedicalization in the Environmental Epigenetic Era of Health and Illness..... 115
Conclusion: The Production of Environmental Biomedical Subjecthood…….. 122
Chapter 4: Principal Investigators and the Matter of Precarity……..... 125
Situating the Funding Landscape of Biomedical Basic Science Research in Academia…….. 128
Principals and Precarity at the Lab……... 134
Funding Precarity in Basic Science Obesity Research at the Lab…… 146
"A Somewhat Suspect But Novel Theory of Remodeling" in the Arcuate Nucleus……. 151
Conclusion…… 159
Chapter 5: Conclusions…… 161
Contributions to the Literature……. 163
Technicians and Anticipatory Caretaking Practices…… 164
Biological Labor and the Production of Environmental Biomedical Subjecthood.. 165
Principal Investigators and Anticipatory Caretaking Practices…… 166
Study Limitations and Future Directions……. 167
Afterward……. 171
References…… 172

LIST OF FIGURES

Figure 1.1. Earth as Maternal Environment ……..…….……. 6

Figure 1.2. Health Endpoints of Developmental Origins of Health and Disease…… 9

Figure 1.3. Proposed Timing of Medical Interventions…….. 36

Figure 3.1. Four Models of Appetite and Adiposity in Developmental Origins Research……..... 96

Figure 3.2. Obesogenic Exposure Affects Genetic Expression and Health Endpoints…… 111

ACKNOWLEDGMENTS

I am grateful for the support of many people who have helped me along this journey. First, I would like to acknowledge members of the laboratory where I undertook this ethnography. I am indebted to their willingness to allow a stranger, no less a person from the social sciences, to enter into their world; something they thought most unusual. I am forever grateful for their time and tutorials as I learned about their laboratory lives and the scientific work they carry out. They all work under tremendous pressure that appears to be endemic to academic, basic science biomedical research. Their intellect and good will cannot be measured by citations alone.

Second, without the intellectual guidance and personal mentorship of my advisor, Hannah Landecker, I am not sure I would be writing this at all. I am so thankful to have found a person that makes me feel at home among those who embrace and traverse multiple intellectual terrains, taking us beyond the usual confines of ordinary disciplinary science into new areas of exploration. She is also an extraordinary human being who makes the journey accessible and enjoyable. This dissertation is a tribute to her generosity over the years.

Along this journey, I have also benefited from the insights of professors at different stages of my time in the doctoral program. In chronological order, I am grateful to C.K. Lee for not only guiding the ethnographic sequence, but for helping me think through questions of labor at my site. I am grateful to Abigail Saguy and Stefan Timmermans for early course work and their help with completing the Master's Paper. I am also very appreciative of my time teaching spring courses within the yearlong General Education Cluster course on biology and gender, which Professor Saguy facilitated. And, in my last few years in the program, I had the

opportunity to teach unique courses in a service-learning environment. I am grateful to Beth Goodhue, Associate Director for the Center of Community Learning, for allowing me to explore this pedagogical path. I am also grateful for meeting Rachel Lee in the context of the Life (Un)Limited reading group, and for agreeing to being on my dissertation committee. I also owe a debt of gratitude to Aaron Panofsky for being one of my faculty advisors and a member of this committee. Finally, I am grateful for the support and technical help that Kat Malinksy and Maria Sanchez Patino provided for me from the start of this dissertation to its finish.

I would also like to acknowledge friends that have taken this ride with me, dating back several decades: old friends, Mike Vivian and Steve Sherwood. I am grateful for your humor and your patience (and free therapy) over the years. Jessica Carbino, Molly Jacobs, and Carrie Sanders—thank you for your friendship. I am also very grateful for my friends, the late Alan Emery and Tony Ellis, both of whom were taken far too soon. They were close friends of mine from the early days of undergraduate life, and who, by different and wonderful circumstances, came to star as prominent figures in my makeshift family over the course of many years. I miss your wit and nerdy intellect.

This dissertation includes very illustrative figures whose publishers have kindly granted me permission to use them. For that, I am indebted to all of them.

Last, but not least, I am grateful for my partner, Wesley, who really provides the foundation for my life. Thank you for being so generous with me. I also owe tremendous gratitude to my step-dad, David, for the love and care he gave to my mom for the last five years of her life. I was newly pregnant when my mom was diagnosed with an advanced form of multiple myeloma, and without David's care for my mom, I would not be writing this dissertation. As well, within the family circle that has grown, I would also like to acknowledge

my stepchildren, Alex and Mira Hein, who bring light into Katy's and my life. Thanks also go to my father, John, and his partner, Eunhyon Chi, who help round out our modern family.

CURRICULUM VITA

INTERESTS

Science and Technology Studies, Medical Sociology, Gender, Health Inequalities, Ethnographic and Interview Methodologies

EDUCATION

University of California, Los Angeles — Los Angeles, CA
Ph.D. Expected — June 2018
- Dissertation: "The Effects of Contemporary Political Economy on Laboratory Labor in the Production of Obesity Knowledge and Environmental Biomedical Subjecthood within the Sciences of Developmental Origins of Health and Disease and Epigenetics"
- Committee: Hannah Landecker (chair), Abigail Saguy, Aaron Panofsky, and Rachel Lee

University of California, Los Angeles — Los Angeles, CA
C. Phil., Sociology — September 2015
M.A., Sociology — December 2011

The London School of Economics and Political Science — London, UK
M.Sc., Sociology, honors — Summer 1995

University of California, Los Angeles — Los Angeles, CA
B.A., Sociology, *summa cum laude* — September 1994

Los Angeles Valley College — Valley Glen, CA
UC Transfer Student Program — June 1992

GRANTS AND AWARDS

Social Science Research Council, Dissertation Proposal Delegate — 2014
Graduate Summer Research Mentorship — 2011
Graduate Summer Research Mentorship — 2010
Center for the Study of Society and Genetics, travel grant — 2010
Center for the Study of Women, travel grant — 2010
Departmental Fellowship — 2009-2010

TEACHING EXPERIENCE

University of California, Los Angeles
- Teaching Fellow, *Society and Genetics 195CE* — 2014-2017

Received 8.3/10 (2014-2015), 8.4/10 (2015-2016) rating for coordinator (TA)

- Teaching Assistant, *Sociology 195CE* 2013-2014
  Received 8.1/10 rating of coordinator (TA) over academic year
- Seminar Instructor, *GE Cluster: Sex: From Biology to Gendered Society, "Sociology of Kink/Knowledge."* Spring 2013
  Received 4.88/5 overall rating of instructor
- Seminar Instructor, *GE Cluster: Sex: From Biology to Gendered Society, "Sociology of Kink/Knowledge"* Spring 2012
  Received 4.75/5 overall rating of instructor
- Teaching Assistant, GE Cluster: *Sex*: *From Biology to Gendered Society* Fall 2011-
  Received 8.89/10 average rating of teaching assistant Winter 2012

Los Angeles Valley College 2000-2012

- Instructor of Sociology (Adjunct), Courses Taught, *Introduction to Sociology* (including Title V Learning Communities with English; Supplemental Instruction; Service Learning); *Crime and Delinquency*; *Marriage and Family*; *Race and Ethnicity* (Title V Learning Community with Chicano Studies); *Sociology of Women; Human* Sexuality; and, *Social Problems*
- Teaching Awards: Apple Award, for teaching excellence 2008
  Above & Beyond Award for service to students 2001

Santa Monica College 2003-2009

- Associate Professor of Sociology (Adjunct), Courses Taught, *Introduction to Sociology*; *Sociology of Sex and Gender*; *Social Problems*
- Teaching Awards: Instructor of the Year acknowledgment, International Students Association 2005-2006
- Professional Service:
  - Student Learning Outcomes departmental representative 2005
  - Senate Committee member on instructor evaluations 2005

Chapter 1

Obesity *Writ* Small: the Molecularization of an Epidemic and the Biomedical Production of Environmental Subjecthood

> Obesity is a disorder, which, like venereal disease, is blamed upon the patient. The finding that treatment doesn't work is ascribed to lack of fortitude. Corpulence in America is regarded along with narcotic addiction as something wicked, and I shall not be surprised if soon we have a prohibition against it in the name of national security…. I wish to propose that obesity is an inherited disorder and due to a genetically determined defect in an enzyme; in other words, that people who are fat are born fat, and nothing much can be done about it.
>
> Edwin B. Astwood, physiologist and endocrinologist, 1962.

> Modernization was indeed enacted on the landscapes of North America but also—and unwittingly—on the people who inhabited those landscapes. It is a question not simply of how a manipulated environment has broadly influenced society but also of how environments have shaped human flesh in minute and profound ways.
>
> Linda Nash, historian, 2006.

According to the Centers for Disease Control and Prevention (CDC), more than thirty-six percent of adults, and approximately seventeen percent of children ages two to nineteen, are obese (Ogden, Carroll, Fryar, & Flegal, 2015). Although these rates have stabilized in recent years, obesity rates in general rose across all social, racial, ethnic, and gender categories over the past several decades (Ogden, Carroll, Kit, & Flegal, 2014). They remain alarmingly higher than they were a generation ago, according to a twenty-five year historical analysis provided by the Robert Wood Johnson Foundation in 2016. The CDC also estimates that treating obesity and its comorbidities cost approximately $147 billion a year; overtaking tobacco as a national health concern (Centers for Disease Control and Prevention, 2012). According to one obesity researcher, James Levine, "sitting is the new smoking" (Johnson, 2016).Within this context, obesity prevention remains a top priority among many health experts who define obesity,

particularly among children, as an "urgent call to action" (Robert Wood Foundation, 2016).[1] In an effort to discover new explanatory models of obesity, and to develop better therapeutic tools, the National Institutes of Health (NIH) funded $965 million dollars in obesity research in 2016 and the American Diabetes Association contributed a further $34.5 million (National Institutes of Health, 2017a; American Diabetes Association, 2017).

Elsewhere, in another corner of biomedical research at the CDC, researchers have found that the American population is exposed to multiple environmental pollutants. Researchers at CDC's Environmental Health Laboratory analyzed over 200 chemical exposures in blood and urine samples from participants in the National Health and Nutrition Examination Survey (Centers for Disease Control and Prevention, 2009). They tested samples for chemicals, or its metabolite (small molecules produced when body tissues chemically alter the original compound it is exposed to) to measure the presence of these 200+ chemicals. Their findings indicate that there is "widespread exposure to some commonly used industrial chemicals" such as fire retardants, bisphenol-A (BPA) used in food packaging, and polymers used in the manufacture of non-stick cookware, clothing, and many other products (p. 3). In another nationally representative sample, public health researchers studied the presence of multiple chemical exposures in children. They concluded that "the percentage of children with detectable

[1] Numerous authors have argued that causal and associative links drawn between obesity and poor health outcomes are problematic. George Bray, an early American obesity researcher, thought obesity and metabolic disturbance posed a chicken or egg problem, which still perplexes scientists today. In the 1960s, he and others wondered, "Does the obesity produce insulin resistance, or does insulin resistance precede obesity and play a role in its development? Are other hormonal and endocrine changes that we measure a result of being obese, or do they contribute to the onset of the obesity?" (2015, pp. 11-12) The difficulties of undoing the Gordian knot in obesity research remain. While important to discussions about stigma and medicalization of fat, this dissertation is not a study of scientific controversy or claims made around the science of obesity in and of itself. For important work that contests claims between body size, fat composition, weight, measurements like body mass index, and health and illness, see Campos (2004), Guthman (2011), and Saguy (2013).

concentrations of an individual chemical ranged from 25 to 100%; the average was 93%, and 29 of 36 [chemical analytes] were detected in more than 90% of children" (Hendryx & Luo, 2017, p. 5336). Together, these data suggest that we are all environmentally constituted by chemical exposures; some of which are known to be harmful. Fire retardants, BPA, and polymers, for instance, are widely believed to affect metabolism and reproduction because of their impact on hormone sensitive organs, including adipose (fat) tissue, skeletal muscle, the liver, pancreas, hypothalamus, and reproductive organs (McAllister et al., 2009). As a result, obesity researchers have begun to consider the physiological functioning of social nature.

Observers of both health trends—rates of obesity and chemical exposure—suggest that the two are linked (Grün & Blumberg, 2009a; Gore et al., 2015; Holtcamp, 2012; Landecker, 2011; Janesick & Blumberg, 2011, 2016; Guthman, 2012). In contrast to thinking solely about obesity as a result of an imbalance of energy expenditure (too many calories, too little exercise), researchers in disparate but overlapping scientific disciplines believe that the two phenomena are related in what might be called an environmental turn in obesity studies. In short, the chemical environment is making us fat and metabolically sick. Concepts such as the "obesogen" (Grün & Blumberg, 2009b) and the "exposome" (Wild, 2005, 2012), both of which emphasize environmental determinants of health, now appear in journals such as *Nature*, *Environmental Health Perspectives*, and *Obstetrics and Gynecology*. In 2011, grants issued by the National Institute of Environmental Health Science added obesity as a new health endpoint in studies that associate early-life exposure with later-life disease and the "propagation of non-communicable disease across the lifespan" (Haugen, Schug, Collman, & Heindel, 2014).

Today, it is more widely acknowledged across seemingly disparate biomedical fields (e.g., cancer, neurology, reproductive health) and in site-specific sites and practices (e.g.,

clinical, laboratory, public health) that "the" environment is a key factor in understanding contemporary biology and illnesses associated with 21st century life. For example, Siroux, Agier, and Slama in their 2016 review of the exposome concept noted that the "number of citations of 'exposome' in PubMed has increased over recent years; from six in 2005 to 150 in 2010 and > 1600 in 2015," marking a shift in attention to the neglected half of gene-environment studies (p. 125). Similarly, I found that a search of "exposome" citations in the Web of Science indicates that its usage grew from 2 in 2007 to 1,469 in 2017. An example of exposome research includes Hendryx and Luo's 2017 analysis of multiple chemical exposures on children in which the authors used data gathered by the US National Health and Nutrition Examination Survey (NHANES) 2003-2012. They found that the average of detectable concentrations of an individual chemical in children was 93% (ranging from 26 to 100%) while more than 90% of children show 29 out of 36 chemical analytes. These authors also found (unsurprisingly) that levels of environmental exposure vary by social class, race, and sex. In general, exposome studies, such as this, could be described as illustrations of the physiological functioning of social nature in which environmental exposures delivered at different times and in different ways throughout the life course (even beginning with the germ line and therefore before conception) are found to affect biological processes.

According to Felix Grün and Bruce Blumberg (2009), exposure to BPA may in fact predispose people to weight gain "despite normal diet and exercise" (p. 1128). In an article on the plurality of obesity *across* species, Yann Klimentidis et al. (2011) found that domestic and feral animals living in industrial environments alongside humans have grown fatter over the past few decades. This same study also found that laboratory animals, despite living their entire lives in highly regulated institutional settings, are also getting heavier. Even babies seem to be

affected. According to Ogden, Carroll, Kit, & Flegal (2012), the heavy weight prevalence for infants from birth to six months of age also has risen since the 1970s, but no one is suggesting that newborns are lazy couch potatoes.[2] At the heart of environmental studies of obesity lies the question of how exactly, at the molecular level, chemicals enter the body and disrupt physiological systems. Disruptions, it is thought, lead to multiple physiological alterations that include adipogenesis (development of fat cells from stem cells), lipogenesis (formation of fatty acids that affects triglycerides, which are key constituents of fat cells), and appetite-satiety pathways (among sets of neurons) located in a small region of the hypothalamus.

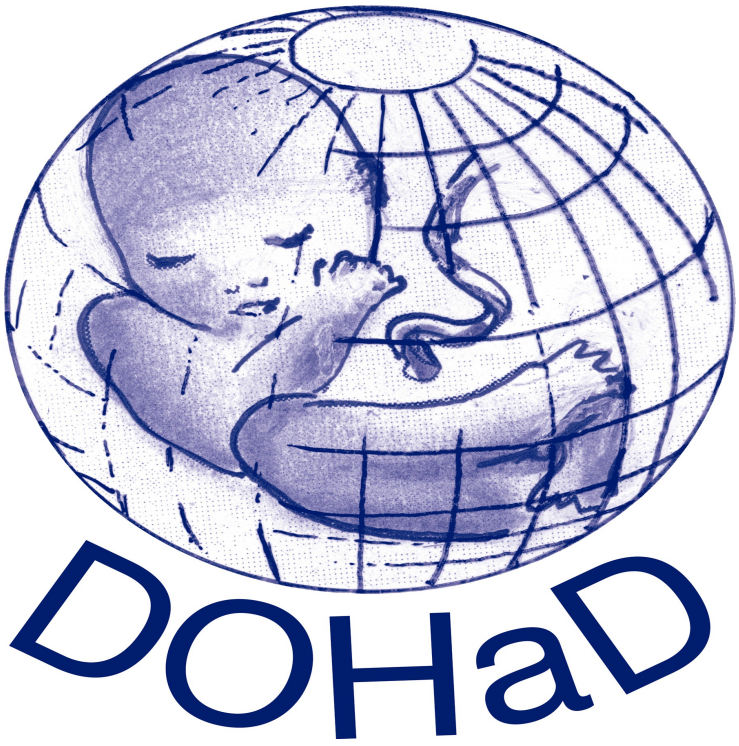
The environmental turn in obesity also argues that timing of nutritional and chemical exposure matters for long-term health. Research from epidemiology, endocrinology, and perinatology shows that exposure during uterine and neonatal life, in particular, affects offspring's health into adulthood; essentially programming children for ill health in later life (Hales & Barker, 1992; Barker, 1998; Nathanielsz, 1999; Gluckman, Hanson, Cooper, & Thornburg, 2008). The theory of Developmental Origins of Health and Disease (Developmental Origins) argues that epigenetic mechanisms (modifications of genetic expression without changing genetic sequence) caused by environmental insults during fetal and neonatal life can permanently alter organs. As applied to obesity and metabolic syndrome, Developmental Origins predicts that nutritional and chemical exposure *in utero* acts epigenetically, leading to an increase and dysfunction of fat mass, glucose intolerance, enhanced appetite, and other obese phenotypes.

---

[2] There is no recommended definition of obesity for children less than two. *Excess weight* is measured by the CDC as "weight for recumbent length at or above the 95th percentile" on sex-specific, weight for recumbent length growth charts. See https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4770258/. In 2000, the CDC revised the 1977 National Center for Health Statistics (NCHS) Growth Charts for infants, young children and adolescents in use since then. See https://www.cdc.gov/nchs/data/series/sr_11/sr11_246.pdf

Sources of chronic disease include multiple types of environments, from the womb at the most intimate and proximate scale, to ambient exposures of everyday life, to the intergenerational transmission of epigenetic tags. To illustrate the layers of environment from the maternal-fetal relationship to distal, external sources, the International Society for Developmental Origins of Health and Disease symbolizes the complexities and interrelations of these exposure scales as follows:

Figure 1.1. Earth as Maternal Environment



Note: Logo of the International Society for Developmental Origins of Health and Disease (DOHaD).

It is because of these profound shifts in the environmental problematizing of obesity through a maternal-fetal framework that I designed an ethnographic study focused on a laboratory site I call University Laboratory (Lab). The Lab is a basic science research laboratory that uses animal models to experimentally study the effects of a maternal high-fat diet and

maternal BPA on the health of rodent offspring. I follow the "ontological politics" of how "bodies are shaped, and lives are pushed and pulled into one shape or another" within the environmental epigenetics of obesity through maternal-fetal medicine (Mol, 2002, p. viii). At the intersection of obesity, gender, and the environment, researchers at the Lab enumerate the social facets of physiological functioning; opening new avenues of causal explanation as to how and why obesity and metabolic syndrome occurs, and how it may persist across generations. Work at the Lab, therefore, provides a "window on the ways that environmentally defined subjects are constituted through biomedical knowledge and politics" around the problematic of obesity (Olson, 2010, p. 171). For sociologists, studying the embodiment of presumptively harmful environmental exposure and its unequal distribution is important for numerous reasons, including a refined understanding of how new patterns of stratified health emerge and reproduce modes of social stratification (Landecker & Panofsky, 2013). For both life and social scientists, a focus on "milieu" (Olson, 2010) in the postgenomic era is a focus not on life *per se*, but on gene-environment interactions as the relevant place of power-laden interventions.

As a result of this scientific-medical paradigm, the logic and practice of environmental epigenetics and Developmental Origins create a new round of politics of reproduction, but one that is thoroughly environmental (Ginsburg & Rapp, 1991). Environmental epigenetics shifts what is perceptible across bodies in space (multiple environs) and time (multiple lifespans) and, therefore, what can be medically managed. Pregnancy and lactation are central sites of investigation at the Lab and they are also proposed sites of medical intervention for humans. As I will show, pregnancy has explicitly become an anticipatory site of medical intervention among Developmental Origins researchers. Such a "regime of perceptibility" in obesity research refigures both definitions and relations between bodies, the environment, and the life course

(Murphy, 2006, p. 24). New forms of surveillance emerge and are expressed through the language of epigenetic risk and the governance of life through maternal responsibility. What is at stake in this model is not an isolated biomedical subject, but rather the "vital technical aspects of milieus" that constitute our very being (Olson, 2010, pp. 180-181). Our bodies are altered by everyday exposures. What begins as an ambient source of exposure becomes internalized; affecting fetal development through the maternal-fetal interface and creating, in essence, an intergenerational and environmental model of health[3] (Wells, 2010; Landecker & Panofsky, 2013).

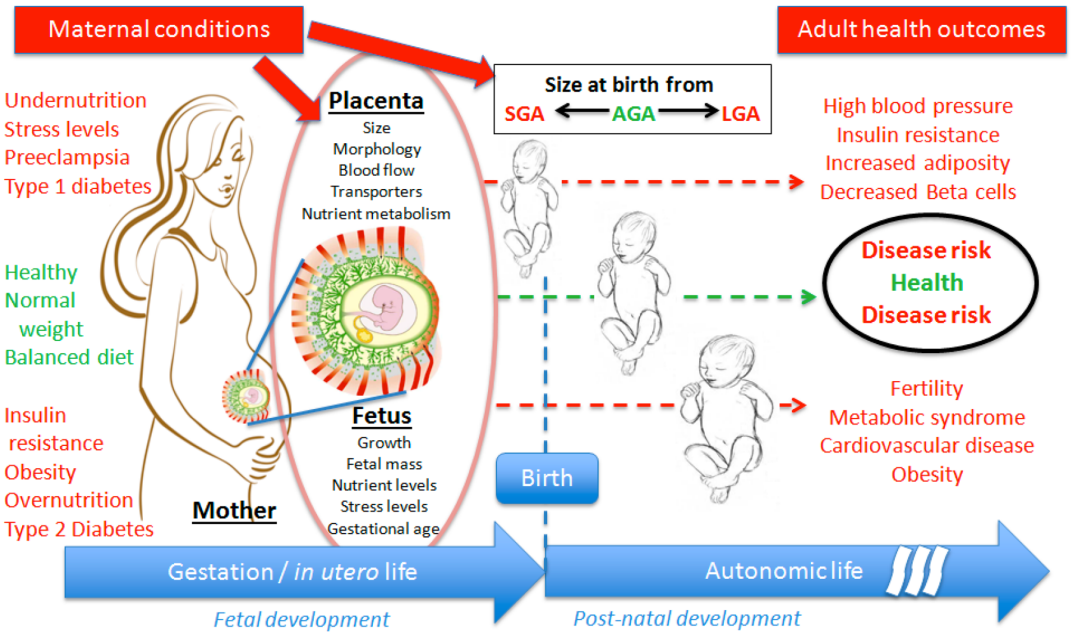
According to one obesity researcher:

> If this [developmental programming] is true, it's hugely important because it means that if we get pregnancy right, we have the opportunity to get that person's later life health in good shape and, conversely, if it goes wrong, that person is destined for later life ill-health. (Anonymous, personal communication, March, 2015)

To illustrate this researcher's position and the model of Developmental Origins in general, I am including a schematic figure created by Chavatte-Palmer, Tarrade, & Rousseau-Ralliard (2016, p. 5).

[3] Definitions of metabolic syndrome change over time and across professional medical societies. See Huang (2009) for a review of various definitions.

Figure 1.2. Health Endpoints of Developmental Origins of Health and Disease



In this figure, the researchers illustrate contemporary models of Developmental Origins in which two sub-optimal nutritional environments (maternal under-and over-nutrition) are believed to program the fetus for increased adiposity (increased fat cells) in small-for-gestational age babies, and obesity and metabolic syndrome for large-for-gestational age offspring. A U-shaped relationship, therefore, exists between the maternal environment as defined by nutrition and the health outcomes for adult offspring.

Against this backdrop of an environmental turn in obesity and an environmental politics of reproduction, I also found that the work of producing laboratory knowledge is carried out by individuals whose own lives and career trajectories are structured by external sources of precarity in grant-based funding and the politics of H-1B visas for immigrant scientists. Thus in addition to showing how the Lab articulates causes of obesity in environmental terms, I illustrate how the political economy of laboratory fact making, in the context of a purported health crisis, shapes new obesity knowledge. Funding precarity, though, is not new. In an autobiographical

account of his career in obesity research, Dr. George Bray (2015) recalled the following:

> Research work requires money. To fund my research work, whatever it was going to be, I wrote two grants for the NIH. One of them elaborated on the work with T3 and adipose tissue, and the other one focused on unraveling the mystery of the Zucker rats. When Study Sections at the NIH reviewed these grants, the one examining the Zucker fatty rats received a high score and was funded; *the other got a low score and was not funded*. My directional sails were thus set—it was the beginning of a career in obesity research from which I have not looked back. (p. 11, my emphasis)

Yet what is new, according to the National Institutes of Health (2012), is that most laboratories "consist of a PI [principal investigator] and one or a *small number of permanent technical staff*, with the majority of the research, carried out by trainees [post-doctorates]" (p. 10, my emphasis). In other words, it is technical staff and students who produce the raw data on which PIs depend. Further, "more than half of the biomedical postdoctoral researchers in this country hold non-U.S. citizenship" (National Research Council, 2005, p. 6). As a result, "it is difficult to consider the U.S. biomedical research enterprise without acknowledging the critical role played by scientists from outside the U.S." (National Research Council, 2005, p. 6). Despite these characterizations of contemporary laboratory life, there is scant data on the international biomedical workforce in general and technical staff in particular.[4] According to the Survey of Graduate Students and Postdoctorates in Science and Engineering (GSS), less than half of the population of post-doctorates in cell and molecular biology that are counted are women. Further, we know very little about the gender balance among permanent technical staff in the field of basic science, academic laboratories because the data is not collected. In sum, we know funding

[4] The Survey of Doctorate Recipients (SDR) is considered the gold standard for data on US-trained PhD careers and training in the sciences. The survey does not, however, collect data on foreign-trained PhDs. The Survey of Graduate Students and Postdoctorates in Science and Engineering (GSS) is the only source of information on international doctorate-holders working in academia. Nevertheless, neither of these surveys account for immigrant scientists with PhDs or MDs who work permanently as so-called technicians in basic science laboratories.

influences the kinds of research questions PIs pursue, but those carrying out the experimental research today are very likely technical staff and students, approximately half of whom are recent immigrants. This means, therefore, that not only is funding precarity built into the doing of science itself but so is political precarity of foreign scientists who are dependent on visas for their livelihood. In this sense, the work of producing laboratory knowledge is characterized by attributes historically associated with so-called women's work, namely insecurity, low pay, and low status (Oksala, 2016).

Therefore, in addition to showing how the Lab produces environmental models of obesity, and therefore medical models of environmental subjecthood, I argue that investigators and technicians in the Lab are differently bounded by larger political-economic forces that are relatively invisible and that this affects how laboratory science is produced. Precarity in grant-based funding and immigration status affects one's occupational positioning within the Lab and, I will argue, the content and course of knowledge production. For temporary technicians, in particular, there is a drive to produce positive findings in keeping with "normal science" (Kuhn, 2012), while for career technicians there is a drive to assist PIs in pushing existing "experimental systems" (Rheinberger, 1997) in novel directions. At stake is knowledge itself, but also the livelihoods and wellbeing of a vulnerable population of scientists who are recent immigrants that exist in relation to shifts in immigration status; all of which is heightened under the Trump administration. Political and economic forces, I argue, have epistemic effects in terms of what the Lab studies and, therefore, what it contributes to the market for new obesity facts.

What I have found is that various forms of labor braid together at my research site, and, based on data collected by various national agencies on biomedicine funding and the biomedical workforce, my findings are not idiosyncratic. Therefore, this dissertation is a case study of how

the political economy of laboratory fact making, in the context of the obesity crisis, shapes new obesity knowledge (Ragin & Becker, 1992). Structural forces shape what technicians and investigators learn to care for and about (i.e., specific tissues and specific regulatory systems that regulate appetite) in terms of creating new knowledge. First, technicians at the Lab engage in *anticipatory caretaking practice*s, a form of labor that is shaped by whether one is a temporary technician or a career technician. Technicians differently perform the embodied labor necessary to "having a feeling for the organism" in the production of laboratory facts (Keller, 1983). I use the notion of *anticipatory caretaking practices* to characterize the persistent sense of anxiety among technicians and investigators in terms of grant renewals, publications, and precarity in visa status. Caretaking, I argue, has situated meaning that varies by one's social location in the Lab's division of labor (Müller & Kenney, 2014). In particular, career technicians learn to care for the Lab's experimental systems (Rheinberger, 1997) in such a way that they are able to explore novel explanatory models of obesity while temporary technicians, on the other hand, are compelled to re-produce "normal science" (Kuhn, 2012). Second, as a result of experimental practices, I also argue that the biological labor of animal bodies is made to perform differently and, as a result, is a source of economic and reputational value for the Lab in what Donna Haraway (2008) refers to as a "biomedical mode of reproduction." Third, I find that PIs labor in offices reproducing their livelihood and the Lab itself through anticipatory caretaking practices at the level of inscription (writing) practices. PIs work at an arm's length from experimental practice; raising questions anew about what constitutes the vocation of "scientist" and who gets to claim that identity when it is technicians who generate raw data.

In summary, a confluence of disparate factors—the national problematic of obesity, a concomitant growth in data on environmental exposure, a highly competitive and precarious

grant-based funding landscape, and the reliance on a low-cost yet politically entangled workforce that consists of recent immigrant scientists—coalesce in the Lab to create specific kinds of laboratory facts about obesity. That is why this dissertation examines 'who' produces the 'what' of obesity knowledge in the process of producing an environmental politics of obesity and metabolic syndrome through the framework of Developmental Origins and epigenetics.

The intersection of contemporary laboratory life with the obesity epidemic helps reveal less visible forms of labor that are necessary to basic science research. It helps reveal who is in the biomedical workforce as part of the larger scientific-medical pipeline and how, I have found, this has epistemic effects on what kinds of empirical questions are asked and, therefore, what knowledge is put out into the world. The process of creating new laboratory knowledge, then, involves multiple moving parts. I borrow a concept from anatomy to illustrate their interdependence. A 'point of articulation' connects two or more bones that allows motion, and these points of articulation that link together various aspects of laboratory labor include: a) anticipatory caretaking practices by technicians as they both create and extend the Lab's existing experimental systems (Rheinberger, 1997) that is discussed in chapter 2; b) the labor of biomass, i.e., animal models, necessary to constructing environmental subjecthood through the creation of valuable data (presented in chapter 3); and c) the anticipatory writing practices of PIs, which are necessary to advance novel theories of maternal/fetal disease transmission in a larger political economy of research funding (discussed in chapter 4). In a turn toward the environmental problematizing of obesity, I argue that these forms of labor link the strategic relations of scientific work in a 21st century laboratory to wider social practices and values about health and disease (Landecker, 2007).

In the pages that follow, I first detail current definitions of obesity and its prevalence across demographic groups. I also document early models of biological determinism in obesity research and incorporate more contemporary challenges that support an environmental science of obesity through the lens of epigenetics and Developmental Origins programming. I then situate this study in an interdisciplinary theoretical framework that builds on laboratory studies, sociology of work, gender, and medical sociology. Within each discipline, I place an emphasis on anticipatory caretaking practices because they affect the final work product that contributes to the obesity knowledge market. In doing so, I highlight what has been described as the "back room" of science, which to me seems very much like the front room (Barley & Bechky, 1994). Lastly, I describe my methodological approach in the study and conclude with an overview of the dissertation chapters.

Obesity and Developmental Origins of Health and Disease

The scientific and cultural context of my dissertation research is the so-called obesity epidemic (Saguy, 2013). According to the CDC, obesity is measured in reference to body mass index (BMI), which is "calculated by dividing a person's weight in kilograms by the square of height in meters" (Centers for Disease Control and Prevention, 2015, para. 2).[5] Based on these measurements, roughly one-third of American adults are obese, and approximately seventeen percent of children between two and nineteen years old are classified obese (Centers for Disease Control and Prevention, 2016). An estimated ten percent of children aged two to five are considered obese and, while obesity is not defined for children less than two, pediatric growth charts exist to track weight relative to recumbent length (Ogden et al., 2014; Ogden et al., 2015).

[5]A normal adult BMI is 18.5 to < 25; 25 to <30 is overweight, and 30.0 or greater is obese. BMI formulas shift for children and adults because a) children and adults have overall different body compositions, b) children vary by age, say from 2 to 11, and c) children vary more by sex as they mature into adults.

Obesity rates also vary across categories defined by income, gender, race, ethnicity, education, and geographical location. In general, those with lower levels of education and income are more inclined to be obese or overweight relative to those with higher levels of education and income. In terms of race and ethnicity adjusted for age, non-Hispanic African Americans and Hispanics have the highest rates of obesity followed by non-Hispanic whites and non-Hispanic Asians. Whereas no sex difference in prevalence was found among youth in the 2015 National Center for Health Statistics Data Brief, the prevalence for adult women is higher than men (National Center for Health Statistics, 2016).

Obesity is of such centrality to biomedical research because of the epidemiological links that are drawn between BMI or waist circumference and a wide rage of health outcomes. Obese women are at greater risk for irregular menstrual cycles, polycystic ovarian syndrome, and therefore infertility (Jungheim, Travieso, & Hopeman, 2013). Overall, between 1980 and 2002, obesity rates rose, and doubled among adults aged twenty and over, leveling off since approximately 2010 (Ogden, Carroll, & Curtin, 2006). Although prevalence rates for children tripled from 1980-2002, they have remained stable from 2003-2014 (Ogden et al., 2016). In spite of a recent leveling off of trends, the historical rise over the past several decades has meant childhood rates remain a priority for public health officials.

The conventional understanding of obesity is an excess of calories relative to energy expenditure. The CDC, however, recognizes there are multifactorial causes of adult and childhood obesity and the key contributors are categorized as either behavior or genetics. "Behaviors can include dietary patterns, physical activity, inactivity, medication use, and other exposures. Additional contributing factors in our society include the food and physical activity environment, education and skills, and food marketing in promotion" (Centers for Disease

Control and Prevention, 2017, para. 1). Excess fat matters because "it is associated with poorer mental health outcomes, reduced quality of life, and the leading causes of death in the U.S. and worldwide, including diabetes, heart disease, stroke, and some types of cancer" (para. 2).

Early challenges to conventional wisdom on obesity. Early obesity researchers suspected a deterministic role for genes and hormones in adipogenesis.[6] This history is important for sketching out the current landscape because in 1949, for example, researchers at The Jackson Laboratory observed "some very plump" mice among a stock of littermates (Ingalls, Dickie, & Snell, 1950, p. 318). After breeding offspring of heterozygote mice (individuals that have two different alleles of a particular gene who can therefore reproduce genetic variability in pups), the ratio of fat to lean mice suggested to researchers that a genetic mutation caused some of the offspring to be fat.

At another East Coast laboratory, the wife-husband team of Lois and Theodore Zucker (1961) identified a new fatty mutation in a stock of laboratory rats in 1961.[7] Like the earlier mice discovery, Zuckers' fatty rats appeared "spontaneously," and the researchers theorized that an "obviously deranged lipid metabolism" had caused the obesity (p. 275, 277). They also stated "they were genetically obese rats that inherited their obesity as an autosomal recessive trait. Weak willpower was clearly not the reason why these rats were fat—it was something in their genes" (Bray, 2015, p. 10-11). Dr. Edwin B. Astwood (a prominent clinician, researcher, and friend of the Zuckers) was so intrigued by these findings, which suggested a "deranged

[6] Adipose means fat. Adipogenesis is the study of fat cell differentiation, specifically how preadipocytes (uncommitted cells) become adipocytes (committed cells that store fat). This is important to know because deleterious changes in hormones, for example, can commit preadipoctye cells to become adipocytes; therefore, increasing one's fat cells. High-fat food and stress, social factors, can become embodied and, therefore, affect cell differentiation; a process in which the social becomes biological.

[7] In 1995, Ogawa et al. showed that the *fa* gene is the leptin receptor gene.

metabolism," that he gave his 1962 Presidential Address to the Endocrine Society on the topic. On the *Heritage of Corpulence*, he argued,

> When obesity has arisen in animals as a consequence of inbreeding or from chance mutation, something about the mode of inheritance can be deduced, but in man the [wrong-headed] conviction of the 'primacy of gluttony' has discouraged genetic investigation, and our information is scant indeed. (p. 338)

Dr. Astwood wondered: Why are scientists content to ascribe height and other phenotypes to inheritance but not obesity? As if to further convince his audience of the biological basis of obesity, he recalled a historical account of doctor-patient medical interaction from 1825 in which the patient describes his misery:

> Sir I have followed your prescription as if my life depended on it, and I have ascertained that during this month I have lost some three pounds, or a little more. But in order to reach this result, I have been obliged to do such violence to all my tastes and all my habits—in a word, I have suffered so much—that while giving you my best thanks for your kind directions, I renounce any advantages from them and throw myself for the future entirely into the hands of Providence! (as cited in Astwood, 1962, p. 341)

Dr. Astwood not only encouraged genetic investigation, he also proposed the novel idea at the time that obesity does not result from a lack of will-power but rather it results from the consequence of "inherited [metabolic] defects [in enzymes]; [and, he concluded] if this be so and we like food, we might as well eat up and be happy" (p. 337). People who are fat are born fat, he and others argued, and "nothing much can be done about it" (p. 337). Early examples such as this contradicted the personal blame theory of obesity and nurtured a more biologically deterministic approach to obesity studies.

In the mid-1990s, Jeffrey Friedman, a researcher at The Rockefeller University in New York, discovered that mice missing a gene (due to a genetic mutation) become fat in its absence. He named this gene, *ob*, and stated that it normally produces a protein (leptin) that signals the

brain to know when fat cells are satisfied. In its absence, the biochemical receptor of that message is not created, and hunger is not inhibited (Howard Hughes Medical Institute, 2008).

Of particular significance to the topic of this dissertation is the intersection of research on metabolic disorders and the rise of a new lifespan model for thinking about chronic adult disease. So while American researchers were stumbling upon mutant fatty rodents, and eventually identifying aspects of biologically determined obesity and diabetes, a British epidemiologist named David Barker was rummaging birth records across the South of England and Wales based on a suspicion that babies with low birth weights were associated with poor cardiovascular health in adulthood and early mortality rates; creating in effect a lifespan model of obesity. During his tenure at the Medical Research Center Environmental Epidemiology Unit in the 1980s, Barker observed that county maps of the United Kingdom in the 1910s and 1920s showed strong geographical correlations between place and weight of birth and cardiovascular mortalities. Later, in 1986, Barker and Osmond argued that death rates and chronic conditions like ischemic heart disease (heart problems caused by narrow heart arteries) were associated with "poor nutrition in early life" because it "increases susceptibility to the effects of an affluent [high fat] diet" (p.1077). In 1995, at the same time Friedman had identified the *ob* and *db* genes, Barker was the first to formally articulate the "fetal origins hypothesis." His original model "states that fetal under-nutrition in middle to late gestation… leads to disproportionate fetal growth [catch up growth that leads to heavier weights and] programmes later coronary heart disease" (1995, p. 171). Barker's findings led to "the initially controversial but now widely accepted idea that common chronic illnesses such as cancer, cardiovascular disease and diabetes result not always from bad genes and an unhealthy lifestyle, but from poor intrauterine and early postnatal health" (The Guardian, 2013, para.1).

As with earlier findings among American researchers on the genetics of obesity, the Barker Hypothesis, as it was initially called, similarly challenged conventional wisdom about causes of obesity. Rather than disparaging people for bad habits, Barker hypothesized that a mother's poor nutritional environment programs fetal tissues and organs in such a way that offspring are destined to have deleterious long-term health effects, including increased adiposity, insulin resistance, and the risk of cardiovascular disease. The key therefore to creating healthier people is to ensure good nutrition for women, particularly those of reproductive age and women in lower socio-economic groups who tended to have smaller babies for gestational age. Early calls for nutritional intervention based on epidemiological findings continue to be found today amongst experimental researchers, including those at the Lab. Food is both exposure and an explicit source of intervention on women (Landecker, 2011).

Contemporary moves toward a developmental model of obesity. The cultural and scientific popularity of obesity today is such that the *New York Times* ran a series on the *Science of Fat* in the summer of 2016. In one article, science writer Gina Kolata contextualized obesity research in a popular reality television show, the *Biggest Loser*.[8] Based on the idea that the contestant who loses the most amount of weight on the show wins the competition, Kolata interviewed contestants to see how they were faring nearly six years after having participated on the show. While initial weight loss was truly impressive, what was newsworthy was the fact that so many individuals not only gained back their original weight but had gained even more weight back in spite of continued exercise and calorie restriction. One winning contestant declared, "I won't be victim to this. It's the hand I've been dealt" (Kolata, 2016, para. 8).

[8] This article drew so many comments that the NYT ran an edited piece two days later with some curated comments. It was also the most shared article on the NYT website and Facebook the day it ran (McDermott, 2016).

Like the patient from 1825 quoted above in Dr. Astwood's account of the biological basis of obesity, contestants on the *Biggest Loser* acknowledge that "it's the hand" they have been dealt, and they are subject to the mercy of "Providence." The Providence, or bad luck in all of this, appears to be metabolism and leptin's role in regulating sense of fullness or hunger, as Jeffrey Friedman had earlier shown. Fothergill et al. (2016) studied the *Biggest Loser*'s participants and found that, after significant weight loss, contestants had a lower resting metabolism that was slower relative to someone of equal weight who had not dieted. This, in turn, required contestants to exercise more and eat fewer calories just to maintain their new weight relative to someone who had not dieted. Kevin Hall, an NIH researcher and coauthor of the study mentioned above, also found that leptin levels dropped significantly such that the contestants had an increased urge to eat, supporting accounts of how people who diet disproportionality suffer cravings for food. Contestants of the *Biggest Loser* appear to be "working against their own biology" (Kolata, 2016, para. 15).

The scientific popularity of obesity draws in researchers from a variety of fields (perinatologists, geneticists, epidemiologists, endocrinologists, physiologists) using different methods and datasets (natural and laboratory experiments, retrospective and prospective studies). In order to build on Barker's early work and to hew in like-minded researchers into a more organized field, scholars convened at a global congress and created an academic society known today as the Developmental Origins of Health and Disease (Developmental Origins) in 2003. While obesity or metabolic conditions are not the only adult conditions captured under the umbrella of Developmental Origins, they have played a central part in its conceptualization. Underlying the current paradigm is an evolutionary approach to understand chronic, non-contagious diseases like obesity and Type-2 diabetes. Researchers in this paradigm study

developmental (timing of exposure) effects, programming effects, and the question of persistence across generations.[9]

As is the case in the laboratory I did fieldwork in, beyond studying "immediate homeostatic responses" to uterine life, researchers today seek to understand how "the developing organism may make predictive adaptive response of no immediate advantage but with long-term consequences" across lifespans (Gluckman & Hanson, 2004, p. 311). Developmental Origins seeks to understand the "persistence of such mechanisms [biological processes] in humans who now live in very different environments from those within which they evolved" (p. 311). For example, the "thrifty phenotype" hypothesis proposed by Hales and Barker (1992) posits that under- or malnutrition *in utero* causes the fetus and young infant to be nutritionally thrifty, which confers an advantage if the child grows up in similarly constrained nutritional environment.[10] If, however, the thrifty fetus lives in a world of caloric abundance, she is no longer optimally adapted to the environment because of changes in glucose-insulin metabolism established during fetal life. According to Hales and Barker (2001), "these changes include reduced capacity for

[9] Although horrific famines were inflicted by on millions of Europeans and Russians during World War Two, detailed record keeping by the Dutch during the "Winter Famine" has been particularly relevant to the conceptual growth of fetal programming over the twentieth century. Data from the famine demonstrated the negative effects of a poor maternal nutrition environment (not to mention stress) on miscarriages, birth weights, and adult illnesses of those who survived (Buklijas, 2013). Following the war, British researchers conducted experimental work on animals that mimicked food shortages experienced during the war. Their findings generated the idea of "sensitive" or "critical windows" of development, underscoring the idea of genetic malleability in contrast to the determinism of early 20th century genetics (Burger, Drummond, Sandstead,& Netherlands,1948; McCance & Widdowson, 1974; Ravelli, Stein, & Susser, 1976).

[10] The thrifty phenotype hypothesis, that Hales and Barker proposed in 1992, was in contrast to James Neel's concept of the "thrifty genes," which argued that over time these so-called thrifty genes would have been selected because they provide an evolutionary advantage during times of famine. The timescale increase of obesity and metabolic syndrome, however, cannot reflect a population genetic change. "Consequently, the most accepted model is that obesity and its sequelae are a result of a gene-environment interaction, an ancient genetic selection to deposit fat efficiently that is maladaptive in modern society" (Speakman, 2006, p. 7).

insulin secretion and insulin resistance which, combined with effects of obesity, ageing and physical inactivity, are the most important factors in determining type 2 diabetes" (p. 5). As disease patterns associated with industrialization and urbanization have shifted over the course of the 20th century from contagions to chronic illnesses like diabetes, cardiovascular disease, and cancer, Developmental Origins reflects a historically specific reading of the biosocial landscape in health and disease (Nash, 2006). Developmental Origins thus demonstrates what environmental historians might call an interpretation of a "biosocial landscape" of health and illness (Nash, 2006).

More recently, the thrifty phenotype hypothesis has been supplemented by a hypothesis that allows for more flexibility in the developmental model of disease: the "predictive adaptive response" (Bateson, Gluckman, & Hanson, 2014), which was suggested as a resolution to some contradictory findings in early fetal origins research. An adaptive response conceptually allows for multiple developmental pathways. Specifically, the traditional concerns of the effects that malnutrition or stress in fetal life have over adult health are still mismatched in an obesogenic environment. However, the second pathway of "maternal obesity, gestational diabetes, and infant overfeeding" can be "interpreted as novel exposures" that still induce "long-term effects through alternative mechanisms including the adipogenic [fat making] effect of fetal hyperinsulinemia [excess insulin relative to glucose, a symptom of type-2 diabetes]" (Gluckman, Buklijas, & Hanson, 2016, p. 7).

Obesity researcher Dr. Kevin Hall offers a metaphor of the body that is helpful to understand a proposed adaptive response to obesity. He analogizes the body to a flex-fuel engine:

> The human body is like a 'flex-fuel' car engine: you can throw in diesel one day, regular gas the next day, ethanol the day after…But the body is even more complex, because

> there is no fuel tank—we are built out of fuel, with each cell a miniature engine constantly turning over its components and all functioning surprisingly well despite enormous variations in diet. (National Institutes of Health, n.d, para. 8)

Dr. Hall's metaphor illustrates an early limitation of the Barker Hypothesis, namely that flexibility or plasticity of the body and disease were yet to be incorporated into fetal origins of disease. Hall's imagery of cells working as a "miniature engine*"* suggests both that cells are conceptualized as technologies and that the food we eat is fuel for our technological selves (National Institutes of Health, n.d, para. 8; Landecker, 2011). In fact, Hall's metabolic model of fuel consumption and energy expenditure accounts for diet composition*,* not merely the quantity of calories consumed by a given individual; suggesting a predictive adaptive response to a rich nutrient environment. His version of adipogenesis locates cause not in calories *per se* but in what constitutes the food itself.

Epigenetics and chemical exposures. Another aspect of the scientific landscape that constitutes the daily activities I observed in my fieldwork are the logics and practices of molecular epigenetics, particularly the area of the field that is concerned with exogenous chemical exposure during early life. Below, I explain what a reader should know about epigenetics for this dissertation.

With the mapping of the human genome at the dawn of the 21st century and failure of population level genetic explanations to interpret variations in disease, epigenetics offered a framework for Developmental Origins researchers to study mechanisms of change at the molecular level. Epigenetics, first defined by Conrad Waddington in 1942, is presently defined as "the study of changes in gene function that are mitotically and/or meiotically heritable and that do not entail a change in DNA sequence" (Wu & Morris 2001, p.1103). Waddington argued that "preformation and epigenesis [two views of embryonic development at the time] could be

complementary, with preformation representing the static nature of the gene and epigenesis representing the dynamic nature of gene expression" (as cited in Deans & Maggert, 2015, p. 887-889). In 2012, Waddington suggested that "between genotype and phenotype, and connecting them to each other, there lies a whole complex of developmental processes" which he termed the "epigenotype" (Waddington, 2012, p. 10). From a Developmental Origins perspective, epigenetics helps explain long-term effects of DNA methylation and histone modification resulting from early life exposures to environmental sources of nutrition, stress, pollution, pharmaceuticals, and chemical toxins on adult health outcomes.

Sciences like that of Developmental Origins and epigenetics resonate with contemporary anxieties around the increase in obesity as well as renewed interests in environmental and heritable determinants of health. In early 2015, for instance, the National Academies of Sciences, Engineering, and Medicine's (NASEM) Roundtable on Environmental Health Sciences, Research, and Medicine held a workshop to explore the role that chemical exposure might play in the development of obesity. Committee sessions were organized around the "life span, possible biological pathways and environmental influences, [including the] effects of food additives and antibiotics…on "increased of weight gain, glucose tolerance and insulin sensitivity, inflammation, and aspects of metabolic syndrome in animal models and human studies" (NASEM, 2015, p. 139).

A post-industrial chemical climate points to the concept of an obesogenic environment from which no one is particularly immune. The concept of "obesogen" refers to "chemicals that inappropriately alter lipid homeostasis to promote adipogenesis and lipid accumulation" (Grun & Blumberg, 2009, p. 1128). In non-medical terms, obesogens are "dietary, pharmaceutical, and industrial compounds that may alter metabolic processes and predispose some people to gain

weight," and also high blood pressure and excess cholesterol (Holtcamp, 2012, A63). Much attention is given to bisphenol-A (BPA) because it is the most abundantly produced chemical in the U.S., at around 15 billion pounds per year as of 2013 (vom Saal & Welshons, 2014). It is also an example of an endocrine disrupting chemical (EDC) that interferes with hormone action. As researchers study obesity through the lens of obesogens, they explicitly create a formal link between bodies and environments, tracing "a 'return' of environment and milieu to the center of biomedical theory and research" (Olson, 2010, p. 172-173).

It is within the sciences of Developmental Origins of Health and Disease and epigenetics that I explore how laboratory facts about obesity are produced through imbricated forms of labor specific to contemporary lab life. As described above, the production of new obesity knowledge relies on several forms of labor, or points of articulation, in daily laboratory life. These sources of labor include technicians' anticipatory caretaking practices at the wet bench, the biological labor of biomass in the production of an environmental subject in obesity research, and principal investigators' anticipatory caretaking practices which are circumscribed by the NIH's call for research applications. Because the model of Developmental Origins is situated in theories of causation during uterine life, transformations to the scientific understanding of fetal and maternal life pose unique implications for the renewed surveillance of maternal-fetal exchange. Consequently, Developmental Origins science produces not only new knowledge about obesity but also an environmental biopolitics of reproduction and a medical subject that is environmentally constituted (Ginsburg & Rapp, 1991; Waldby & Cooper, 2008).

Theoretical Framework – The Environmental Biopolitics of Reproduction and Medical Subjecthood

Feminist technoscience studies is "a relentlessly transdisciplinary field of research" that demonstrates ways in which gender "is entangled in natural, medical, and technical sciences"

(Asberg & Lykka, p. 299).[11] As with science and technology studies (STS) more generally, basic science research is seen "as entangled in societal interests…as [are] the technological practices and interventions to which it may give rise" (p. 299). This literature intersects gender and science and is useful to this project in two ways. First, in my fieldwork site, women perform the majority of labor; a fact that is historically and contextually shaped by the connection of Developmental Origins to the field of obstetrics and gynecology, and developmental studies in general. Additionally, this perspective is useful to understand how laboratory work itself is gendered. Laboratory labor performed by technicians (both career and temporary) working in the wet lab is characterized by political and economic precarity, low-pay, low status, and therefore vulnerability; all of which are attributes typically found in so-called women's work (Oksala, 2016).

My ethnographic work was designed and analyzed around the following principles: First, I draw on work in the STS tradition around laboratory fact making with a particular focus on how labor itself is conceptualized, i.e., what constitutes labor in the early 21st century. Whereas previous scholars have clearly delineated social forces involved in the making of facts, few have studied different kinds of labor that go into the social production of experimental laboratory facts. This matters for a broad range of questions around who counts as a scientist, the politics of authorship and, particularly in my study, how the landscape of financial and political precarity enters into experimental designs to shape what is known about obesity in the Developmental Origins framework.

[11] By gender, I refer to cultural attributes assigned to biological (sex) differences between males and females. That said, the category of sex is also socially constructed as illustrated in studies of disorders of sexual development (see Dreger, 1998, and Karkazis, 2008).

Second, building on studies of biopolitics and (bio-)medicalization in social theory and medical sociology, I show how "life itself" (Rose, 2006) and its management under the logics of Developmental Origins and epigenetics shifts away from earlier analytic frameworks based on individual genetics and genomics to one of a biopolitics of the environments of pregnancy, health, and disease. Third, in relation to the interdisciplinary work of sociologists and anthropologists on the politics of reproduction, I show how postgenomic understandings of the causes of obesity are gendered and impact proposed medical interventions on maternal and fetal life.

From each literature, I highlight key "sensitizing concepts" and methodological orientations that help inform the dissertation because they provide an initial way of "seeing, organizing, and understanding experience" at my research sites (Blumer, 1954, p. 7; Charmaz, 2003, p. 259). My dissertation builds on these literatures to argue that material and conceptual biomedical labor specific to Developmental Origins and epigenetics produce new biomedical subjecthood at the intersections of body and environment.

STS: Laboratory labor and scientists in action. In their seminal study *Laboratory Life: The Construction of Scientific Facts*, Bruno Latour and Steve Woolgar (1979) rejected common notions that the work of doing science was somehow outside of human influence and that 'what's really out there' simply waits to be discovered through valid intellectual and empirical design. Rather, they approached the work of doing science as one might approach any workplace, affording no special status to scientists or science. Because of their refusal to accept received wisdom about scientists or science, they were able to demonstrate "how decisions about the credibility of knowledge claims and methods involve a mix of social and technical factors" (Hess, 2001, p. 234-235). Texts, technologies, and animals, for example, all contribute to the

circulation and production of laboratory facts alongside their human counterparts. These "actants" work together in myriad ways to ultimately produce literary inscriptions that may (or may not) become stabilized as facts through "trials of strength,"i.e., the resistance and endurance of a finding in relation to competing hypotheses that can, over time, come to be settled as 'fact' (Latour, 1987, p. 84-89; Callon, 1986; Bennett, 2010).

Latour and Woolgar adopted a position of "anthropological strangeness" in their ethnographic observations of "routine work" at a neuroendocrinology laboratory (1979, p. 27). By focusing on processes of transcription (the work of inscription machines and translation), they traced the so-called facticity stages of a molecule's life as it travels from a state of fiction to non-fiction, or *fact*, within a knowledge network (p. 107-108). Scientific papers, texts typically deemed most free of human influence, are the most social. For example, Michael Lynch (1985) observed a neuroscience lab, capitalizing on unusual findings and disagreements to demonstrate the role of interactional processes on the accomplishment of scientific sense making. In their work on colloid chemistry, Michael Zenzen and Sal Restivo (1982) argued that anomalies or "contingencies (social and otherwise) do not simply affect the course of scientific work…rather they are an integral part of that work" (p. 448-465). Contingencies in the colloid lab, for example, include the researchers' specific background knowledge, the type of equipment available, the degree of funding as abundance or scarcity, social organization of the lab, and the degree of respect among researchers. Variations in these contingencies "form an interlocking structure" that is "inseparable" from "'the research itself'" (p. 465). Such ethnographic accounting of laboratory fact making led to a growth in science studies that emphasized how social forces are at work in the lab without having to appeal to or take sides in epistemological arguments about

'what is really out there'. Science is a cultural practice these authors argued, and literature in this tradition is characterized by…

> the way in which concerns with evidence and consistency were interwoven with situationally contingent events, local decision-making processes, negotiation among a core set of actors in a controversy, the interpretive flexibility of evidence, additions and deletions of rhetorical markers (modalities) to knowledge claims, and other social or nontechnical factors that shape the outcome of what comes to be constituted accepted knowledge and methods in a field. (Hess, 2001, p. 234)

While at my fieldsite, I used these ideas of how local, social, and "nontechnical factors" like political and economic precarity shape experimental questions and therefore "subject natural conditions [e.g., animal physiology] to a "'social overhaul'" that leads to particular epistemic outcomes for obesity knowledge (Knorr-Cetina, 1999, p. 28-29). Such a focus illustrates, in part, what Hannah Landecker (2016) shows in her analysis of how one experimental animal, the agouti mouse, shifted from a model organism of coat color for geneticists to a model organism of adiposity for epigeneticists (and others) because of rising interests in metabolic disorders (p. 92). Epistemic effects were derived from a new situation: although plump agouti mice had existed, it was not until researchers became interested in obesity that the mouse became fat and a model for human disease. In a related example, Nicole Nelson (2013) has shown how anxiety researchers working with a mice model construct, or scaffold, facticity about human disease by "reconfiguring the evidence" to support (or inhibit) claims about a particular human disorder (p.3). Again, new epistemic effects are born out of the work of doing science. Experimental methods, which are often presumed to be immune to outside forces of human interaction, are anything but that. By moving ethnographically into the realms of scientific work, ethnographers of science throw into question perceptions and enactments of methods, rationale, and ultimately scientific epistemology.

Similarly, I approach science as a practice at a site where scientists grapple with understandings as to how cells, tissues, and organs can be permanently programmed in very early life to produce specific adult phenotypes. Like Latour and Woolgar's study of molecule TRF at the Salk Institute, I am interested in "how, where, and why" Developmental Origins facts are created and "how [their] endurance obtains 'in situ'" through scientists' practices (1979, p. 127). Additionally, I consider the larger effects of economic and political precarity on the Lab's division of labor and how this affects the course and content of knowledge production.

Although Latour and Woolgar describe the laboratory workforce as consisting of doctors or scientists who occupy one space in the lab and technicians and secretaries who occupy another, little attention is paid to the everyday work of technicians. Throughout their study, which includes attention to the microprocessing of facts, macro controversies and settlements in scientific fields as well as cycles of credibility, the focus is almost exclusively on doctors. For this reason, *Laboratory Life* reflects a "studying up" of how facts are made (Nader, 1972). While "everybody is made to give a hand" in a working lab, if we want to understand how "reality" is the consequence of debate rather than its cause in Developmental Origins of obesity, we ought to follow the minutiae of debates, the everyday handling and tinkering of technicians, and the way their own positioning in the field enters into experimental questions (Latour, 1987, p. 182).

In this dissertation, rather than asking how the mouse physiology translates into an inscription on paper and how doctors build trials of strength, I detail how the technicians' work of actually handling the animal translates into the finished knowledge product. My question, therefore, intervenes in an earlier moment in the process of translating and inscribing and necessarily focuses on the forms of labor technicians employ at the bench. Unlike the neuroendocrinology lab of *Laboratory Life*, scientists at the Lab are not physically present

alongside the technicians, and are today quite removed from the everyday work of basic science research. Principal investigators may be better understood as managers, authors, and salesmen; a point Latour and Woolgar acknowledge in a description of their lab's "group structure," but which has become heightened to the extent that today investigators are largely removed from the production of raw data (1979, pp. 221-223).

Laboratory labor is a key element of this dissertation. I deploy Latour's (1987) rules and methods in *Science in Action* as a sensitizing orientation to detail forms of labor in a contemporary biomedical laboratory. Latour writes that to understand technoscience ("all elements tied to the scientific contents"), all actants and resources need accounting for (p. 174). By studying the "*activity* of making science," then we can make the precarious "recruitment drive" visible (p. 174). What I add to this picture has emerged since Latour wrote these classic works: I detail the role of a precarious workforce—an imported, highly skilled technical workforce—who produces virtually all the raw data on which principal investigators depend.

The war on obesity is, as I have explained above, a key contextual element to the detailed observations of one location that I pursued. Latour's description of technoscience as a "military affair" thus informs my analysis of how funding impacts research agendas, support for laboratory research, and by extension lab workers' careers (p. 171). War and health, he argued, are the two research areas where scientists have been able to pursue technoscientific studies because both the "body politic" and the "survival of the body [are] subject[s] in which everyone is directly and vitally interested" (p. 172). For war and health, he writes, "money is no object…[T]he health budget, like that of defence, is a gigantic treasure chest where spending is made without limit" (p.172).

Competition for the treasure chest is fiercer today. An important trend in basic science research has been towards soft-money funding; meaning scientists have to raise their own money through competitive grants in order to run an independent lab.[12] A combination of the large presence of scientists in obesity research with the hard realities of soft-money funding impacts the way scientists pursue ideas (more risk taken with novel approaches), develop new techniques (to provide causal rather than descriptive narratives), design experiments (mice are cheaper models than rats), fund additional laboratory personnel (use of voluntary workers vs. paid technicians), purchase equipment (buying more technically sophisticated visualizing tools), and write for publication (more stringent definitions of intellectual integrity for publications vs. grant applications, grant-writing cycles, and choice in journals). That is, not only are facts produced for a market in obesity research, but the Lab also reproduces that very market and, in doing so, creates a particular maternal-fetal "regime of perceptibility" (Murphy, 2006) in the production of *environmental biomedical subjecthood*.

In order to be competitive, lab researchers must argue the novelty and feasibility of their work while also claiming its translational potential for human subjects in terms of clinical and/or pharmacological interventions. These claims of scientific promise and interventions around obesity help shape and give rise to the possibilities of new biomedical realities. Indeed, Latour (1983) argued that "[m]icrobiology laboratories are one of the few places where the very

[12] Soft-money is money that scientists have to be apply for, as with grants, and is usually offered in 1-5 year commitments. At University Lab, the PIs live and die by grants. Recently, a junior PI was asked to withhold her findings and name from grant applications because it was feared any association with her would jeopardize the "group's" chance of success. In other words, she was asked to forestall progress in her own career for the sake of the group's financial wellbeing. Without soft-money funding, this lab cannot even claim office or space to carry out its research. The hard realities of soft-money funding at the Lab appears to be typical of the precariousness of laboratory work across the country (see Barinaga, 2000).

composition of the social context has been metamorphosed," and it is precisely where biomedicine gains its legitimacy and, therefore, power to govern (p. 158).

Biopolitics. Because governance and disease are at the heart of my analysis, I must also briefly touch on the scholarly literature around biopolitics and (bio-)medicalization. Critical inquiry into relations between the biological and the social has run the course of the twentieth-century, but it was the philosopher and historian Michel Foucault's (1978) analysis of power that provides, through the concept of "biopolitics," ways to analyze emergent relations between the body, polity, and the modern state. Foucault (2003) argued that governance in a modern state is indirect since people follow norms of self- regulation. The modern state invokes its right "to make live" or to foster life via the health of the population (p. 241). Having co-evolved with neo-liberal forms of social regulation with an emphasis on individual responsibility, biopolitics, according to what Thomas Lemke (a scholar of Foucault) said in an interview, reveals itself "when [a] population emerges as an object for politics, as something that can be transformed, that can be optimised, on which one can intervene, [and] which can be used to achieve certain ends" in varying ways (Baele, 2008, para. 10). According to the anthropologist Paul Rabinow and the sociologist Nikolas Rose (2006), biopolitics extends further to encompass "all [of] the specific strategies and contestations over problematizations of collective human vitality, morbidity and mortality" (pp.196-197). Rose (2007) and Lemke (2011) agree with that point of view. It is in the laboratory where health is problematized and strategies of intervention on the population are most acutely realized. Sarah Franklin (2006) argues that,

> The extent to which biomedical knowledge, biotechniques and biology 'itself' reshape each other, and co-evolve, may never before have been made so explicit as today, through the redesign of the biological in the context of bioscience, biomedicine and biotechnology. The rebuilding of 'life itself' in the laboratory is redefining the future of the environment, food, wealth and health and is consequently crucial to definitions of progress, social justice and power. (p. 168)

In the case of the environmental problematizing of obesity through women's lives, I am interested in how the practices and power effects of a self-evident response by medical scientists to the obesity epidemic *naturalizes* new definitions of environmental causes of obesity and interventions on women's bodies.

Biopolitics, then, acts as a further sensitizing concept for my dissertation. It helps provide ways to question not only what the geneticist and gender scholar Anne Fausto-Sterling (2000) calls the "social nature of physiological functioning" in making 'gender' differences a somatic fact, but what I find, in the case of environmental epigenetic models of health and illness, is the physiological functioning of social nature (p. 235). In other words, our physiology is reconfigured by the social nature of industrial exposures. Drawing on Foucault (1978) and Haraway (1990), Fausto-Sterling argues that "our bodily experiences are [discursively and materially] brought into being by our development in particular cultures and historical periods" (p. 20). In the case under study in this dissertation, the bodily experiences of pregnancy and adiposity are being brought into being in an era of harmful environmental exposures. At my fieldsite, within the context of obesity and metabolic syndrome, human vitality has been problematized where indeed the vitality of the population, including the unborn, is said to be a stake; consequently, maternal and fetal bodies have been enrolled in new political-economic arrangements of scientific funding and fact making.

In the framework of biopolitics, the management of "life itself" rests on self-regulation, knowledge of risk, and medical intervention (Rose, 2006). For example, in her gene-environment study of lead regulation, scientific work, and children's exposure, Sara Shostak (2013) demonstrates the cultural logic of self-regulation institutionalized within commerce, science, and regulatory agencies. She concludes that these logics constitute a biopolitical regime of scientific

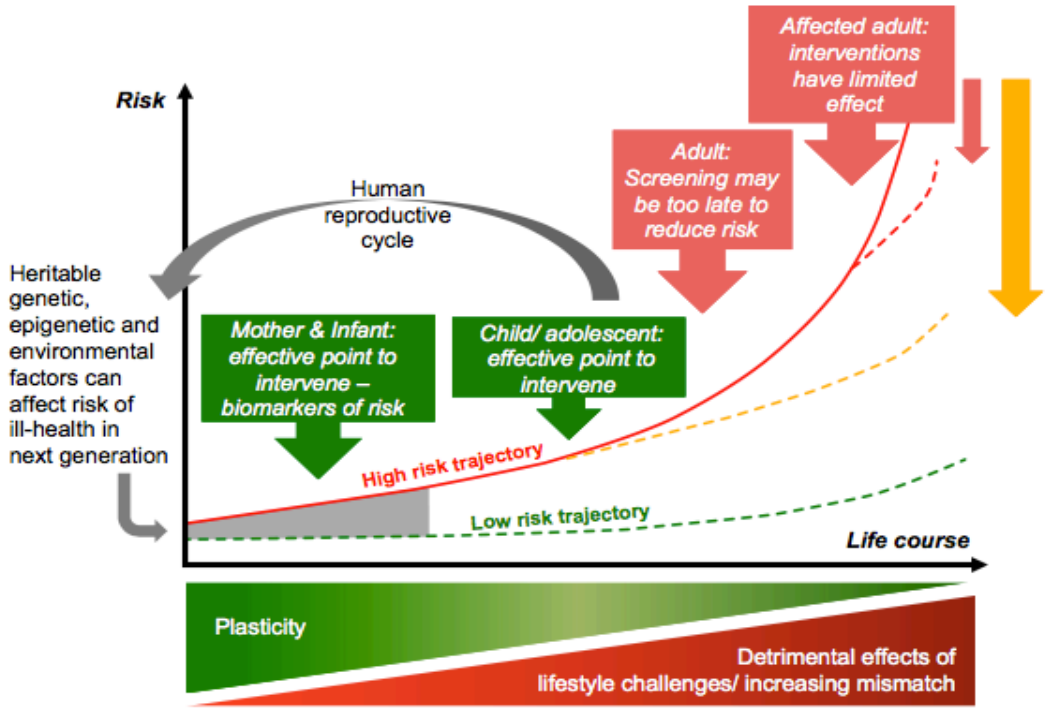
knowledge and political regulation. In the case of spina bifida, Salim Al-Gailani (2014) argues that "political concern" with the disorder in the United Kingdom in the 1970s "stimulated" medical research into the efficacy of folic acid (p. 286). Terming this historical outcome as a "politics of risk reduction," Al-Gailiani argues that the clinical rationale for folic acid supplementation shifted in the post WWII years from concern with women's risk for anemia and problems of poverty and malnutrition to preventative, self-administered measures taken to protect the unborn fetus from birth defects. Likewise, maternal nutrition in David Barker's (1995) initial fetal origins research was proposed to help the growing fetus in particular, not the mother.

My reading of the scientific literature produced by Developmental Origins researchers is that the legitimation of investigators' work explicitly rests on intervention, and I saw this in the daily work of the Lab. Translational researchers must sufficiently demonstrate, or persuade, how experimental animal findings fit human models of disease and, therefore, propose targeted models of intervention. Moreover, nutritional intervention is framed in terms of human capital because poor health in early life reduces "life chances," perpetuating social inequalities across generations (Almond & Currie, 2011, p. 2). It is in this way that Hannah Landecker and Aaron Panofsky (2013) link traditional concerns of many sociologists, namely around intergenerational transmission of social inequalities, with epigenetics to show how the social (exposures to poor nutrition, for example) becomes "biologically meaningful" (p. 335). In that sense, ethnographic work at the Lab details how intervention is conceptualized and materially acted on.

The logic of Developmental Origins and epigenetics situates intervention in new temporal and spatial arrangements, shifting care of the self to include offspring (during pregnancy or even before conception as an imagined maternal future). Risk is managed prenatally or

periconceptionally and viewed as mothering work in the form of healthful nutrition, weight management, and the avoidance (if possible, but usually not) of toxic exposures (Lappé, 2014, 2016). For example, a typical representation of "effective" temporal interventions in Developmental Origins research indicates the need for early intervention in order to positively impact later-life health.

Figure 1.3. Proposed Timing of Medical Interventions



Note: Taken from Hanson & Gluckman (2014, p.1032).

New scientific models of inheritance and medical models of intervention as shown above problematize the classic analytics of medicalization and biomedicalization, to which I now turn to below.

Biomedicalization. Self-regulation and individual responsibility are central elements of biomedicalization; a framework Clarke, Shim, Mamo, Fosket, and Fishman (2003) proposed as

successor to theories of medicalization which were first articulated by Irving Zola (1972, 1991) as well as Peter Conrad and Joseph Schneider (1992). In a more recent iteration of biomedicalization, Clarke, Mamo, Fishman, and Shim (2010) argue that theories of medicalization focus on processes that exert "*control* over" social phenomena that had once lay outside the scope of medicine's domain, like premenstrual syndrome or alcoholism (p. 2). Through a process of medicalization, human conditions or behaviors defined as troublesome are transformed into medical illness, which are then subject to clinical intervention and regulation. Building on this perspective and to account for late 20th century changes in "technoscientific" practices around the management of life, Clarke et al.(2010) define biomedicalization practices as "*transformations of*" the very same types of phenomena, including "diseases, illnesses, injuries, and bodily malfunctions" (p. 2). Whereas medicalization emphasizes an overt control of a condition or state of being defined as pathological, biomedicalization views medical science and biotechnologies as "hidden instruments of social control so that a rational and self-disciplining governance permeates society at various levels" (Riska, 2010, p. 155). Aspects of each framework help organize findings on the governance of obesity.

In relation to processes of medicalization, I analyze how Developmental Origins and epigenetics research shifts definitions of obesity since its logic moves the very perimeters (and visibility) of where conditions are theorized in body-environment interactions. Epigenetics complicates the locality of disease and therefore its control because of its potential heritability. From biomedicalization, a focus on *transformation* of biological phenomena appears legible in Developmental Origins and epigenetics, but the model presumes geneticization. Even though a focus on health risk and surveillance involves a reconceptualization of bodily matter (from gene

expression to organ function in both frameworks), medicalization and biomedicalization do not prioritize the environmental and historical contexts of bodies.

The spatial and temporal specificities of postgenomic sciences such as epigenetics and Developmental Origins stand in contrast to a more homeostatic reading of the body, disease, and control over it in the classic models of medical management or current "invisible" sources of medical power. Both lack nuances of the effects of shifting temporal and spatial dimensions in their founding assumptions. Postgenomic sciences embrace a theory of the body deeply embedded within and constitutive of environments. If bodies and environments (to put in unfortunate dualistic terms) have plasticity and are always imbricated with one another, the questions I raise in this literature are what or who is being medicalized through environmental re-definitions of obesity and therefore its management? Who or what is being "transformed," in the language of biomedicalization, through the logics of environmental epigenetics and Developmental Origins of disease? How do processes of medicalization and biomedicalization work in an era of postgenomic science in which a hybrid of programming in Developmental Origins and epigenomic flexibility inform maternal-fetal obesity research? To reiterate Bruno Latour, the body politic depends not only on the survival of the body, but the reproduction of it. The inherent reproductive politics of Developmental Origins and epigenetics offer a reorientation to body-environment interactions that have eclipsed the reign of twentieth century genomic sciences. With this change in the conditions of how diseases, causation, and transmission are framed, I explore how the concepts of medicalization and biomedicalization both necessarily require conceptual refinement.

Drawing it all together: An environmental biopolitics of reproduction. In this section, I incorporate literature on politics of reproduction to illustrate discursive and material

shifts in meaning and practices in the postgenomic era. Philosophical, religious, juridical, and medical ideas of inheritance, from family guilt to intelligence, have a long standing in the history of ideas, religion, and science (Müller-Wille & Rheinberger, 2012). Today, we live in an era of scientific articulation about the relevance of "maternal impressions" for the future health of unborn children; ideas perhaps not as intensely pursued since the eighteenth century (Paul, 2010, p. 145). "[E]veryone [remains] directly and vitally interested" today in the objects of knowledge produced through the lens of postgenomic sciences; that interest is distinctly situated in maternal and fetal bodies in the context of environmental obesity research (Latour, 1987, p. 172).

The anthropologists Faye Ginsburg and Rayna Rapp (1991) contend that a politics of reproduction entails the examination of "multiple levels on which reproductive practices, policies, and politics so often depend" (p. 313). Building on their concept, I detail throughout this dissertation how a gender politics inheres in Developmental Origins and epigenetics; how it stems from scientific discourse and practices of Developmental Origins experimental work at the local level and rises up through national and global policies found in American and international obesity and endocrine medical professions as well as in US public health and environmental agencies such as the National Institutes of Health. These levels of analyses reveal the "many ways that power is both structured and enacted in everyday activities" through the creation of an environmental subjecthood for reproductive women (Ginsburg & Rapp, 1991, p. 312).

In her later work on the impact of amniocentesis on expectant women, Rapp (1999) studied the work of a laboratory to understand how diagnostic fact is produced. Her analysis demonstrated how the maternal body, through processes of scientific decision-making, evidences culturally embedded truth(s) around motherhood, dis/ability, and potentiality in future life. Two decades later, in her work on the science of epigenetics, the historian of science Sarah

Richardson (2015) builds on Ginsburg and Rapp's line of inquiry to argue that "[e]pigenetics research situates the maternal body as a central site of epigenetic programming and transmission, and as a significant locus of medical and public health intervention" (p. 210). For Richardson, the maternal body is nothing less than an "an epigenetic vector" of risk (p. 211). I focus on the maternal line because the work of University Lab is situated in the maternal-fetal environment (Curley, Mashoodh, & Champagne, 2011; Day, Savani, Krempley, Nguyen, & Kitlinska, 2016).

My conceptual framework also draws inspiration from the ethnographic work of the medical anthropologist Valerie Olson. Through her 2010 study of astronauts and space biomedicine, Olson argues that "space biomedicine [is] a form of environmental medicine that seeks to optimize and manage technically enabled human ecologies where life and environment are dually problematized" such that "space biomedical subjecthood is fundamentally environmental rather than biological" (p. 171). Space medicine may initially appear to have nothing in common with experimental laboratory work, but I argue (as did Latour) that the work done at the Lab is itself an instantiation of an extreme environment epistemologically organized to mirror life outside its walls. Developmental Origins "is engaged with knowing and intervening in life-environment interactions in spaces where environment, whether conceived of as [food] or as a [womb], cannot be bracketed out from life itself" (Olson, 2010, p. 171). And as with space biomedicine, obesity and environmental exposures are "dually problematized" in Developmental Origins and epigenetics (p.171). Not only are women fundamentally environmental, rather than biological, the logic of postgenomic science suggests that we are all environmental.

Finally, Olson's move provokes a reconsideration of Michel Foucault's (1978, 1997) concept of biopolitics and Nikolas Rose's later extension of "how modern forms of normality, health, and sickness come about as power and knowledge are invested in the basic processes of

'life itself'" (Olson, 2010, p. 171). Olson's conceptual refinement "ecobiopolitics" is defined as follows: "truth claims based on knowledge of milieu processes, power relations that take milieus as their object, and the modes of subjecthood and subjectification that designate subjects as milieu elements" (p. 181). She indicates that a focus on "milieu" reflects a postgenomic era in which the focus is not on life per se but on "milieu as the site of power-laden interventions" (p. 181). Similarly, the work of Developmental Origins and epigenetics into environmental causes of obesity reflects biomedical focus that entangles milieu because it does not analytically bracket off a self-contained body from its habitats.

In sum, I draw on Ginsburg and Rapp's sensitizing concept of politics of reproduction, Olson's ecobiopolitics, and the larger frameworks of medicalization and biomedicalization to analyze Developmental Origins and the environmental epigenetic model of obesity as situated in reproductive, female bodies. Bringing together reproduction, bodies, biomedicine, health and disease, social disparities, health risks, governance, and environments, I propose that we face an *environmental biopolitics of reproduction* in light of what might be called *exposomic medicalization*. This framing allows us to examine the milieu of power-laden interests that inhere in scientific fact making and proposed interventions on environmentally constituted reproductive bodies. At stake is scientific knowledge production, the extension of medicalization outward into the environment and across lifespans and its effects on the population, the effects of a gendered science such as Developmental Origins on reproductive women in particular, shifts in disease classifications, potential pharmacological interventions, cultural ideas of care and responsibility, the reframing of public health policies in environmental terms, shifts in national research funding priorities, and, perhaps optimistically, the possibility for better environmental regulation (Ginsburg & Rapp, 1991; Canguilhem, 2001; Landecker, 2007, 2010).

Methodology

This project received IRB approval in January, 2015, IRB#15-000006. I was also approved through the Lab's home institution to act as a Non-Compensated Employee beginning January 2015 through 2016.

In order to investigate how and under what conditions environmental obesity facts are produced in a "costly ghastly" laboratory (Latour, 1992), I follow the methodological orientation of "anthropological strangeness" (i.e., a naïve observer) of Latour and Woolgar's *Laboratory Life* as well as George Marcus' (1995) principles of multi-sited ethnography and the principles of grounded theory (described below) to develop the ideas of an *environmental biopolitics of reproduction* and environmental, medical subjecthood *de novo*.

Objects of study are informed by different modes or techniques that allow us to follow association or connection among multiple actants across time and space (Marcus, 1995). In particular, I draw on Marcus' framework of following people and things (including animals) involved in the work of producing laboratory facts about obesity. He describes a multi-sited ethnography as,

> designed around chains, paths, threads, conjunctions, or juxtapositions of locations in which the ethnographer establishes some form of literal, physical presence, with an explicit, posited logic of association or connection among sites that in fact defines the argument of the ethnography. (Marcus, 1995, p. 105)

I disaggregate these chains, or points of articulation as I have described above, into chapters. In chapter 2, I analyze the work done by technicians in the production of raw data for scientific claims about the environmental and developmental causes of obesity. In chapter 3, I follow the biological labor of model animals made to function differently in the creation of human futures defined by environmental exposures, and in chapter 4, I track the larger effects of a political

economy of research funding for PIs on the production of translational obesity knowledge and the constitution of a contemporary basic science workforce at the Lab.

I also follow the "metaphor" in that epigenetics represents a mode of thought about genetic flexibility or plasticity which complements the concept of precarity discussed earlier in this chapter (Marcus, 1995, p. 108). Like the notion of flexibility, precarity similarly entails malleability but it hinges on uncertainty and questions of risk, and this seems to me to be an underlying essence of both the science followed here as well as those who practice it. Precarity inheres in the material work of the lab in regard to funding as well as its biomedical workforce.

As an ethnographer, I play the role of "anthropological stranger" and follow people, things, and metaphors while adhering to the complementary orientation of grounded theory with its emphasis on an inductive approach to collecting data and developing concepts *de novo* (Strauss, 1987; Glaser & Strauss, 1967; Strauss & Corbin, 1994). Building on the tenets of symbolic interactionism, grounded theory places importance on inductively understanding how social processes are organized and objects given meaning (Tavory & Timmermans, 2009, p. 256). The role of the ethnographer is to gather and generate data from the field, portraying the world *in situ* as lived and understood by its members (Blumer, 1986; Becker 1998). Additionally, I find Adele Clarke's (2005) notion of situational analysis as a refinement of grounded theory; productive for situating the Lab within a larger network of elements, including human and non-human actants, the material and discursive work of Developmental Origins and epigenetic stakeholders, and the relations of money to science and back again. In this light, I agree with Clarke when she says "everything in the situation *both constitutes and affects* most everything else in the situation in some way(s)" (p. 72). By following actants at the Lab, I track how meanings are assigned to objects in the field, and how knowledge about the Developmental

Origins of disease is historically and socially situated. Together, the points of articulation link the strategic relations of scientific work in a 21st century laboratory to wider social practices and values about health and disease, specifically a turn to the environmental problematizing of obesity (Landecker, 2007, p. 222-224).

Standpoint and positionality. The immersion process at the Lab was a welcome one from first contact. In response to my inquiry and an initial meeting, the head principal investigator generously welcomed me to meet his colleagues and staff members. Some technicians working at the bench and one junior investigator were suspicious and wondered what was so interesting about their work. While there was some initial caution on their part, after a few visits our rapport became friendly and inviting. For example, I was invited out for coffee chats and to observe for myself the culturing of cells as they appear under magnification. These are but two examples of how I was allowed into informants' worlds. As my research progressed, I spent the majority of my time at the wet bench with women who have also received tertiary degrees. Except for the most senior principal investigator who was also a clinician with professorial and editorial roles, the other investigators and technicians all happened to be women who had all immigrated, except one, to the United States to work as laboratory scientists or to become medical doctors.

As observers in fieldwork we are always "on our own side," i.e., biased by our own interests, and, as a consequence, relations of domination "render our knowledge partial" (Burawoy, 1998, p. 23; Haraway, 1988). Demographic similarities to the technicians, particularly around education, may have subconsciously influenced the type of data I collected, from explanations about what scientist-technicians are doing at the bench, to invitations to peer into their work, to rich autobiographical detail offered up as to how they landed in this Lab and why

they stay. When it was appropriate, i.e., not in the middle of technical tasks that required intense concentration, I was able to inquire about a multitude of topics, including careers, immigrant visa status, employment conditions, how they view and conduct basic science research today, and questions about funding and politics of authorship. As detailed in Chapters 2 and 4, I supplemented personal narratives and observations made at the Lab with demographic data gathered from the National Institutes of Health, National Academies of Science, and the National Resource Council on today's basic science workforce in biomedicine.

Data collection. My sources of data collection were participant observation, interviews, and textual analyses of scientific papers and popular press media. The vast majority of my fieldwork was spent at the Lab. As Marcus (1995) notes, a "strategically situated (single-site) ethnography" may "not move around literally but may nonetheless embed itself in a multi-sited context" because "what goes on within a particular locale in which research is conducted is often calibrated with its implication for what goes on in another related locale or locales" (p. 110). The Lab itself was multi-sited in the meaning Marcus ascribes to it. For example, there were more than four physical spaces in which Lab members work on a day-to-day basis, and their work was always directed outward toward happenings in other locales. I followed the "circulation of cultural meanings, objects, and identities in diffuse time [and] space" as they relate to the Developmental Origins, epigenetics and the work of doing technoscience (Marcus, 1998, p. 79). In addition to fieldwork, I gathered data from structured and unstructured interviews with participants over the course of my time at the Lab. I supplemented fieldwork and interviews with analyses of textual materials gathered from scientific and popular presses.

Fieldwork. I engaged in participant observation in several settings while at the Lab, including the wet bench (wet lab), vivarium (animal room), photographic dark room,

technicians' offices, and weekly meetings which were held in one senior principal investigator's office. Fieldwork in each of these settings cumulatively totaled approximately ten hours of fieldwork each week for one year.

Lab meetings included all staff members. In this space, investigators led meetings by making inquiries of the technicians-scientists about the prior week's work. The latter reported on results of their work, troubles encountered, and each person asked questions about how to proceed. Everyone brainstormed ways to move forward from the most seemingly banal issues like dry ice shortages and ordering brain atlases, to creating unique experimental designs to see if something works. Grant deadlines and the timing of animal husbandry organized much of the decision making around experimental work. During the weekly lab meetings, I was often addressed by PIs so that they could clarify what it was they were studying and why, or to explain how it helped to move concepts forward or to explore new areas of interest.

The wet lab was situated in a neighboring building adjacent to PIs' offices. I spent two or three days a week in the wet lab shadowing technicians as they organized, studied, prepared for, carried out and analyzed experimental results. I shadowed four main groups of people in the wet lab and animal room: those in charge of fat tissues, those responsible for brain and stem cell experiments, the bisphenol-A model, and, lastly, the animal care-taker who was also responsible for the sacrifices (killings) and performing phenotype work like measuring weight, blood pressure, glucose levels, and other indicators of metabolic health. There was often cross talk in the Lab so I asked follow-up questions about how the work went on days I was absent or when I needed clarification. I was frequently shown data on computer software and developed films to "see" results of testing and experimentation that the technicians obtained.

As conferences are part of academic life, the making of science, and therefore part of the Lab's circulation, I attended conferences as they arose over the year. I attended local conferences at a nearby university, including, in 2012, "Plastic Bodies: Biopolitical Implications of the New Environmental Epigenetics," as well professional scientific talks such as the annual Society for Reproductive Investigation meeting in March, 2015, and the Obesity Society's annual weeklong meeting in November 2015. At professional conferences, PIs offered to have me alongside some of their review work, e.g., evaluating posters. This provided a more detailed landscape of how the Lab travels in maternal-environmental studies of obesity and metabolic syndrome.

Interviews. I held both structured and unstructured interviews with all of the Lab participants. Formal interviews with each PI were recorded with permission and coded following grounded theory methods. Structured interviews typically lasted two hours, and unstructured or open-ended questions with all Lab members occurred regularly in the course of daily fieldwork. I also had the opportunity to interview editors affiliated with journals that incorporate Developmental Origins and epigenetic perspectives.

Textual analysis of literature. I tracked literature in science and the popular press to follow the scientific metaphors of flexibility, precarity, and programming in Developmental Origins and epigenetic sciences. In order to follow the science, I used several databases including PubMed, Web of Science, ProQuest, JSTOR, and Social Science Citation Index to identify relevant medical journals and articles from the fields of Developmental Origins of Health and Disease, epigenetics, endocrinology, nutritional epigenetics, and epigenetics more broadly. Typical peer-review scientific publications include: *Nature*, *Cell*, *Clinical Epigenetics*, *Environmental Epigenetics*, *New England Journal of Medicine*, and the *Journal of Developmental Origins*. I used search words and combinations of words that included: obesity,

metabolic syndrome, environment and obesity, developmental programming, developmental origins of obesity, epigenetics of obesity, obesogens, nutritional epigenetics, and animal obesity to identify relevant scientific literature.

I also searched government databases about topics of environmental obesity, focusing on the search words mentioned, and federal databases on science grants and the biomedical workforce. Further, I targeted sites of professional organizations such as the Endocrine Society, the World Obesity Foundation, and the International Society for Developmental Origins of Health and Disease as well as public health sites that focus on obesity. In lieu of traditional brick and mortar classes, I also took advantage of podcasts available through the University of Oxford to learn more about the sciences of obesity. In addition to search words listed above, I included biopolitics, sociology, and anthropology since the seminars were interdisciplinary and housed in an educational center. Finally, I tracked mass media sources to identify how obesity was being positioned and by whom for a lay audience. Scientific, government, university, and popular press resources helped to identify actors and stakeholders in the field while mapping out cultural and scientific understandings offered on the putative environmental causes of obesity and attendant diseases.

Data analysis. In order to analyze the points of articulation above, my approach and techniques were based in principles of grounded theory, techniques of multi-sited ethnography, and situational analysis. I used tools associated with an inductive approach, including coding, diagraming, memo writing, and comparisons in order to inductively generate concepts, hypotheses, and theories.

Drawing on the orientations and tools provided by these three articulations of doing ethnography, I collected, transcribed, and coded all personal communication based on

observations, interviews, and textual analyses into electronic form, and considered the ways in which people and things were made to work. By following people and things and considering the conditions by which things happened, the concepts of anticipatory care-work, precarity, maternal burden, and environmental subjecthood emerged out of the data. Consequently, they formed the conceptual apparatus of the dissertation: technicians, economic and political precarity, and the epistemic effects of one's status as a career or temporary technician on knowledge production (chapter 2); biomass at work in the production of environmental subjecthood (chapter 3); and the political and economic climate of scientific funding and its iterative effects for PIs on the production of maternal-environmental obesity knowledge (chapter 4).

Overview of the Dissertation

In chapter 2, I follow technicians who work at the Lab to produce new knowledge about the epigenetics of pregnancy and the development of obesity and metabolic syndrome in experimental animal models. While the material labor of producing facts is central to this chapter, I was surprised to find people other than PIs circulating at the bench. This raises a question about the economy of bench life. I found that highly trained immigrant scientists work as so-called technicians and that these observations reveal a unique kind of scientific pipeline that consists of immigrant scientists who hold doctorate and medical degrees. These technicians either work as *career technicians* or as *temporary technicians* in this Lab, and elsewhere, while studying for US medical board exams. Because of their different temporal orientations in the Lab, I argue that they develop different caretaking practices with respect to the Lab's experimental systems. This, in turn, affects the course and content of laboratory fact making.

In chapter 3, I trace the labor of biomass, i.e., animals and their constituent parts. By focusing on biomass, I explore what it means to be environmentally constituted in an extreme

context of creating molecular based, epigenetic findings of obesity and metabolic syndrome. A focus on nutritional and chemical exposures and their effects on phenotypic outcomes allow us to see the ways in which environmental forces are biologized in the framework of Developmental Origins. This, in turn, lets us ask how obesity research, in particular Developmental Origins, shifts what the medicalization of fat or body shape and size means in an epigenetic era. How does medical surveillance work when individual patients stand before doctors as the consequences of evolution rather than consequences of their lifestyles? What are the changing conditions of medicalization as the explanations of causation shift with epigenetics, as social things become seen as biologically causative in ways that they were not before, when the mode of proof rests on triangulation between epidemiology and laboratory work? In answering these questions, I propose the idea of a new environmental biomedical subjecthood that implicates gene-environment interaction studies in general.

In chapter 4, I focus on principal investigators, and the political economy of basic science obesity research. In it, I follow the national public health metaphor that defines obesity as a national biomedical security threat. Highly prized grants from the National Institutes of Health in particular increasingly demand molecular explanations of obesity with translational potential for human health. I argue that precarity of funding shapes the biomedical workforce as much as it shapes research agendas. Lab work is organized temporally where daily work is oriented toward grant money already received and for which findings must be produced while simultaneously oriented toward the next round of grants and their deadlines. A precarious and intensely competitive funding landscape has multiple effects on the production of obesity knowledge. As a result, I am interested in how the problematic of obesity itself, and the effects of funding, shape how and by whom knowledge production works in an experimental animal laboratory.

Finally, in the conclusion, I review the main arguments of each chapter and the study's limitations and contributions to literature across the fields of sociology, anthropology, science, and technology studies with a particular bent toward feminist studies in technoscience literature on science and the bodies. By ethnographically following people, things, and metaphors, I found that the work of Developmental Origins and epigenetics of obesity produces new conceptual and material relationships in maternal-fetal health. A new environmental biomedical subject emerges out these sciences where, in the light of inheritance, obesity is differently medicalized as maternal-fetal and, because they change the very perimeters of where the condition is said to be, theories of medicalization and biomedicalization should similarly shift to accommodate new causal theories and intergenerational models of disease transmission in a post-genomic era.

Chapter 2

Laboratory Technicians and Anticipatory Caretaking Practices

> My last but most important slide is my acknowledgment slide. Clearly, I am just a spokesperson for a whole lab of very talented students and post-docs whose hard work and enthusiasm give me all the data I've got to share with you today.
> Anonymous, personal communication, SRI conference, 2015.

> To ignore the technician's contribution is…to act as if scientific knowing begins only after scientists have come on stage and to perpetuate the sort of cultural myth that modern sociologists of science wish to transcend.
> Stephen R. Barley and Beth A. Bechky, 1994, p. 86.

In the spring of 2015, I attended the annual Society for Reproductive Investigation Conference in San Francisco, California. After each presentation, I noted that speakers graciously acknowledged the work of many others who, according to one principal investigator (PI), produced "all the data" necessary for her talk. As I sat in the darkened conference hall and reflected on photographs of lab personnel from one presentation to the next, I wondered why the division of laboratory labor is organized today such that PIs are "just a spokesperson" on behalf of others' labor (the main focus of chapter 4). This initial observation provoked additional questions about the organization of laboratory labor at University Lab (Lab) regarding how PIs' dependence on technicians affects the course and content of knowledge production, as well as the reproduction of the Lab itself in the knowledge market for environmental models of obesity and metabolic syndrome.

In this chapter, I foreground the work of technicians, those individuals "whose hard work and enthusiasm" produce "all the data" necessary to help run a laboratory. I do so for three reasons. First, my observations of the Lab's division of labor are not idiosyncratic. They mirror the National Institutes of Health's (NIH, 2012) description of a typical, academic, biomedical

lab:

> [It] consists of a PI and one or a small number of permanent technical staff, with the majority of the research, carried out by trainees…Today these scientists bring stability to many labs and provide important functions as part of institutional core facilities, but have a wide variety of titles and employment conditions. (NIH, 2012, p. 10)

Further, the National Academies of Sciences, Engineering, and Medicine (2015) has found that that "the availability of research funding drives not only the specific research questions investigated, but also the scientific workforce available to carry out that research" (p. 35). Ethnographic and national data suggest that contemporary laboratory life is organized by a division of labor in which permanent technical staff and itinerant student technicians, who have a wide variety of titles and employment conditions, are the primary producers of raw empirical data in the early 21st century.

My second reason for focusing on technicians is because they represent a global "reserve army" of highly educated scientists (Engels, 1845). Foreign talent fills the contemporary, biomedical lab. Immigrant scientists at my research site and elsewhere help backfill the absence of PIs at the bench. According to a 2005 report by the National Research Council about opportunities and challenges to foster the independence of young investigators in the life sciences, "more than half of the biomedical postdoctoral researchers in this country hold non-U.S. citizenship. It is difficult to consider the U.S. biomedical research enterprise without acknowledging the critical role played by scientists from outside the U.S" (p. 6; NIH, 2012).

While I do not focus on post-doctorates, the division of labor at the Lab similarly relies on an international, scientifically trained workforce. Relative to post-doctorates, however, far less is known about "staff scientists," in particular those who are immigrant scientists and labor alongside native student trainees at the empirical interface of conceptual and material work (Barley & Bechky, 1994, p. 98). Based on NIH (2102) estimates, the number of staff scientists in

academia has grown from approximately 4,000 in 1993 to 8,500 in 2008. In recognition of staff scientists' contributions to biomedical research, the NIH has called for its own "study sections to be receptive to grant applications that include staff scientists and urges institutions to creation position categories that reflect the value and stature of these researchers" (2012, p. 39).

Third, in spite of technicians' advanced degrees, the precarity of grants and federal immigration laws suggests that, even for the most economically productive members of society, a new normal defined by political and economic precarity characterizes much of contemporary laboratory life (Standing, 2014). Indeed, the general conditions of technicians' employment are reflective of broader trends in post-industrial globalization, which are "historically present in female work—precariousness, flexibility, mobility, fragmentary nature, low status, and low pay" (Oksala, 2016, p. 281). In contrast to some immigrant laborers who have little formal education, (e.g., women who labor in nail salons), academic laboratory technicians hold tertiary degrees. Nonetheless, they experience similar effects as their counterparts in other service sector jobs in terms of employment and immigration precarity (Kang, 2010). I will show how these forms of precarity impact the types of experimental questions posed by technicians in concert with PIs.

In this chapter, I work to restore visibility of technicians. I build on the works of historian of science Steven Shapin (1989) and labor sociologists Stephen Barley and Julian Orr (1997) as well as Stephen Barley and Beth Bechky (1994), who studied the role of technicians in science. I argue that the social organization of the Lab itself has epistemic effects on what comes to be known about obesity and metabolic syndrome because precarity (economic and political) is built into the Lab's division of labor and its experimental systems. Specifically, technicians' work conditions affect their caretaking practices around the Lab's experimental systems because "care for the Lab's experimental systems" varies by technicians' position within the Lab as either a

career technician or a temporary one, creating, in effect, a division of caretaking labor (Rheinberger, 1997, p. S246). Although caretaking has been previously identified as a critical element of technicians' labor, my findings suggest that caretaking entails multiple valences that are specific to one's occupational position at this Lab (Barley & Bechky, 1994; Barley & Orr, 1997). Academic lab technicians are not a homogenous occupational group. At the Lab, they circulate along very different career trajectories and anticipations which structure their caretaking practices, either as a career or temporary technician. To describe these practices, I adopt the phrase of *anticipatory caretaking practices* to show how career anticipation structures technicians' "way of actively orienting oneself temporally" to care for the Lab's experimental studies (Adams, Murphy, & Clarke, 2009, p. 247; Fortun & Fortun, 2005).

My central argument in this chapter has two elements. First, technical staff, who have a variety of job titles, are the primary producers of laboratory facts, and they are living very specific forms of life. This particular observation reflects national data collected on the composition of the biomedical workforce today. The second part of my argument is that technicians' different employment timelines and aspirations impact the Lab's experimental systems and, therefore, the production of different types of knowledge. Immigration status (on either an educational J-1 or an employee H-1B visa) intersects with professional ambitions; shaping. In short, I argue that different laboratory facts, in part, emerge depending on career status. For example, career technicians help establish experimental systems in which to pose fundamental (i.e., basic science) questions that can, in turn, reshape the trajectory of those systems. Temporary technicians, on the other hand, use the Lab's existing experimental systems to create small slices of facts that help proliferate "normal science," i.e., working within a relatively settled explanatory framework (Kuhn, 2012). The key difference between these two

activities amounts to 'finding the question' and establishing new experimental systems, on the one hand, and 'finding the answer' within established experimental systems on the other (Landecker, personal communication). As a result, what each subset of technicians learns to anticipate needing to care for, in turn, shapes knowledge production in obesity and metabolic studies. To make this argument, I build on previous science studies as I consider the structural effects on how knowledge is produced and by whom.

In the pages that follow, I first provide a theoretical framework for studying technicians who labor in the "backrooms of science" to produce new obesity knowledge (Barley & Bechky, 1994). Second, I use ethnographic and interview data to provide Lab technicians' biographical backgrounds, career anticipations, and trajectories within the Lab. Third, I utilize examples from the maternal-obesity and maternal- bisphenol A (BPA) experimental systems to demonstrate how the work of anticipatory caretaking practices shapes laboratory fact making. In doing so, I illustrate variation in technicians' modes of anticipatory caretaking, depending on whether one is a career or temporary technician.

Theoretical Framework: Anticipation as Affective Labor

Although early accounts of "laboratory life" generally eschewed the work of technicians, notable contributions exist in intersecting bodies of literature. For example, historian of science Steve Shapin (1989) argues that, because cultural ideas of "work and authority" in Western societies devalue manual labor and since laboratory technicians are viewed as providing manual labor, they have been overlooked in studies of scientific practices. Greater prestige, autonomy, and authority are bestowed on those with higher social class and status; i.e., those who are said to possess "truly reflective and rational philosophical knowledgeability" (p. 561). In this cultural framework that has persisted for many centuries, scientific activity is viewed "predominantly as

thought rather than as work," i.e., using head rather than the hand (Shapin, 1989, p. 561). Additionally, because servants work in exchange for a wage (unlike gentlemen of science), they were also made politically invisible. In the 17th century, to work for a wage was to forgo a man's birthright as a free Englishman, which meant that servants were bounded economically and politically to their masters. As a consequence, laboratory servants were made doubly invisible in science and the political climate and that view has somewhat survived through today.

In the 21st century, parallels exist for basic science laboratory technicians. As it happened to Sir Robert Boyle's 17th century servants, the historical significance of one's birthright today plays out in the context of immigration policies in the US via the J-1 educational visa and the coveted H-1B professional visa, which are designated for individuals with advanced degrees. At the Lab, the work of producing new scientific knowledge is predicated on the availability of a relatively low-cost yet highly skilled pool of individuals who work (sometimes as volunteers) under very precarious financial and political conditions. While their contributions are increasingly acknowledged by PIs (as in the ubiquitous "acknowledgment slide"), academic technicians remain an economically and politically vulnerable workforce today as they were in the 17th century.

Although some technicians today have gained visibility as co-authors on scientific papers as part of the reproducibility of labs and PIs, they are a largely unexamined in social science accounts of science. Sociologists Stephen Barley and Beth Bechky (1994) argue a two-fold reason for this: early sociological accounts of science and medicine were primarily concerned with institutional analyses, and, in spite of a subsequent turn toward scientific practices, ethnographic lab researchers emphasized epistemic questions about how knowledge itself is situated by its sociohistorical contexts (Latour & Woolgar, 1979; Lynch, 1985; Traweek, 1988;

Haraway, 1988; Shapin, 1989; Barley & Bechky, 1994; Whalley & Barley, 1997; Russell, Tansey, & Lear, 2000; Tansey, 2008; Iliffe, 2008; Wylie, 2017). As a result, social scientists trained their analytical focus on authors who had social and economic capital (i.e., authority) and, in so doing, perpetuated the historical invisibility of technicians who nonetheless constitute part of a laboratory's social life and who perform work, and indeed invest in theirs and the lab's capital, on behalf of investigators.

In contemporary studies that do incorporate the role and contributions of technicians, they are often manual workers who have comparatively little education and are therefore seen as mere hands in processes of fact making. Or, they are technicians who work in a non-academic, i.e., clinical, hospital, or industrial setting. For example, science studies scholars Lynch, Cole, McNally, & Jordan (2008) found that lower-level technicians in a forensic lab perform "highly routinized work" in which "thought and creative variation [were kept] to a minimum" (pp. 89-90). Even in their classic treatment of laboratory life, Bruno Latour and Steve Woolgar (1979) footnoted the existence of technicians while nonetheless noting that "[t]heir importance in the production of facts is usually underestimated" (p. 232). But, in basic science labs today, technicians are not merely "hand" help or even "super-techs" that can be easily exchanged or circulated on the market (Latour & Woolgar, 1979, p. 223). They must possess, as Rayna Rapp (1999) has argued in her analysis of the interpretive work technicians perform in chromosomal diagnoses, both formal and contextual knowledge. Academic technicians must work with hand, head, and heart in order to help produce new knowledge (Rose, 1983). Further, they cannot simply "lend their skill to an investigator, in exchange for a secure position and some nonmaterial satisfactions" because of their precarity as recent immigrants and the larger funding landscape in basic science (Latour & Woolgar, 1979, p. 223). Career technicians reminded me

continuously that, without grants, there is no funding, no security, and no job. But, as well, no visa.

According to labor sociologists Barley and Bechky (1994), technicians in academic laboratories perform two central roles: translation, which occurs at the "empirical interface" between "material and symbolic realms," and caretaking, which involves the maintenance of "machines, organisms, and other physical systems" (1994, p. 90; Whalley & Barley, 1997). What these examples illustrate is that brokering new laboratory knowledge occurs through translation and caretaking practices, both of which entail the use of formal and contextual knowledge by technicians. Expanding this discussion on caretaking practices, I contend that learning to have a "feeling for" an organism at my research site reflects something akin to visceral affinity, an affective state complementary to but different from Barley and Bechky's invocation of "having a feeling for" as simple familiarity or tactile skills (p. 100).

Therefore, in the process of learning to care for the Lab's experimental systems, I argue that technicians' work resembles aspects of affective labor, a mode of immaterial labor that emphasizes the role of caring work in the embodied processes of scientific knowledge production (Hardt, 1999; Hardt & Negri, 2000). How is affective labor defined? According to Michael Hardt (1999), affective labor refers to caring work "immersed in the corporeal, the somatic" which, among other things, produces biopower (p. 96). Moreover, he writes that affective labor has become a dominant way to extract value from workers in post-industrial work environments (p. 97). Hardt argues that, in addition to the labors of knowledge, information, and communication in the production of scientific knowledge, affect "play[s] a foundational role in the production process" (1999, p. 93). Drawing on this definition of affective labor, I argue that forms of laboratory labor include not only theory and technical practice as emphasized by Barley

and Bechky (1994) above, but also the embodied labor of care work where 'caretaking' is defined as much more than the daily maintenance of laboratory subjects.

Affective labor is shown, for example, in sociologist Carrie Friese's (2013) analysis of a translational laboratory that employs animal models as representatives of human bodies and conditions. Friese argues that the "unseen element of care plays a constitutive role in the organization and setup of an experimental system" affecting "both the biology of the mouse or rat as well as the scientific understanding of that biology" (p. 131). The affective dimension of care not only informs how experiments are or should be carried out but also that the outcomes of care (new knowledge) have epistemological value in that they shape our understanding about what we claim to know. This is congruent with Rose (1983), Puig de la Bellacasa (2011), Lappé (2014), Fortun & Fortun (2005), and Oksala (2016).

In her work on autism research, Martine Lappé (2014) demonstrates how care becomes implemented in research study designs with the effect of shaping how knowledge about autism is produced and acted on by stakeholders, including parents and scientists. The ethnographer of science Natasha Myers (2008), in her work about the bodily engagement and attachment of molecular biologists to their 'objects,' also "shows the crucial affective labour and care involved in 'giving life' to a molecular model" (Puig de la Bellacasa, 2011, p. 98.). Ethnographic illustrations of affective care labor in science, such as the ones mentioned, reflect an extension of Evelyn Fox Keller's (1983) earlier concept of what it means to have a "feeling for" the objects on which a scientist intervenes. What scientists learn to care for and about in the laboratory often reflects larger social concerns about what human ailments matter the most which, in turn, shapes the conduct and course of scientific investigation. This logic suggests a "double significance of care" at play (Puig de Bellacasa, 2011, p. 90). What people care about in terms of health (namely

obesity and diabetes in this instance) funds specific research topics and designs. This impacts what scientists study, and indeed mold their entire careers to. An added element to this dynamic is that economic and political precarity also influence matters of care. Care for the Lab's experimental systems depends on technicians' occupational status as career or temporary technician, which is intimately connected to their immigration status and thus livelihood.

Lastly, within the literature on politics of care and caretaking, social scientists consider the role of anticipation on how we care for future selves. Medical sociologists Vincanne Adams, Michelle Murphy, and Adele Clarke (2009) argue that "anticipation" is hegemonic today in that the work of living has itself become a form of work. The work of anticipation is viewed as something that is a moral obligation that we enact by managing and optimizing our own biomedical futures. Anticipation, therefore, is not "*just a reaction, but a way of actively orienting oneself temporally*" toward living life in a speculative fashion while, at the same time, optimizing it (p. 247-248). This notion of anticipation reflects what sociologist of science Aaron Panofsky (2009) terms "personal social policy," an outcome of living life speculatively.

In summary, I borrow this concept of anticipation as one dimension of affective labor and apply it to technicians. Technicians anticipate their own career trajectories and therefore mold their caretaking practices in relation to them. This, in turn, affects how they actively orient themselves to fostering the technical conditions of laboratory life in order to produce facts. In sum, anticipation, through the organization of work as modes of anticipatory caretaking, has epistemic effects for what we come to know about obesity and metabolic syndrome. What facts make it to the light of day outside the Lab itself, depends as much on funding for hot topics as it does a low-cost and vulnerable workforce of 'technicians'.

Now that I have described the theoretical framework that shapes the notion of anticipatory caretaking practices, I first turn to an overview of technicians' background, their training, and career trajectories in the Lab. Once I have illustrated their social positioning in the Lab, I then draw on ethnographic examples to show how anticipatory caretaking practices vary by social location in the Lab and how this affects the production of new obesity knowledge.

Technicians at University Lab

The Lab's division of labor not only mirrors the NIH's characterization of a typical laboratory given above but also dovetails with Rayna Rapp's (1999) observations that "laboratories are staffed by an international circulation of highly skilled scientific workers," and that their work is often perceived as "women's work" (p. 199). By "women's work," I mean to suggest that the labor itself is gendered, i.e., it is marked by "precariousness, flexibility, mobility, fragmentary nature, low status, and low pay" all of which are qualities "historically present in female work" (Oksala, 2016, p. 281; Standing, 2011). In many ways, the Lab's labor organization and technicians' work practices reflect a metaphor drawn long ago by physiologist Claude Bernard (1865) in which he described a physiology laboratory as a "ghastly kitchen," where science resembles a messy kitchen rather than a highly-organized experimental endeavor, a metaphor that Bruno Latour (1992) would later articulate as the "costly ghastly kitchen" (p. 300). And yet today, it is not science 'great men' who are 'cooking' down in the laboratory, but rather an amalgamation of science technicians who, precisely because of their economic and political vulnerability as immigrants, bring "stability" to the Lab in the course of producing new facts about environmental determinants of obesity and metabolic health (NIH, 2012, p. 10).

Technicians at the Lab 'cooked' in several locations across the larger campus. First, the main laboratory area consisted of two 'wet' rooms filled with an array of machines, tools,

bottles, flasks, and other items used in the course of everyday work. Second, adjacent to the wet lab, there was a shared computer room with computer stations for four people, but never enough space to occupy everyone on a full day at the Lab. Within this office, technicians strategically combed through scientific papers, investigated protocols, annotated lab notebooks, stored and prepared images and drawings, and utilized software programs to document, interpret, plan, and troubleshoot results. Third, a short walk away from these rooms was the Lab's procedure room. A space, shared with other institute's employees, where animals were sacrificed and early steps of experimentation, like whole animal fixation (perfusion fixation), were performed. Fourth, in another common room, the Lab stored tissue samples in refrigerators and freezers for future use, and lastly technicians shared a common darkroom to expose films that had been treated according to particular visualization techniques. This was a place where "Did it work?" was a familiar question because, once films had been developed, technicians eagerly read out a series of smudges that they interpreted as a sign that their earlier experimental work was a success, i.e., a positive result.

The organization of empirical work at the Lab typically began with weekly meetings, which provided a time and space for Drs. Harris and Moore to meet with most, if not all, of the technicians to review events over the preceding week and to map out plans for the upcoming one. Lab meetings were held in Dr. Moore's office in a building located adjacent to the laboratory. She led the weekly meetings and her questions to the team were often organized by technician because each one was responsible for specific biomass in a specific experimental system. The overall pace of experimental work was dictated by a need for data for use in imminent grants, conference presentations, and publications. As I was once informed, "you are

always getting data for either grants or publications," and "it takes a long time to get a good story" (anonymous, personal communication, February 2, 2015).

Career technicians. In this section, I provide biographical data and career trajectories for the Lab's three career technicians: Eleanor, Olivia, and Carrie. Eleanor, the most senior and entrusted technician, primarily worked with adipose (fat) tissue and cells in the maternal obesity model.[13] She was also called upon to teach less experienced technicians in techniques, to troubleshoot problems as they occurred in myriad circumstances, and to help establish a new experimental model around programmed appetite pathways in the hypothalamic region of the embryonic mouse brain (discussed in chapter 4). I was told that the Lab "would be doomed" without Eleanor's contributions (personal communication, January 4, 2016). Dr. Moore told me that "to find someone of Eleanor's caliber and passion, at her level, would be difficult." Dr. Moore could suggest a protocol for Eleanor to follow on a particular study and, although Eleanor "has [previously] nailed down the techniques," she usually had to navigate unforeseen circumstances in everyday experimental work.

Once, for example, in the midst of a protocol disagreement between Eleanor and Olivia, a junior investigator whispered to me that Eleanor has a "strong personality" as a way to explain why she seemed so impassioned, but that situation also revealed to me how they each care for particular details of a protocol. Both technicians were pressed continuously with uncertainties and, as a result, they oriented their caretaking practices towards whatever end each one deemed best for the task at hand. In fact, the degree of their personal investments in the Lab's success could be measured by the timing, pitch, and tone of their voices during disagreements with one another. If they did not care, they would not argue about the best way to handle either anomalies

[13] To maintain anonymity, I use pseudonyms.

or even give suggestions about what particulars of biomatter may be relevant to their investigations.

I also found that having a "strong personality" in general must have served both career technicians well since the work sometimes entailed long and erratic hours, constant learning, problem-solving, and supervision of others (including me) for which they received relatively low pay, in the range of 50k per year. Eleanor suggests that this type of job is so stressful that many people eschew basic science research altogether and opt for more stable careers in biomedicine. She explained to me:

> Look around. Why are there no young people here? Students [undergrads and graduates] won't stay here. They won't take this kind of job. They just get experience, a recommendation, and then go on to another thing like medical school because the salary is too low, and it is stressful. If you don't have a grant, you don't have a job. Nobody can guarantee you get a grant all the time.

After a pause, she added, "Look how many Chinese are here. They are all foreign here, and they come because this job is easy [for obtaining] a green card, and no other American wants to do this job, especially now. My son wouldn't."

Although Eleanor received a medical degree in her home country where she also worked as a professor before immigrating to the United States, she had worked as a technician for several labs during her nearly two decades at the Lab's home institution, University Biomedical Park (Bio Park). She was a permanent resident. Eleanor had spent approximately the last ten years with Drs. Harris and Moore. Because she was the most trusted and experienced technician who anticipated continuing her career with these PIs, Eleanor's positioning within the Lab structured her daily anticipatory caretaking practices. As she planned a work schedule to be done in any upcoming week, Eleanor routinely asked the PIs, "okay, what kind of data do you need for that grant renewal?"

Working in anticipation of their future needs, she cultivated and optimized environmental *in vitro* and *ex vivo* conditions for primary adipose cells, explored epigenetic mechanisms associated with adipose development and function, and mapped out new experimental terrain for the PIs by "searching for the unknown" in daily cell culture techniques. Outside of the Lab itself, she read scientific papers to help keep her informed but also to consider new experimental directions with fat cells such as how to get white fat cells to act more like brown fat cells (to burn fat rather than store it). Lastly, she helped discipline data, particularly with Western Blotting (a technique and tool that makes protein expression visible to the eye), which is often one of the final techniques done to "see whether or not her experiment worked."

Although precarity, flexibility, and low pay were the reality of her work, Eleanor chose this path and, as I asked why, she said,

> I really like my job. That is why I stay for my whole career. I graduated from medical school, but I don't like to deal with patients. I like research. [The] clinic is boring: same patients, same problems, same questions. It is very, very boring. With research, I keep learning, keep doing, keep reading. It is very interesting.

When I asked "in what ways?", she said that "In one type of lab, you only do one type of experiment and then pass it on to the boss, but here, at this Lab, it is another experience, where one project might need a lot of experiments" (personal communication, February 6, 2015).

Again pointing to the impact of grant precarity on employment, Eleanor remarked that when she started working at Bio Park in 1997, the NIH had more funding to give, a fact reflected in the NIH's data and which is often repeated in the Lab. She correctly noted that, in the late 1990s, approximately thirty percent of applications were funded, but today it is around ten percent or less, and "that is really hard," she said (personal communication, January, 2015). Consequently, unlike clinical technicians who typically work at a hospital or medical office and

enjoy the prospect of regularized employment, academic technicians like Eleanor are permanently dependent on PIs' grants for their own employment.

Olivia, the newest career technician, transferred from another major university research center doing stem cell research. She also held an advanced degree and, like Eleanor, she was an international scientist-technician. In a description of her experience as a foreign scientist, she told me, "You can work for a PI for 10-15 years on a visa, make very little pay ($26-45k), especially in academia versus the private sector. By the time you get your green card, you're old, and there is an age ceiling." Feeling "captive" by immigration policies, she added,

> Even when you first get the visa, employers take advantage of the system and treat the first year of employment as a trial phase. When switching jobs, the visa rules won't even allow like a month break so one can take a longer vacation. Labs here at Bio Park are very immigrant based. Look at the local universities, and you will find the same thing.

Olivia's reflections on the conditions of her employment echoed not only what Eleanor had separately described to me, but also confirmed Dr. Harris' appraisal of technicians' precarious positioning within the contemporary biomedical workforce. In an interview, he remarked that, while being "technical staff" is a good path into the United States and, for some, the medical profession, it is hard on career technicians in academia because they "get to a certain salary level, then [if] that lab closes…they can't get hired at that same level" (personal communication, December, 2015). To underscore this point, Dr. Harris reflected on his family and added that none of his children or their friends wants to "be a Ph.D. in the sciences… it is not a very common field to pick. And maybe because it is not that secure, particularly as you get into academic stuff" (personal communication, December, 2015). From his and others' perspectives in the Lab, those at the laboratory bench today tend to be drawn from outside countries and, although there is a potential path toward residency, it does not come without costs.

In the course of her work, Olivia had learned to care for and about both mouse physiology and how neuronal pathways form during embryogenesis within the maternal-obesity experimental system. As she learned to care for the hypothalamus, and specifically a region within it referred to as the arcuate nucleus (an appetite regulating region of the brain), she found that working with stem cells that have not yet differentiated into mature, or fated, primary cells makes them "far more tricky" than mature adipocytes. Nevertheless, she derived and cultivated neuronal stem cells from the hypothalamus taken from mouse pups one or two days old, learning how to handle dime-size brain tissue. Cells were then derived from brain tissue, specifically the arcuate nucleus, and Olivia cultured them to the point she determined that they were "pure" of surrounding tissue, "healthy," and in sufficient number to experiment with via transfection techniques (used to study and control gene expression). Like Eleanor, Olivia also explored obesity pathways through multiple experimental interventions and study design to figure how to best collect and cultivate 'appetite' and 'satiety' neurons in order to test theories of enhanced programmed appetite.

Lastly, Carrie, the 'animal tech,' managed the Lab's experimental animal models. Like the other career technicians, she held an advanced degree, but Carrie was the only American-born personnel other than the most senior PI, Dr. Harris. Carrie was responsible for the care of mice and rats in multiple senses: purchasing them from particular breeders, attending to their husbandry, safety, housing and other physical milieu such as lighting and temperature, exercise, medical care, and, of course, overseeing their nutritional milieu. Finally, she "sacrificed" animals as fresh samples were needed for experimental work and also when animals began to take on "old person problems" and were deemed not valuable to future studies. More than any other

technician, her anticipatory caretaking was molded to the temporal lives of animals in terms of routine maintenance, reproduction, and experimental interventions.

Before Eleanor and Olivia could go "on a fishing trip" with tissues and cells, Carrie collected data on animals' phenotypes in a quiet room set apart from the laboratory. She routinely measured animal weight, food and water intake, blood pressure, performs body DEXA scans (a noninvasive measurement of fat distribution in the body), conducted glucose tolerance tests, and collected data on animals' plasma to capture changes in cytokine levels. These baseline measurements were critical for PIs who "need the measurements so [that] we know what we are doing" (personal communication, February, 2015). The "molecular stuff," I was told, "all starts with the phenotypes [because they] tell us which direction to go" so that they can hypothesize what is "driving" molecular mechanisms that increase fat cells or appetite in exposed animals (personal communication, February, 2015). And because phenotypes represent intersection effects of an organism's genotype and its environments, Carrie was often confronted with anticipatory caretaking and troubleshooting specific to the whole organism in relation to environmental regulation of their biology. The results of her practices, therefore, helped the Lab shape and characterized a molecular "story" about regulatory changes in multiple organs, tissues, cells, and pathways that influence appetite and satiety.

In sum, because of financial and political precarity, career technicians mold their careers to the Lab's experimental systems, which they help establish and maintain as a result of their anticipatory caretaking practices. The main aspects of anticipatory caretaking practices include cultivation, exploration, and discipline in order to help refine or push existing experimental systems in new directions. Carrie cultivated the livelihood of animals in the vivarium while the other career techs, Eleanor and Olivia, fostered environmental conditions of cell culture and *in*

vitro studies with gene expression. Together, they "search around the unknown for direction," mapping out new experimental terrains on behalf of PIs, and discipline findings that result from a sustained "absorption in, even identification with" their material subjects (Keller, 1983, p. xxii). With a gaze on environmental causes of obesity and metabolic syndrome, anticipatory caretaking reflected ways in which career technicians embodied the Lab's hypotheses, helping to provide an organized sequence to the Lab's staggered studies around grant cycles and the reproductive lives of experimental animal models.

Temporary technicians. The Lab also employed several people who worked as technicians but who went by different titles, volunteering their time from one summer to two years. For temporary technicians, a more obviously transactional relationship existed whereby unpaid, volunteer labor was exchanged for letters of recommendation from PIs and co-authorship on conference abstracts and scientific papers. The Lab typically offered an academic fellowship to an accomplished high school student during the summers and provided greater in-depth training to other itinerant students, including undergraduates from a nearby university. Longer-term volunteers included a Medical Fellow, Julia, and two International Medical Graduates (IMG), Maryam and Veronica. In this section on temporary technicians, I specifically focus on the IMGs, medical doctors who entered the Lab as practicing OB/GYN surgeons in their sending country. IMGs illustrate the intersectionality of global north-south migration in medicine as well as the political economy of grant funding. Because they are considered students and dependent on publications and strong letters of support for future residency positions, their own positioning within the Lab differently structured their anticipatory caretaking practices.

According to the Association of American Medical Colleges (2015), IMGs are part of a trend in which one out of four doctors in the US today is a foreign medical graduate.[14] Under the Trump administration, however, this has become more complex and precarious for immigrant scientists, especially for those from countries designated as "State Sponsors of Terrorism." Maryam and Veronica, both of who were from one of those countries, agreed to work for approximately two years at the Lab while also completing all three testing steps of the US Medical Licensing Examination. In order for Maryam and Veronica to be competitive in future residency programs, they must acquire excellent referrals from American doctors in addition to having at least one publication. Despite the hard work involved, they considered their position in the Lab as a form of "privilege" that enabled them to become a licensed physician with long-term American residency not only for themselves but also their young children (personal communication, June, 2015). Their contribution to the Lab's translational profile within the developmental programming field of obesity reflected a "mutual extraction" between PIs and temporary technicians because, in caring for PIs, temporary technicians also engaged in their own anticipatory professionalization practices (Lappé, 2014, p. 305; Shapin, 1989).

IMG's anticipated becoming medical practitioners in the United States and, because their anticipations exceeded the limits of the Lab itself, they developed anticipatory caretaking practices more closely aligned with reproducing "normal science" through the Lab's experimental systems and, outside the Lab, reproducing its capital via conference presentations. As part of their anticipatory care practices for the Lab's experimental systems, IMGs had to "get results." By results, they explicitly meant "positive" results because, in their words, reviewers

[14] See also http://annals.org/aim/fullarticle/2654788/u-s-immigration-policy-american-medical-research-scientific-contributions-foreign; http://www.ama-assn.org/ama/pub/about-ama/our-people/member-groups-sections/international-medical-graduates/imgs-in-united-states/imgs-country-origin.page?; H1B visas: https://usimmigrationlaw.net/h-1b-professionals/n

and readers wanted a "happy ending;" that is, they wanted "results correlated with each other" across publications (personal communication, May, 2015). Maryam described their experimental work as like "making a story," and as with "directing a film, there are a lot of scenes, [and sometimes] actors don't always play well together, and people want a happy ending" (personal communication, May, 2015). To that end, the anticipatory need to create positive results for future audiences structured emergent experimental questions in one of the Lab's maternal-BPA model. One day at the bench, for example, Maryam was encouraged to "get that muscle done!" in anticipation of an upcoming conference she needed to participate in. In this example, technicians examined the programmed effects of BPA exposure *in utero* on offspring' fat tissue, liver, and skeletal muscle—effects that they were not planning on studying in any imminent sense. But because they needed "positive results" in anticipation of what was in fact an imminent conference, they learned to care for fat tissue, liver, and skeletal muscle rather than another biological object.[15]

Ironically, these temporary technicians acknowledged that "socially" it would be great to find that the effects of BPA do not persist in the model, and that "the scare of BPA may not be so great after all," but they anticipate and even *hope* to find that the deleterious effects of BPA persist because that is tantamount to a positive result (personal communication, July, 2015). The demand to produce "happy endings" that correlated with other findings in an existing experimental system amounts to caring for particular organs and epigenetic pathways in animal models of obesity and metabolic dysfunction and the course of doing science was paced by technicians' own timelines within the Lab. In spite of recognizing the social good of negative

[15] Liver and skeletal muscle help regulate glucose production, utilization and storage. Dysfunction in insulin signaling can lead to an increase in glucose intolerance. Therefore, it is important to studies of environmental determinants of obesity and metabolic dysregulation in type-2 diabetes.

results in the case of BPA, the professional pressures on temporary technicians to get positive results for publication, and to circulate those findings at conferences, encouraged anticipatory caretaking practices that not only enhanced their professionalization but also increased PIs' reputation in the field, and more broadly reproduced the problematic of obesity in the field of Developmental Origins of Health and Disease.

Although investigators and technicians in the Lab described their work in passionate terms, it would be an overstatement to say that intellectual interests determine research agendas. Investigators recognize that what matters for funding is a combination of factors from "hot topics" as well as the skills of their technical staff because it will be they who produce "all the data" on which everyone's career depends. It is no surprise, then, that Drs. Harris and Moore took into consideration what their "lab [is] good at doing" as they contemplated study designs (personal communication, December, 2015). Such a practical reality of basic science research at the Lab reflects national trends cited above whereby funding affects both the type of scientific questions that are likely to be supported as well as the makeup of the biomedical workforce.

Anticipatory Caretaking Practices

Having explained the Lab's division of caretaking labor as well as the distinction between career and temporary technicians, I shift my focus below to highlight anticipatory caretaking labor in greater detail. I draw on examples from the care of animals in the vivarium and procedure room, and the *in vitro* work done in the "maternal-obesity" (career technicians) and "maternal-BPA" (temporary technicians) experimental systems. Anticipatory "care of the data" operates at many levels, including for animals, tissues, cells, genes, and proteins (Fortun & Fortun, 2005). It also operates at the broader level of concept renewal, which is necessary to the

reproduction of the Lab in the field of epigenetic and developmental programming models of obesity.

Career technicians: Cultivating life's "technical conditions" while "searching the unknown" for a "good story." Labs are extreme environments where optimizing the technical conditions necessary for fostering health (and illness) in animal life involves complex processes.[16] To illustrate the work of anticipatory caretaking among career technicians, I highlight three technical and physical environments: animal housing in the vivarium, animal handling in the procedure room for *in vivo* studies and sacrifices, and biomass handling at the wet bench for *in vitro* studies with tissues, cells, and proteins. In each of these environments, the physical milieu, including the effects of handling, presents a tremendous source for potential experimental variability and, because of that, they are closely monitored (Balcombe, 2006). They also involve different sources and types of stress.

An ethnographic focus on stress is useful for two reasons. First, an awareness of stress and its physiological effects on biomass "relocate[s] the human subject within that [laboratory] environment, making the researcher integral to, controller of, and obligated to, the laboratory animals' well-being" (Kirk, 2014, p. 258-259). In other words, the reality of environmental stress for animals (and their parts) due to their living conditions requires management by human interlocutors. This often unspoken intervention on stress by technicians is an element of care that affects animal physiology. Second, relatedly, this observation conveniently emphasizes the role of an animal technician as an important element in the "relational, epistemological, and ontological interdependence of the knowing human and the animal object of (or means to) knowledge" (Kirk, 2014, p. 258-259). Third, animal stress from handling and housing conditions

[16] It is also filled with some irony. How can the wellbeing of experimental animals be realized when they are being made sick?

has been shown to alter animal metabolism and negatively impact reproduction all of which are of vital empirical importance to this specific Lab (Ghosal, et al., 2015; Kamakura, Kovalainen, Leppäluoto, Herzig, & Mäkelä, 2016).

Metabolic processes and reproductive outcomes are particularly relevant to a lab that studies maternal-environmental conditions of obesity and metabolic syndrome. If, for example, housing conditions around temperature and humidity are not optimal, stress affects change in food intake and weight. Regarding the Lab's *in vitro* studies, cells experience environmental stress during culturing techniques (such as seeding and passaging) that could even alter the cell's morphology and genetic expression, leading to false outcomes. As well, transfection techniques used for gene silencing can be so toxic that treatment itself kills the cells. Lastly, not all cells are alike. Technicians must also learn to care for and cultivate different kinds of cells and tissues. Consequently, how technicians handle animals and biomass, while managing the physical aspects of the vivarium and the wet lab, is critical for cultivating life, creating new protocols, and making novel interventions. Their anticipatory care practices are not just reactive to laboratory animals and their parts, or the physical conditions of experimental work. They illustrate the active, interdependent ways that technicians orient themselves to the optimization of life's technical conditions in the laboratory (Adams et al., 2009).

The prime considerations for rodent housing include types of cages, temperature, humidity, air quality and ventilation, sound and vibration, a constant 12-hour light/dark light cycle, food and water provided *ad libitum*, enrichment toys, and bedding. While Carrie, the animal technician, did not make purchasing decisions, she was attentive to variation and changes within each cage and the vivarium as a whole, adapting her practices to help ensure the well-being of animals, which in turn affects experimental outcomes. The macro and

microenvironments were, in fact, an area of care that Carrie closely monitored on a daily basis. Mouse and rat cages vary, but gone are the days of wooden cages, galvanized metal and wire, and stainless steel. Today, enclosures are made of plastic with variation in specific chemicals used to manufacture them, e.g., polycarbonate, polysulfone, and polypropylene. Many, such as polycarbonate cages, leach bisphenol-A into laboratory animals and, therefore, the Lab uses BPA-free (but still plastic) cages in its maternal-BPA study with rats. To further help control for estrogenic effects on rats, the Lab used estrogen-free bedding, water bottles, and stoppers because microenvironments of the housing can produce estrogenic effects on the animals. (Thigpen et al., 2013) Despite these accommodations, it is entirely possible that other chemicals such as bisphenol-S can still leach, and this is discussed in Lab meetings as a potential confounding problem in their BPA studies. Compared to control rats, for instance, current BPA rats showed no enhanced appetite or weight gain, which perplexed PIs and technicians. In fact, the control animals showed elevated levels of BPA in their blood serum.

Temperature and humidity are particularly important variables to control and maintain. Ideally, rodents are housed in a "thermoneutral zone" so as not to alter their metabolic pathways. This includes the potential for heat production if animals get too cold, or evaporation (sweat) if they get too hot. Even though the thermostat is on a controlled setting, the number of animals in a cage affects the micro-climate of each individual cage because bodies regulate thermogenesis differently depending on the group size in each cage. The cozier it is, the warmer they will be. Cages also have weight restrictions, which presents a dilemma for group and individual housing. If a rat weighed 500 grams or more (a very fat rat), Carrie would move it to single housing but, if she had two females that each weighed 250 grams, she would keep the two together in the smaller BPA-free cages because "they like to be together." Otherwise, she noted that they

"probably get stressed," and "some will not eat," creating an ethical and experimental dilemma for her. Carrie attempted to anticipate potential housing problems by daily keeping track of variables like temperature and humidity across cages and the vivarium. Carrie acknowledged the constraints around body weight, social thermogenesis, and animal sociality as she attempted to mitigate environmental stressors by tending to the animals' social nature. This dynamic underscored a perpetual tension between ethical and experimental demands that shaped her active orientation to the animals.

Because mice and rats have ultrasonic hearing, they are also particularly sensitive to sound and vibration. Except for one occasion when we realized that our talking was disturbing her ability to take measurements of blood pressure, Carrie kept noises to a minimum when she handled the mice and rats. As for other housing conditions, although the 12-hour light/dark cycle was automatically regulated, Carrie performed most of her work during the day when these animals would prefer to sleep; presumably disrupting their natural circadian rhythms. Rodents are known to be active in the day (especially if the lights are on), but they are most active during evening hours when they prefer to play, eat, and reproduce. She also ensured animal cages were maintained with access to food and water *ad libitum*, which is a species-specific preference. While rodents once had red plastic toys to chew on, they now have paper towel rolls to play with. The red toys are common for rodent housing but, due to concerns with plasticizers, the Lab switched them out.

In addition to Carrie's anticipation of housing effects on mice and rats, she also was attuned to her handling practices. Her practices around touching, both invasively and non-invasively, closely reflect an active orientation toward the animals. One non-invasive but very time consuming and mundane task is to keep track of new litters by number, sex, and diet. Carrie

did this by marking pup tails with different colors of sharpie markers. However, as she noted, moms continually licked the color off of her pups' tails, and so Carrie had to reapply color every day, including on weekends, to help ensure she could track each pup's identity. How exactly this particular activity and exposure may affect dams and pups is unknown. For pups who mature to three weeks of age, Carrie then used ear punctures (which to an untrained eye looks as if a bite was taken out of an ear) to keep track of the animals as they matured to adults. Carrie also administered injections, such as glucose tolerance tests, and this was particularly challenging with large rats. Rather than using a typical product called DecapiCones (a tapered tube of plastic) that traps the animal inside a conical tube, she cuffed the rat by its neck and then wrapped the animal in a towel in a "football" hold position. Carrie noted that the plastic of DecapiCones can slide around the animal's body and, because the needles are only 5/8 of an inch, it is difficult to give the full dose of glucose. From Carrie's perspective, she anticipated how a rat, particularly a large one, might react to how she handled them and adjusted her practices accordingly. Her caretaking practices presumably help calm the animal while also administering treatment according to the protocol. For non-invasive, mundane, and yet critical practices, such as tracking pups with sharpies and handling larger animals in soft towels to administer injections, Carrie actively oriented herself both to the animals' needs but also, in the end, to the experimental apparatus itself, which she hoped to optimize for the sake of "clean" data.

Another example of invasive touch is the practice of "sacrifice," which I limit here to decapitation. Carrie anesthetized rats according to protocols set forth by the Animal Care and Use Committee (as well as others). She checked pain sensory by squeezing the rats' tail for a response. Once the animal had been prepped with alcohol like a shower spray, she used large scissors rather than a typical "rat guillotine" to decapitate their heads. In spite of the fact that

scissors were hard on her hand, she found they were more precise than the guillotine. Rats, she said, “don't have a neck like you and me, one that is distinguishable. So it is hard to tell where the back of the skull is sometimes, especially when they are fat, so you might accidentally cut into their brain” (personal communication, April 8, 2015). Once animals were decapitated, she moves quickly to dissection because “how we collect tissue is very important” to preserve its integrity. Carrie explained:

> We need the tissue as fresh as possible, so I remove it once a rat is decapitated. It is in minutes that things degrade. And it depends on the tissue, too, because whatever enzymes or proteins we are looking for can degrade very quickly, so as soon as I take the tissues out, I put them on liquid nitrogen for freezing at -80F, and some of the tissues I also fix in paraffin for sectioning and staining later.

Carrie cut through the rat’s skin and skull, scooping out the brain with a spatula, laying it flat on a tray set on top of liquid nitrogen. This freezes the brain so that it “keeps shape so we can identify the proper regions later.” Once she removed the brain, Carrie then dissected the remainder of the body, removing organs and tissues. Using a narrow tool, Carrie peeled brown fat from between the shoulder area and white fat from the legs. For a large rat, this process took approximately forty minutes, and for newborn pups, it was approximately ten minutes. Organs and tissues were then placed in a buffer to keep them “alive” until they could be fixed or stained. At this stage, career technicians “search for things” in biomass that might prove fruitful for future experimental questions.

While Carrie took steps to minimize stress, the details of her caretaking practices were not documented in the Lab’s publications. This mirrors results found from a 2018 meta-analysis in the journal *obesity reviews* on the behavioral and physiological effects of housing on animal appetite and fat mass. According to that analysis, “housing conditions are rarely considered as possible moderators of reported outcomes” in obesity research (Schipper, Harvey, van der Beek,

& van Dijk, 2018, p. 1). Science papers do include data on how animals are housed (e.g., type of cage, number per cage, sex of grouping, and weights), but what is missing, according to this report, is a discussion of how stress from housing and handling can affect a lab's results. For example, a typical entry in a methods section includes information on the model organism, type of cage, bedding, enrichment toy(s), source of food and water, and type of access to them. And, depending on the study, housing information might also include the number of animals housed in a group, their sex, and data on time and duration of isolation, if relevant. The Lab included certain details like how housing group size changes with body weight and when siblings in a litter were studied and needed to be isolated, but other than these descriptive details, which are indeed given in theirs and others science papers (there is nothing idiosyncratic or unique to the Lab in this regard), the possible physiological and psychological effects of the animals' housing environment and handling techniques were nevertheless only tracked in the Lab's internal record keeping documents.

Perhaps, partly because of this, definitions of the optimal technical conditions for laboratory animals, organs, tissues, and cells vary by laboratory and by technician. No wonder, then, that I was repeatedly reminded, "there is no protocol out there waiting for you. You have to create [protocols for each study and each experiment within the larger study]" (personal communication, February 26, 2015). Even in other labs studying similar epigenetic and programming effects of maternal-HF and maternal-BPA on offspring, no one appears to do it quite the same way. I was informed that 90% of published papers are "rubbish" since they cannot be replicated. I was also told that not every bit of minutiae is included in papers' method sections, such as handling and the micro details of vivarium housing conditions.

In addition to *in vivo* studies, the Lab performed *in vitro* studies that, ideally, were meant to support them, and which detailed the role of molecular mechanisms in enhancing or diminishing obese phenotypes. Eleanor explained that…

> [We] use the *in vivo* studies because they are close to humans, but [it is] limited by a lot. [You] can't make conditions the same. Animals have different attitudes. Some are more or less active, so you want to control all the conditions. You hope to support the *in vivo* study with the *in vitro* results.

For Eleanor, sorting out the technical conditions of cell culture practices represented one way that anticipatory caretaking occurred in her work with tissue and cells derived from mice in the maternal-obesity studies. Eleanor specifically studied white and brown fat tissue, including adipogenesis (how white and brown fat cells are formed), thermogenesis, and the role of mitochondria in both types of cells. This was done to grasp functional differences in fat and glucose storage and expenditure between both types of cells. In gene knock-out experiments, Eleanor attempted to suppress or amplify specific proteins that are thought to be integral to adipogenesis, including the role of epigenetic transcription factor DNMT3a in the proliferation of preadipoctyes (stem cells) and their differentiation into fated white and brown fat cells (i.e., adipogenesis). But, as she said, there is "no protocol out there waiting for you," and to "get…the optimal conditions," and she learned to anticipate and care for experimental conditions that embody the Lab's evolving hypotheses. By doing what appears to be controlled tinkering, she helped establish new experimental systems.

One morning, as I sat in the laboratory watching Eleanor, I noticed the intense care she took to physically handle brown adipose cells as she delicately transferred them from one platform to the next, so as to not disturb them in the culture plates. Occasionally, wanting no interruptions, she would politely say, "Sshh, no talking now," which was also often later followed up with a gentle smile, inviting me to observe cells under a microscope. During this

observation, Eleanor was in the midst of staining primary, brown fat cells derived from mouse sacrifices recently performed. Her goal was to help explain how thermogenesis in brown fat cells is impacted by fatty acid synthesis, storage, and metabolism; processes that complemented Carrie's *in vivo* studies with mice and rats.[17] On that particular day, Eleanor was testing the technical conditions of her cell culture to see if the cells had "eaten" the palmitate she fed them earlier in the week. Once they had been fed, Eleanor stained them with Oil Red O (ORO); a dye that is commonly used for visualizing triglycerides and lipids under microscope. With ORO, fat cells turn red, and the cell's nucleus turns blue. In short, it is a histological (tissue) visualization of fat cells. With this method, Eleanor checked for confluency (the proliferation and number of cells in a medium), and any possible contamination. The images were depicted on a computer screen and would "tell" Eleanor whether or not her cells were "happy."

As Eleanor peered down a microscope's lens studying these cells, she simultaneously raised a finger, pointing to a computer screen nearby that projects a fluorescent image of the cells onto it. Looking at the screen, I saw a constellation of red spots punctuated by blue nebulae. With a nod, Eleanor turned back to me to say they looked "beautiful," and that Dr. Moore would be very happy with her results. Reflecting on this, she explained that [this is] "why I like my job, and why I will stay for my whole career." In a brief moment that nonetheless seemed to capture her well-earned pride, Eleanor further reflected:

> Cells *are just like your baby*. Your cells are growing. You don't just respond. You want to see how they respond, e.g., to alcohol, to glucose, or fatty acids. You want to see their reaction. Do they survive? You have to know if your cell is happy or not. How to tell? Based on your experience. Every cell is different. There are a lot of signs to tell you if

[17] As previously described, brown adipose tissue contains more mitochondria and high oxidative capacity (burns fat), raising questions about the thermogenic capacity of brown (and white) tissue and their possible role on whole body metabolism. As a result, work on brown adipose tissue at this Lab and elsewhere is pursued as a novel approach to obesity treatment in the conversion of white adipose tissue to "behave" like brown.

> happy or not. First of all, the medium. Look at it. If it is dirty, the bottom of dish is foggy, a lot of other stuff, the cell won't be happy. We don't want a dirty bath. If there is floating stuff in medium, that is not happy. If the medium color changes, it is not good. If I have cells in the incubator, first thing I check are the cells. If I have to do an experiment, I have to check the cells. Before you leave, you check. Even if it is a holiday, you check. If it is the weekend, you might check once a day. Fortunately, I live very close.

Eleanor's view that animal cells have become her babies reflects a common refrain among laboratory technicians. If any experiment is to work, cells and their milieu must be intimately understood and handled. It is her anticipatory caretaking through learning to read the "signs" (and anticipating possible signs of distress) that she is able to nurture cells, getting them to multiply and differentiate depending on the empirical question of interest.

In other work with adipose tissue, Eleanor pharmacologically altered the message a protein received from the gene's messenger RNA by introducing a chemical that should silence the epigenetic transcription factor DNMT3A[18]. This process is known as transfection and is a technique that opens up pores in a cell's membrane so that its cytoplasm can take up the material introduced into it. The Lab had previously found that DNMT3a was suppressed in their maternal-food restriction model relative to the controls, so they were then exploring early suppression in their maternal-high fat experimental system. But, as with other experimental interventions, Eleanor modified her caretaking practices in anticipation of how a particular type of cell responded to her interventions.

As I watched her transfect cells, she told me that she had to "try different conditions." That day, she told me that it was "not the real experiment." By this, Eleanor meant that she

[18] "DNA methyltransferase 3 alpha is particularly important for establishing DNA methylation patterns during development before birth. The enzyme also functions in early cells that can give rise to more mature cell types. In early blood cells, called hematopoietic stem cells, the methylation patterns established by DNA methyltransferase 3 alpha promote maturation (differentiation) into different blood cell types" (National Institutes of Health, 2017b, para. 2).

created multiple cell culture conditions—the cells' physical milieu—in order to figure out what worked best. Introducing a chemical into cell culture medium has to be "sneaky." Cells have to think the chemical carrier (transfection reagent) is food so, like a Trojan horse, it can sneak in. Eleanor elaborated on this process by saying,

> The silencing – if it is successful or not depends on the cell density. Too much or too little, and it won't work. Very healthy cells are needed, so you need the ability to assess the density; you can't just follow the protocol. The protocol may tell you how many cells, but, no, that is not enough. You have to base on your experience because everybody's cell sizes are different. If a cell is big or small, the cell density is different, so then you have to seed the cells. Also, the silencing RNA—is it good? Every company says, 'my silencing RNA is good', that it can guarantee you will make silent. But no, you don't know. So, then, what carrier do you use? Every company says 'mine is good'. They say 'my carrier is non-toxic', but if it is non-toxic, it is not a good carrier. All these three together—the right cell numbers, a good silencing RNA, and a good carrier—they tell you whether the cell happy or not. For example, my cells might like the carrier, but Olivia's cells might refuse it. That is why we have testing all the time, how many cells, silencing RNA, and what and how much carrier to use. Lastly, siRNA and carrier ratio—you have to test it. It is very complex.

After explaining this to me, she informed me that her work that day was testing cell numbers, siRNA, the carrier, and the ratio of carrier to cells needed to silence the protein without killing it. For each of the twelve wells in the culture plate she used for this initial testing phase, Eleanor created a different physical environment. Each well had a different technical condition meant to optimize the cell's health. "So I hope at least one well will work," she said so that she could use that wells' "condition to do my real experiment." After 48-72 hours, Eleanor checked the cells by staining them, collecting protein from inside them, and running a Western Blot to see which well (physical milieu) she created had silenced DNMT3a. Following this, Eleanor used the "well with the right condition" from her initial testing phase "to do the real experiment." She commented that "sometimes testing works, but the experiment doesn't. That's frustrating. It gives you a headache because you have to go back, and start over."

Eleanor's anticipatory caretaking practices epitomize the argument here that technicians do not work in reaction to what is before them. They anticipate. Eleanor, and other career technicians, strategically plan and negotiate how and when to interact with the biological world. Each develops a "feeling for the organism" and, as they interact with the material world, it is necessarily transformed by their very interactions. On an intuitive plane, the process of caring for cells that once belonged to an entirely different organism became for Eleanor *her* cells, her "babies." Thus, like an attentive parent who identifies with an organism, she learned how to optimally care for them, including how to feed, change, passage, and cultivate them. In this particular example, whether or not the cells ate palmitate was tantamount to decide if her cells were healthy and happy. Failing in any of this, her cells would not only die, but experiments would collapse, papers would not be published, and lab doors would eventually close. "No funding, no job," she reminded me.

Temporary technicians: Searching quickly for "some" positive story. Temporary technicians had two major roles in the Lab. First, they supported the career technicians and, therefore PIs, at the bench and, second, they helped represent the Lab in conference presentations with scientific posters they had co-authored. International Medical Graduates (IMGs) and the Medical Fellows need to fulfill their own medical school residency obligations that require them to generate scientific abstracts for conferences and, hopefully, produce at least one publication. For IMGs in particular, they need three abstracts (conference posters) during their brief tenure in the Lab while they also study for the US medical licensing exams. A revolving question at the bench and, in concert with PIs, is "if the data is good, this can be your abstract for next year." In contrast to career technicians who have different long-term stakes in the Lab's continued success, these technicians simply need to "get *some* story to tell."

And these stories are inherently different than those cultivated by career technicians. Temporary technicians' studies were descriptive; they did not study causal epigenetic mechanisms like, for example, DNA methylation in gene knock out mice. Under their work conditions, I was told that "you don't get to do groundbreaking research. It is incremental, bits of research that are very descriptive. It is not cause and effect. You cannot aspire [to publish in] *Science* because the work is not revolutionary or major." But, at the same, "it also doesn't have to be," they said. In their words, then, "a mid-level journal is okay." What these technicians needed to show was some modicum of evidence that they contributed to basic science research in order to graduate. While this is a rational approach to their work in the Lab, it nonetheless shapes the larger scene of obesity and metabolic syndrome studies and there is an epistemic effect, if minor, in the proliferation of "normal science" (Kuhn, 2012). The temporal positioning of their time in the Lab literally shapes the course, content, and trajectory of anticipatory caretaking in a more conservative direction (compared to career technicians) in obesity and metabolic studies.

During one summer, for example, I observed temporary technicians as they built on the Lab's existing maternal-BPA experimental system to create their own trimmed down experimental models. Building on the Lab's previous work, the IMGs (Maryam and Veronica) and Medical Fellow (Julia) examined glucose and insulin signaling in the liver and skeletal muscle of rats. As a result, they studied biology and physiology while learning to handle tissues and cells for which they had little experience. Carrie sacrificed rats at day one of life, 21 days of life (at time of weaning and end of direct BPA exposure), and again at 10 months (adulthood) so that the temporary technicians could examine effects at different life stages. Empirical questions included whether or not deleterious health effects were present at day one and day 21 but, more

importantly, whether any effects persist beyond birth and weaning and whether effects might vary by sex.

In order to quickly get "some story," then, temporary technicians focused on the hypothesized effects of BPA exposure on glucose and insulin signaling in the adult liver and skeletal muscle because, like fat tissue, they are relevant to metabolic health.[19] The liver plays a central role in controlling glucose production (a vital source of energy) and the regulation of insulin secretion. Likewise, skeletal muscle is an important organ for glucose homeostasis. According to Batista et al. (2012), even short-term treatment of male rats with low doses of BPA "slows down whole body energy metabolism and disrupts insulin signaling in peripheral tissues" like skeletal muscle (p. 1). In translational research terms, this data helps address risk of diabetes and insulin resistance due to BPA exposure. Because they need a relatively quick and, therefore safe study, Maryam, Veronica, and Julie explored the possible effects of early BPA exposure on male and female rats, but with an emphasis on *timing* at weaning and adulthood.

Over the course of a few months, Maryam, Veronica, and Julia were indeed able to get "positive results." They concluded that maternal-fetal BPA exposure has sex and tissue specific effects on adult rats. For example, adult males had impaired glucose tolerance and higher insulin secretion. This finding is also reflected in the scientific literature, but at present the underlying mechanisms that produce sex-specific outcomes are unknown (see footnote 7). Lab technicians, and others, theorize that the sex difference may be explained by BPA's interactions with estrogen receptors and estrogen-signaling pathways (Shanle & Xu, 2011). At the time of weaning, the main site of disruption in insulin signaling was the liver, but in adulthood it was the skeletal muscle. Glucose transporters such as "GLUT" proteins were disrupted at both weaning

[19] My research site is not unique in studying the effects of BPA on metabolism. See, for example, Mullainadhan, Viswanathan, and Karundevi (2017) and Batista, et al. (2012).

and adulthood, and they reflected general principles of the Developmental Origins of Health and Disease. Based on their "positive findings," each of the temporary technicians authored conference posters and subsequently published their work with PIs.

For temporary technicians, in particular, the subject of authorship carries a different valence than it does for career technicians. While career technicians are often named third or fourth author, there is an understanding that authorship goes first to the PIs because career technicians work in exchange for a wage. For temporary technicians who are not paid, their compensation is in the form of shared first and second authorship. On occasion, this arrangement raises questions among technicians about "who" counts as a scientist and how, therefore, they are publically recognized. At a minimum, technicians at this Lab were the primary producers of data, whether that data was defined as "raw" or not. It is evident "their hard work and enthusiasm," went into caring for the Lab's experimental systems, as well as each other as they co-produced laboratory knowledge together. In a contemporary lab such as my research site, there is no clear distinction between PIs and technicians in terms of intellectual labor, but there is a clear distinction about forms of anticipatory caretaking of the data that is itself shaped by one's temporal positioning in the Lab.

Conclusion

As I have argued in this chapter, biomedical research in the Lab, and at academic, basic science labs across the country, is dependent on a class of highly educated scientific workers who are vulnerable to a precarious funding landscape as well as political shifts in immigration status. The ethnographic data on "technicians" in the Lab reflect larger trends in the biomedical workforce as documented by the National Institutes of Health, the National Research Council, and the National Academies of Science. Based on national-level, biomedical workforce data, "it

is difficult to consider the U.S. biomedical research enterprise without acknowledging the critical role played by scientists from outside the U.S." (NRC, 2005, p. 6). Yet these same national committees also acknowledge that we know very little about non-citizens who help constitute our biomedical workforce and how this might shape the overall pace and course of scientific endeavors.

My observations suggest that technicians' political and economic vulnerability translates into occupational stability for the PIs. While all technicians (and PIs) in the Lab experience economic precarity because of grant-based funding, there is variation in caretaking practices that depends on their occupational status as either a career or temporary technician. In addition to the scientific knowledge technicians bring to the Lab, I have argued that they develop what I call anticipatory caretaking practices in the course of doing experimental science. All technicians "search around the unknown for direction," but in ways that are subject to their own temporal positioning in the Lab. Having a "feeling for" something is contingent on one's own positioning as either a career or temporary technician. Academic career technicians are much more likely to push conceptual and material interventions in new ways as a form of taking care of the Lab's experimental systems. Temporary technicians, however, are more inclined to pursue conservative research topics within established paradigms because they need quick, positive results that will reinforce their own professional development timelines, performing what Thomas Kuhn (2012) referred to as "normal science." For both, the knock-on effects of anticipatory care practices shape the finished knowledge product. It is not that negative results are uninteresting for the Lab (because they do raise additional empirical questions), or that they are somehow perceived as a poor reflection on the Lab's reputation or its citations. Rather, it is

the fact that the pressure to produce positive results is built into the systems of science-based immigration and medical professionalization, which non-citizen "technicians" are subject to.

I have found that technicians are not only producing "all the data" for PIs, they are doing so within larger structural forms of power that enter into and shape experimental systems to begin with. It is impossible for me to say exactly what the impact of these forms of precarity are on the overall production of scientific knowledge in obesity and metabolic studies, but I have observed that the overall production of findings in the Lab, and especially the production of positive findings, are profoundly and intimately connected not just to technicians' economic livelihood but to citizenship itself.

Chapter 3

Biological Labor and the Biomedical Production of Environmental Subjecthood

> Even when we are asleep, our bodies are performing biological labor in our circulatory system, respiratory system, even at the cellular and molecular levels of metabolism and gene expression…A body that never stops laboring is also a biology defined by production, a species being defined by its own particular type of labor.
> Eugene Thacker, 2005, p. 117.

> Vitality can now be decomposed, stabilized, frozen, banked, stored, commoditized, accumulated, exchanged, traded across time, space, across organs and *species*, across diverse contexts and enterprises in the service of both health and wealth.
> Nikolas Rose, 2007, p. 3, my emphasis.

In this chapter, I draw on my observations at University Lab (Lab) to argue that epigenetics and Developmental Origins of Health and Disease (Developmental Origins) of obesity offer a "window on the ways that environmentally defined [biomedical] subjects are constituted" (Olson, 2010, p.171). In making this claim, I build on Hannah Landecker's (2007, 2010) argument that "altering any kind of biology…is to alter what it is to be biological" (2007, p. 223-235). Specifically, "to be biological" at the Lab is to be fat, to eat fat, to store fat, and to reproduce fat offspring because of uterine and neonatal exposure to either a high-fat diet or the ubiquitous chemical bisphenol-A (BPA) (p. 235). In short, the Lab experimentally studies the social nature of physiological functioning, i.e., how chemical and nutritional exposures change biology and physiology in animal models of human health. Their work reflects the larger concern raised by environmental obesity researchers who argue that we live in an "obesogenic" (Grün & Blumberg, 2006) landscape specific to the "Anthropocene" era (Crutzen & Stoermer, 2000). Hence my assertion here, and in chapter 1, that we are witnessing an environmental turn in obesity and metabolism studies.

How, then, does the Lab investigate the physiological functioning of social nature? How do mice and rat models of human health become environmentally constituted by the altered nutritional and chemical landscapes of the Lab's "experimental systems" (Rheinberger, 1993, p. 444)? What are the nutritional and chemical epigenetics of programming in obesity and metabolic studies? And, what sensitive windows of development are most salient to studying the effects of exposure on long-term health outcomes? To answer these questions, I illustrate *in vivo* and *in vitro* practices with model animal organisms in two of the Lab's experimental systems: maternal-high fat and maternal-BPA. For *in vivo* studies, I focus on characterizations of health that investigators and technicians derive from living animals, and for *in vitro* experimentation, I focus on the cellular and molecular work done with animal cells and tissues. These elements evidence the broader claim that there are new forms of "what it means to be biological" in an age of anthropogenic obesity (Landecker, 2007, p. 223-235). As a result, I argue, the Lab produces environmental, biomedical subjecthood and the epigenetically at-risk individual (Waldby, 2002; Rose, 2000).

In addition to showing how the Lab produces environmental, biomedical subjecthood, I further argue that animals and their disaggregated bodies are part of the production process in which laboratory facts are made. I therefore argue that to "be biological" at the Lab not only entails a reorganization of animal biology and physiology in environmental terms, but it also means that animal subjects (biomass) physically labor alongside the "fluid properties of human and nonhuman agents in powerful interactions and productions" in the build-up of scientific knowledge and capital (Landecker, 2007, p. 223-235; Rapp, 2000, p. 211; Bourdieu, 2004). This argument builds on questions of what it means to labor in an era in which scientific capital (Bourdieu, 1998, 2004) accumulation often depends on the molecular appropriation and

extraction of use and exchange value from non-humans (Marx, 1867). In short, I argue that "innovation value" (Cooper & Waldby, 2004, p. 3) derives as much from the labor of animal biomass as it does humans enrolled in clinical trials. Model animals, no less than humans, are mined for novel disease patterns that investigators use to create valuable scientific data. The labor of animal "vitality" (Rose, 2007, p. 3) is harnessed to produce use and exchange value for the obesity market, and to reproduce symbolic (reputational) and economic (grant) capital for the Lab's investigators (Bourdieu, 2013). The fact that it is non-humans who are enrolled in a labor relation to scientific capital makes no difference in the material outcome: valuable data for the obesity market is extracted from their biological labor in the laboratory. Therefore, in addition to showing how the Lab's produces environmental subjecthood, I also argue that animal biomass (as living elements of experimental systems) is itself entrained to be productive of scientific data within a very specific economic framework informed by a national health crisis.

To illustrate these points, I organize this chapter as follows: First, I draw a broad picture of the Lab's Developmental Origins systems used to study adiposity and appetite. Second, I explain two experimental systems, maternal high-fat and maternal-BPA, in greater detail. Within each system, I characterize the animal models, their routine care, and illustrate experimental treatments via food and BPA exposure delivered through the animals' water supply. I organize ethnographic observations by scale, meaning I move from whole body organism (*in vivo* work) to cells in a flask (*in vitro* work). Through each layer of intervention, animal biology is reorganized by exposure to food and a toxin; their biology serves as a model for human biology. It is particularly in the epigenetic work with transcription factors (proteins that directly bind to genes and regulate the gene's transcription to messenger RNA) that the Lab gets most mechanistic and deterministic about the long-term effects of harmful exposure on offspring.

Third, I analyze my observations in the context of literature drawn from science and technology studies, feminist technoscience, and sociology of work. In this section, I use interdisciplinary literature to help illustrate ways in which the Lab produces an environmental model of obesity through maternal-fetal theories of disease causation. I also detail recent scholarship on theories of labor after biotechnology, i.e., how use and exchange value are extracted from biological matter in a post-industrial context to argue that nonhuman subjects live and labor in specific ways within the Lab's experimental systems. The value of biomass is realized as scientific data that circulates in the obesity market. I conclude the chapter by considering what it means to (bio-)medicalize obesity through the lens of Developmental Origins and epigenetics.

Developmental Origins Models of Appetite and Adiposity

Broadly speaking, the Lab employs multiple developmental "experimental systems" to study two primary outcomes: appetite regulation and the development and function of adipose tissue (Rheinberger, 1997). The systems vary by treatment source: a high-fat "Western" diet or BPA, the timing of exposures, developmental and metabolic pathways associated with appetite and adiposity, and proteins believed to modify gene expression and, ultimately, the downstream function of cells, tissues, and organs.

In the Lab's nutrition-based experimental systems, a maternal obesity/high-fat diet model was used to elucidate mechanisms that program the hypothalamic appetite pathway in a rodent model of human health. This model is also used to identify mechanisms that govern adipogenesis (the process of cell differentiation in which preadipoctyes become adipocytes, i.e., mature fat cells) and lipogenesis (the process of fatty acid and triglyceride synthesis). Further, two models of food restriction were explored to study the effects of a calorie-restricted diet (via the thrifty

phenotype hypothesis of fast growth catch-up for low birth weight pups) on the same two outcomes.[20] Interestingly, the Lab also began to study the possibility of re-programming a balance of neurons in the hypothalamic appetite pathway by changing nutrition conditions in infancy.[21] The Lab's work in the "appetite remodeling" experimental system is explored in detail in chapter 4.

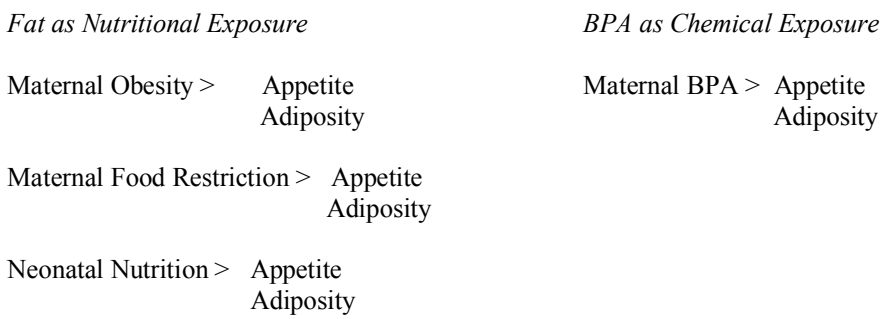
In the maternal bisphenol-A systems, appetite and adiposity are both studied because it is widely believed that BPA interferes with hormone action, including perturbances in adipocyte cell number, size, and function (growth of adipose tissue), and the hypothalamus-pituitary-adrenal gland axis (appetite regulation). Animal studies show that BPA exposure is associated with higher body weight and shifts in body fat composition. Among humans, there is also an increase in breast and prostate cancer and reproductive dysfunction, including infertility, early menarche, and sex development disorders (Braun 2017; Janesick & Blumberg, 2012).

To help visually illustrate the Lab's experimental systems, below is a schematic representation defined by exposure on phenotypes:

[20] The theory of Developmental Origins of Health and Disease was described in chapter 1. Recall that both low and high-birth weight offspring are said to be at higher risk for adult obesity. Thus, there is a U-shaped curve to birth weight and health outcomes.

[21] If a low-birth-weight baby/pup is food restricted to the degree it cannot experience a "catch-up growth" period, which the thrifty genotype hypothesis predicts (see chapter 1), then there is also the potential to reduce maternal burden because of the temporal framework of interest shifts from pregnancy itself to infancy (however, lactation can then be reified as a site of maternal burden).

Figure 3.1. Four Models of Appetite and Adiposity in Developmental Origins Research



Animals are studied at different times throughout their life-course (e.g., fetal, neonatal, at weaning, adult, and during pregnancy) and at different scales (e.g., *in vivo* and *in vitro* studies). Technicians perform *in vivo* studies during pregnancy, the lactation and weaning periods of newborn pups, adult life, and the offspring of (previously) exposed adult mice and rats to explore intergenerational effects, if any, of maternal obesity and BPA on offspring. Relevant measures include amount and function of fat cells as a measure of adipose tissue (obesity), food intake as a measure of appetite, and glucose tolerance as a measure of insulin insensitivity, particularly in the BPA systems. Animal tissues, which can be stored for years in -80 freezer conditions, include fat, liver, skeletal muscle, and brains of sacrificed animals. These tissues are studied using *in vitro* techniques to shed light on changes at the level of organs, including skeletal muscle, brain, liver and adipose tissue. At the smallest scale, *in vitro* work is performed with both fresh and frozen cells and tissues to examine the regulatory effects of transcription factors (specific proteins) on genetic expression. The level of granularity thus moves from broader, descriptive assessments provided by *in vivo* studies of whole animals, to the more mechanistic *in vitro* analyses of tissues, cells, hormones, and proteins. A principal investigator once explained the practice of scaling environmental insults along temporal dimensions as follows:

> Carrie, [the animal technician], monitors the animals, and what we start with are phenotypes like body fat measurements taken through DEXA scans, glucose tolerance,

> and blood pressure. We call these the phenotype of the animal. And we also collect blood samples and tissues where we say okay what are the insulin levels, what are the corticoid steroid levels, what are the leptin levels, and all of that goes into the plasma data, and that is part of characterizing the phenotype too.
>
> Once we get that, suppose we say okay they have increased adipose and that's when we get mechanistic and say okay what is driving this increase in adiposity? This is where the molecular and cellular work comes in. So Eleanor [the most senior career technician] looks at adipose, Olivia [the second career technician] looks at the brain. We are investigating mechanisms to see why they are eating more, storing more fat. Then, you characterize the molecular stuff like the protein data and ask which protein levels are up or down.
>
> Once you get a handle on that, then you get into further mechanisms like why have the NPY neurons [located in the arcuate nucleus], or the PPAR [a protein-coding gene], which is in the adipose tissue, increased. So then you say what regulates this? What are the transcription factors, what is upstream? And we go right to the stem cell level.

In a very practical sense, then, stages of empirical investigation are shaped by findings that result from inductive *and* deductive reasoning. Beginning with the most descriptive detail provided at the level of whole animals (*in vivo* studies), the Lab delves into the particularities of molecular mechanisms in adipogenesis, lipogenesis, and altered glucose states. In the course of these studies, the productive and reproductive labor of biomass contributes value to the obesity market as raw material, or data, that is transformed ultimately into scientific capital. This data, in turn, is advocated as a necessary contributor to the global fight against obesity and metabolic syndrome. Indeed, a reading of the literature on the environmental epigenetics of pregnancy shows that authors begin and conclude their publication with a translational call to apply their data toward better and earlier preventative and therapeutic approaches to metabolic disorders.

Now that I have drawn a broad picture of the Lab's experimental systems, I go into further ethnographic detail in the pages below. I elaborate on the maternal obesity model on programmed adiposity. Using two examples from this system, I will show how the Lab's practices with gene silencing, on the one hand, and the transdifferentiation (functional

conversion) of white fat cells into the behavior of brown fat, on the other, reveal how the vitality of biomass is harnessed in the name of human health and therefore made to biologically labor differently in order to become environmentally constituted subjects. In the second experimental system, the maternal BPA system for programmed adiposity, I will to show how environmental subjecthood is produced through the lens of chemical exposure. For both experimental systems, the biology and physiology of animals is quite literally caused to function differently (i.e., to become fat) as a result of exposures. In both systems, biomass is differently and environmentally constituted.

Experimental system #1 - Maternal high-fat diet and programmed adiposity. The Lab used a classic strain of laboratory rats called Sprague Dawley®, which were purchased from Charles River Laboratories, a long-standing breeder. The Sprague Dawley® rat, created in 1925, is a widely used animal model in biomedical research in particular for *in utero* studies of reproductive toxicity and nutrition because of its similar metabolic pathways to humans. Due to the rat's scientific popularity, a British research firm goes so far as to describe it as the "workhorse of reproductive endocrinology" (Rothrock, 2017). Another breeder of the Sprague Dawley® rat highlights—in its technical data—that in addition to color (white) and physiological parameters, females are "good breeders" with "strong maternal instinct" (Janvier Labs, 2018). This may or may not mean dams (mothers) are less likely to commit infanticide than other strains of rats.

The average life expectancy of a Sprague Dawley® is three years, but one breeder characterizes the "economic reproductive ability" of both males and females as only six-to-nine months (Taconic, n.d.). Reproduction can occur as early as week six, and gestation lasts 21-23 days. A typical litter consists of eleven pups. They tolerate high-fat diets, and they have similar

hemochorial placentation (fetus establishes its own circulatory system) as humans. Importantly, rats are nocturnal and therefore only mate (and prefer to eat) when the lights are off. In a humorous recollection of how her initial set-up of a rat colony was failing, Dr. Moore said that she and others were confounded by the fact that their rats were not reproducing. A security guard who walked the rounds at night inquired as to why no one turned off the lights in the animal room when leaving for the night. Apparently, no one at the lab had realized the lighting cycle had been tripped. Lastly, epigenetic tools (e.g., microarrays, gene knock-down assays, and antibodies that are used for tissue specific gene profiling to measure protein expression) are widely available for mice in particular.

In a dedicated animal housing room (vivarium) located a building away from the PIs' office, there were several hundred ventilated cages nested on top of one another, from floor to average adult height, containing over one hundred animals, both rats and mice, individually housed. Within this large space, Carrie, the animal technician, monitored the air temperature, humidity, sounds, 12-hour light/dark cycles, and "environmental stimuli" (simple toys) for the animals in order to support their physical wellbeing and to reduce environmental stressors as much as one can in an extremely artificial milieu. For example, the fact that Carrie conducted her work during the day, with the lights on, disturbs their natural, nocturnal circadian rhythms and, therefore, she created the data (blood pressure in particular) out of the labor of a sleepy rat, at least to some degree.

In addition to routine maintenance and measurements, Carrie tended to animal feeding and reproductive labor, which is highly orchestrated in an experimental laboratory. Regarding animal feeding, the Lab used, from an array of possible diets that vary by composition (sugar, fatty acids, carbohydrates, protein) and percentages of individual ingredients, a control diet

containing 10% fat (control) and an experimental high-fat 60% (HF) diet purchased from a well-known external vendor of obesity diets. The HF "food" is a pink pellet that resembles a tube of lip balm and is so greasy it has to be shipped on dry ice to prevent melting. An HF diet begins with rats before and during gestation. They are provided water and fed *ad libitum*, i.e., without restriction, and are assigned to either the control or HF diet for eight weeks before pregnancy. Within this experimental system, pregnant rats will eventually be sorted into four exposure groups that are circumscribed by the timing of pregnancy and lactation. The four exposure groups, then, are control (pregnancy)/control (lactation), HF/HF, HF/control, and control/HF. The biological and physiological effects of "food as exposure" on animals are shown in the molecular and cellular work described below (Landecker, 2011, p. 169).

The experimental point of switching diets during lactation is to address questions around the plasticity of epigenetics that exist in tension with the determinism of programming; questions like: Once harmful exposure has been removed (i.e., pups are switched to the HF/control diet), how does the animal's metabolic profile change, if at all, as a result of an improved nutritional environment? What happens in subsequent generations is also materially explored to understand better whether or not epigenetic marks persist in the germline (which will develop into eggs and sperm) in the *absence* of harmful exposure during pregnancy and lactation. In other words, do the effects of what "grandma" ate persist into the third generation? To this end, rats are further bred to study the transgenerational effects, if any, of control and HF diets on long-term metabolic health. Questions like these involve the biological labor of mother and her offspring.

Carrie collected multiple data points used to characterize the animals' phenotypes; the first stage of "getting a handle on what is going on." On a daily basis, she measured individual weight and food intake, and at postnatal weeks three and twenty-four, she performed an *in vivo*

DEXA (dual-energy x-ray absorptiometry) scan to record body composition of lean and fat tissue mass. She also measured blood pressure using a tail cuff and performed an anesthetized tail bleed procedure to draw blood for glucose tolerance tests. Although it may seem these are straightforward measurements, they are in fact time-consuming. During a day of observation with Carrie, it took more than three hours to obtain blood pressure for a handful of animals. The size and disposition of the animal (some are more physically active than others even though Sprague Dawley's® are noted for their docility) affect the ease or difficulty of obtaining measurements, and so does environmental stress. For example, although we sat in a quiet room adjacent to the vivarium, we noted that our talking and the fact that it was during their sleep cycle were likely stressing the animals, which complicated Carrie's task and affected the measurements she took. In more invasive and terminal procedures, she also collected plasma to measure levels of insulin, corticoid steroids, triglycerides, and leptin from several animals at three weeks of age and again at week twenty-four. To collect a relatively larger sample of blood (techniques yield varying amounts), one male and one female of each litter were additionally "deeply anesthetized" in a terminal procedure called cardiac puncture. After this, animals were dissected, and Carrie removed tissues and organs so that Eleanor and Olivia could begin the molecular and cellular work of analyzing fat cells, proteins, hormones, and metabolites extracted from the animal's biomass.

Sacrifices are made at various stages of life, from embryos to adulthood. Embryos (and therefore their mothers) are sacrificed on a specific gestational day in order to halt a so-called critical window of development and to collect tissue, organs, and derive cells from them. At the other end of the life course, adults are sacrificed to study possible long-term programming effects on their health. Brains, brown and white fat tissue, skeletal tissue, liver, and other

metabolic organs are scooped and stripped from animal's body according to research protocols. Immediately following dissection, the tissue is kept *alive* by either fixing or freezing it according to specific research needs. Cells can then later be derived from various tissues in order to measure protein expression, i.e., epigenetic expression of a gene.

In each of these steps, the type (control versus high-fat diet) of environmental exposure has a distinct temporal rhythm to it. Female animals labor, for example, through the course of experimental treatment from purchase or birth into a cycle of pregnancy, birth, lactation, weaning, adulthood, and pregnancy once again (if they are not sacrificed, of course) to produce new generations. Within these different fragments of time, shaped by the animals' own developmental and reproductive rhythms of their life course, snapshots of environmental exposure are rendered first through *in vivo* measurements of weight, food intake, blood pressure, and invasive (and some deadly) techniques for drawing blood.

Once the animals are sacrificed, multiple techniques of cell culturing are then performed to help fill out the phenotypic picture. In order to identify molecular mechanisms, animal cells and tissues—from stem cells in newborns and adults, white and brown adipose tissue, bone marrow, skeletal muscle, plasma data—are all used in *in vitro* studies to assess how the maternal nutritional environment affects organ development, cellular signaling responses, and epigenetic modifications in offspring.

Before detailing examples of the Lab's molecular and cellular work with maternal obesity, I will describe certain basic cell biology and epigenetic mechanisms: First, stem cells are non-specialized cells from which all other cells with a specialized function are derived, such as skin, hair, and heart cells. With appropriate transcription factor and other cellular signals, stem cells divide to produce daughter cells. These cloned daughter cells will then either become new

stem cells, in a process called self-renewal, or they will become cells with specialized functions. The two most common types of stem cells are embryonic (a 3-5 day old blastocyte that has not implanted) and adult stem cells located in adult tissue such as fat. Embryonic stem cells are called pluripotent because they self-renew or become specialized. Mesenchymal stem cells (MSCs) are a type of stem cell that can be isolated from tissue, including adipose and others. These stem cells (often called somatic cells to distinguish them from the germline and embryonic stem cells) can also self-renew or differentiate into different lineages (except blood cells) in what is called the mesengenic process, including fat cells, bone cells, and cartilage used to support the formation of connective tissue.

Second, stages of adipogenesis include stem cell proliferation (replication of the cell), commitment (to a particular tissue), lineage progression, differentiation (into the dedicated cell type), maturation, and terminal differentiation into, in this example, mature white and brown adipose tissue. How commitment and differentiation occur -how a preadipoctye commits to the adipose lineage and then differentiates into an adipocyte- is a hot topic of research for labs across the world. The exact mechanisms are not yet known, but, in general, signals inside and outside of cells control the differentiation process. Internal signals are controlled by the cell's genes, which carry DNA that contain instructions for cell structure and function. External signals include: "chemicals secreted by other cells," "physical contact with neighboring cells," and molecules in the cell's "microenvironment" such as "nutrients and growth factors in the fluid surrounding a cell…which play an important role in determining the characteristics of the cell" (National Institutes of Health, 2016, Stem cell basics II section). The interaction of signals during differentiation leads to epigenetic marks, which, in turn, affect how genes are expressed.

Epigenetics is the addition of information (text) on top of an underlying sequence of letters or base pairs called (for short) A, C, G, and T that make up the DNA (deoxyribonucleic acid) of a gene. Signals from the environment such as maternal nutrition activate proteins called transcription factors, which themselves are encoded by genes. According to the historian and philosopher of science Evelyn Fox Keller (2015), "today's genome, the postgenome" is more of a regulatory device for the production of specific proteins than a collection of genes that deterministically form traits (p. 25; Keller, 2014). Indeed, the logic of epigenetics asserts that exogenous and endogenous environmental exposures alter the expression of genetic material, amplifying or suppressing genes over the course of one's lifespan without changing the underlying sequence of genetic code. As a result, epigenetics "represents the new age of genomics in which nature and nurture are seen to interact in profound ways that overturn the old reductionism and determinisms of Watson and Crick's genetic code" (Stevens & Richardson, 2015, p. 4).

There are three main epigenetic mechanisms: DNA methylation, histone modification, and non-coding RNA (Janesick & Blumberg, 2011, p. 257). Each of these epigenetic mechanisms modifies how "the text" is read out. DNA methylation, for example, refers to the addition of a methyl CH_3 group to DNA, rendering it unreadable. Histone modification refers to how open or closed chromatin wrapping around DNA itself is. Chromatin consists of DNA packaged around histones; and the more open the packing around DNA is, the more susceptible it is to have transcription factors (other than those coming from the cell's DNA) which affect how the DNA is intended to be read out/transcribed. The more tightly packed it is, the less likely external signs will modify DNA. Non-coding RNAs (ribonucleic acid), both long and short, are associated with gene silencing and regulation. They are transcribed from DNA (which is why

they are called non-encoding) as part of the central dogma in genetics whereby DNA (transcription) makes mRNA (translation) builds protein, but they are not translated into proteins. However, they are considered epigenetic because they do not change the underlying DNA sequence and because they are post-transcriptional regulatory mechanisms that affect gene expression. In epigenetics, "biology is itself constituted by those interactions [between genetics and cellular environment]" (Richardson, 2015, p. 29). Neither the environment nor biology are imagined in binary terms within the logic of epigenetics and yet, precisely in order to do research, PIs must formalize differences between the two.

Now that I have explained some basics of cell biology and epigenetic mechanisms, I illustrate two examples of molecular and cellular work in the HF maternal model through two technical practices: gene silencing via transcription factors associated with adipogenesis, and the transdifferentiation (lineage reprogramming) of white adipose tissue for the purpose of altering its biological function to act more like brown adipose tissue. It is through molecular and cellular work that the Lab really tries to determine causal mechanisms. The *in vivo* work described above is the equivalent of a start position on a board game. It gives initial clues, and is therefore when they start to imagine connections between various biological pathways.

During an early morning meeting at the Lab, I sat with investigators and technicians as they mapped out possible directions to take with a transcription factor called HES-1[22], a protein involved with cell proliferation and differentiation of preadipoctyes into mature adipocytes. Epigenetics is a controlled selective way to show how gene silencing might contribute to the

[22] Gene names are written in italics, and the transcription factors they encode for are not.

phenotype of fat storage. While the technician Eleanor explained some initial results with gene-silencing work, an investigator elaborated on their intentions[23]:

> We've said they [animal tissue] store more fat than the controls, but what is driving that? We find in stem cells [MSCs] that they produce more fat cells…there is an increased proliferation of them. One of the factors is called transcription factor HES-1, so in order to prove that it is HES-1 driving the increase, we are going to suppress the gene's expression. That experiment is called siRNA or silencing the gene. If you silence the gene, then we should see no proliferation. To prove that this is the relevant factor, one way to prove it is to knock down the gene, or you overexpress it or use pharmacological modulators that either activate or suppress it. This is the stage where we are trying to prove that HES-1 is the one. (Personal communication, February 2, 2015)

HES-1 might indeed be an essential factor in adipogenesis. For example, according to Ross, Rao, and Kadesch"s 2004 study, "Hes-1 has two roles in adipogenesis: one promotes adipogenesis, possibly through the down-regulation of inhibitory proteins…and the other inhibits adipogenesis at a step prior to the induction [of the 'master regulators' of adipogenesis] C/EBP and peroxisome proliferator-activated receptor (PPAR)" (p. 3505; Farmer, 2007). This is an important contribution because it provides a possible explanation, "at a step prior" to differentiation, as to how MSCs proliferate and become pre-adipocytes. Understanding the commitment phase of MSCs to preadipoctyes, then, illustrates a novel and burgeoning area of adipocyte biology and reflects the particular work done with transcriptional gene silencing at the Lab, as shown in the passage above.

In order to practice gene silencing, adipose tissue is extracted from laboratory animals (from both control and experimental groups) at different life stages between early age and adulthood. Even though fresh biomass is considered trickier to handle by technicians, it is preferable to buying, for example, a well-known preadipoctye commercial cell line such as 3T3-L1 or 3T3-F422A (a preadipoctye cell line established in the mid-1970s). Commercial lines are

[23] Very similar experimental studies are done in other labs. See, for example, Ross, Rao, & Kadesch (2004) who also studied HES-1 concerning adipogenesis.

suspended in cell culture for years and, as a result, are often genetically and phenotypically different from their tissue of origin. While commercial cell lines are easier to proliferate (increase the cell numbers) through an *in vitro* cocktail of inducers, the cells show altered morphology because of their lifetime spent in artificial culture. In fact, technicians at the Lab hotly disputed how much passaging (expanding the number of cells in a flask) primary cells ought to be allowed to go through because the more passages they endured, the fewer cells resembled their original state. Therefore, it was culturally significant for this particular Lab to use cells derived from animals used in their *in vivo* studies.

In the HES-1 gene silencing study, the career technician, Eleanor, used a toxic chemical, what is referred to as a pharmacological knock-out, noting that the "the cells might not stand it" because HES-1 might itself be important to the cell and therefore attempting to silence it may also kill the cells. Using mouse cells cultured from white adipose tissue at different ages, Eleanor grew the cells in a nutrient-rich medium, seeding and separating them (controlling for number and density of cells). Within a few days of culturing the cells, she hoped to be able to identify cells under a microscope so that she could then induce the experimental knockout. She noted that silencing HES-1 with a toxin means that "you have to try a lot of different ratios of siRNA and the transcription chemicals" to see what works (personal communication, February 2, 2015). Eleanor used different ratios, from twenty to eighty percent, in order to figure out the right medium to keep cells alive while experimenting with them. Within twenty-four hours of siRNA treatment, she observed their reaction to the treatment and extracted proteins from the cells if they had not degraded. According to a PI at the Lab, while this technique is not new, "every factor is different, so you have to iron out the conditions, and that takes a lot of time. Sometimes, by the time you've ironed them out, you don't have any more cells and so you start all over

again" (personal communication, February 2, 2015). Assuming cells had not died, and proteins were successfully extracted from them, a Western Blot (technique described in chapter 2) was performed over the course of a week, and the results visually illustrated whether the gene was silenced and to what degree.

In another example of how the Lab's modulate animal biology, the PIs and technicians attempted to get white fat cells to 'act like' brown cells function (Cedikova et al., 2016). White fat tissue stores fat while brown fat burns fat through thermogenesis (the production of heat and burning of calories). Because of this functional difference between white and brown adipose tissue, brown fat is routinely regarded as 'better' because it burns rather than stores fat. This type of experimental practices at this Lab and in others is sometimes referred to as the 'beige-ing of adipose tissue. One experimental possibility is to increase the number of mitochondria (cytoplasmic organelles inside cells that are involved with metabolic pathways and adipocyte differentiation) in white fat cells because mitochondria itself it is thought to be a critical fat burning property in brown adipose tissue. In this vein, investigators discuss whether or not "we can convert properties" of white adipose tissue to be more like brown adipose tissue (personal communication, January 26, 2015). According to a PI, the white adipose tissue would still "be" white adipose tissue under these conditions, but it would newly possess "pseudo-brown characteristics" by burning off more energy through extra mitochondria. In this example of browning or beige-ing fat, we find that not only the material conditions of biological possibility are altered, but biology is itself understood as something akin to a chemical technology that can be destabilized and reconstituted into a new biological form: beige fat. If this form of altered biology ever succeeded here or elsewhere, the Lab considers it would be hugely profitable for the medical market.

In both of the examples above—gene-silencing HES-1 in order to curb adipogenesis, and the molecular attempts at browning white adipose tissue by pushing white fat cells toward an increase in cellular mitochondria—the Lab transformed biology via experimental means. In order to mimic human obesity within the maternal-fetal model of Developmental Origins and epigenetics, animals were exposed to a maternal high-fat diet, programming them for obese phenotypes. Subsequently, the Lab performed molecular and cellular work, studying transcription factors that regulate the cascading effects of exposure on developmental pathways. Early developmental pathways and their transcriptional regulators become subject to the "molecular gaze" of epigenetics and Developmental Origins models of health and disease. These processes reflect Rayna Rapp's observations (1999, p.192) that the "natural" and the "social" are entangled in laboratory practices. She also wrote:

> Laboratories do not accommodate objects like human chromosomes in their natural forms; they are not bound by their locations in the wombs of pregnant women or their natural chronology…When viewed through the lens of the laboratory, the 'chromosomes' produced by cell culture are not natural objects but cultural ones…they become knowable only through the myriad sociotechnical interventions which enable and produce them as objects of scientific investigation. In this sense, 'tissue culture' has a doubled meaning: It is at once a technique for growing cells, and an enrollment of biological material for making human meanings. (p. 213)

As with chromosomes in Rapp's study of amniocentesis, biological matter (tissues, organs, cells) is extracted, decontextualized, and disrupted from the temporal rhythms of a previously embodied life. Unlike Rapp's study, though, the work done at the Lab was for the larger knowledge market and was therefore part of the circulation of scientific capital, raising additional questions about capital accumulation and labor in biomedical modes of production (Haraway, 2008). The Lab's practices with gene silencing and the transdifferentiation (functional conversion) of white fat cells into the behavior of brown fat reveal how the vitality of biomass is

harnessed in the name of human health and made to labor in the material construction of an environmentally constituted subject.

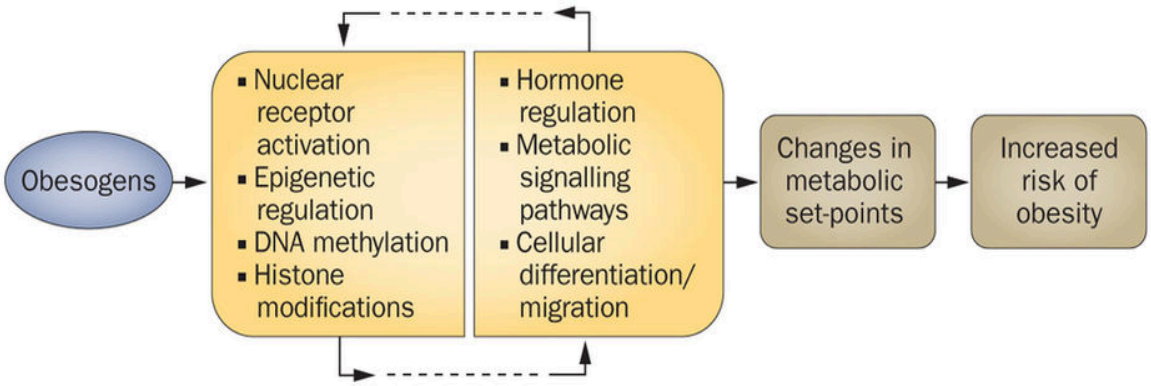
Experimental system #2 – Maternal-BPA and programmed adiposity. The Lab's work with the maternal-BPA similarly demonstrates how animal labor is harnessed to produce "innovation value" (Waldby & Cooper, 2004, p. 3) In this context, the harmful exposure is chemical rather than nutrition-based. Recent epidemiological and laboratory studies indicate that environmental toxins may have similar adverse effects as a maternal high fat diet on offspring programming of adiposity. This is because ubiquitous chemical toxins, such as bisphenol-A (BPA) act as endocrine disruptors, meaning the chemical "interferes with the synthesis, secretion, transport, metabolism, binding action, or elimination of natural blood-borne hormones that are present in the body and are responsible for homeostasis, reproduction, and developmental process" in organs that are hormone-sensitive (Environmental Protection Agency as cited in Diamanti-Kandarakis et al., 2009; Janesick & Blumberg, 2011; McAllister et al., 2009).

Endocrine-disrupting chemicals (EDCs) such as BPA have been termed "obesogens" by Felix Grün and Bruce Blumberg (2009b). According to these obesity researchers, "[o]besogens can be defined functionally as chemicals that inappropriately alter lipid homeostasis [stable maintenance of fat] to promote adipogenesis and lipid accumulation" (2009b, para. 3). Remarkably, more than 90% of Americans have detectable levels of BPA (National Institute of Environmental Health Sciences, 2017).

Thus EDCs, or obesogens, such as BPA, are viewed as an external source of harmful industrial exposure that becomes internalized through food and drink consumption and the use of plastics in food preparation and storage. As a result, biological processes that regulate

metabolism, adipose tissue function, cardiovascular health, and reproduction are affected; altering basic biology and therefore states of health and illness. In short, the effects of industry in the form of obesogens destabilize and reconstitute biology. The figure below helps illustrate the effects of obesogens on long-term health via the regulation of genetic expression:

Figure 3.2. Obesogenic Exposure Affects Genetic Expression and Health Endpoints



Note: Taken from Heindel, Newbold, & Schug (2015, p. 655).

As it was shown in the high-fat diet experimental system, the age of exposure to BPA is particularly relevant because it is during very early life that organs and physiological systems develop. Unfortunately, according to data from the National Health and Nutritional Health Survey, BPA has been detected in maternal serum, breast milk, placenta, amniotic fluid, and fetal serum (Woodruff, Zota, & Schwartz, 2011). Intrauterine programming is said to occur from environmental chemicals such as BPA that enter into the intrauterine environment, which, in turn, impacts postnatal outcomes in metabolism, endocrine systems, cardiovascular system, reproduction, and behavior (for example, appetite). As a result, the field of endocrine disruption describes early exposure and effects of EDCs and obesogens on biology and physiology as the "developmental basis of adult disease" (Diamanti-Kandarakis et al., 2009).

In the Lab's maternal BPA experimental systems, researchers examined altered cell number of adipocytes, modified adipocyte tissue function (tissue inflammation), modified gene expression, and the effects of BPA on re-setting the hormonal axes (between the hypothalamus, pituitary gland, and adrenal glands) in offspring. One hypothesis, similar to that of the maternal high-fat diet system, is that maternal exposure to BPA during pregnancy and lactation increases offspring adiposity and adipose tissue inflammation (a response to overnutrition that triggers inflammation which induces insulin resistance). If fat cells are more sensitive glucocorticoids (which BPA binds to), then fat cells may divide more quickly and may, therefore, contribute to the BPA animals becoming fatter (Emanuela et al., 2012). A second related hypothesis is that maternal exposure to BPA during pregnancy and lactation is also associated with metabolic abnormalities such as glucose intolerance in the offspring.

Once again using a rat model organism, the Lab mated males and females at nine weeks of age, and then exposed pregnant females to either the control group (BPA-free drinking water) or the experimental group (BPA is added to the water supply). After lactation, the pups were weaned and both groups of offspring were given BPA-free water supply. Both male and female pups were measured using DEXA (dual-energy x-ray absorptiometry), a non-invasive tool that produces data on body composition, including total mass, fat mass versus lean mass, and percentage of the overall body. Pups were measured at three weeks of age and again at six months. Once these measurements were gathered, the animals were sacrificed to study transcription factors associated with adipogenesis. Some of the transcription factors were the same as in the maternal high-fat model (because they are crucial to adipogenesis) and included PPARy2 and C/EBP. In a concurrent study, additional rats were sacrificed at different time points, from one-day-old to three weeks of age to test protein expression *in vitro.* In this manner,

the Lab grappled with the timing and effects of exposure, and also its absence, to assess whether or not effects persist over the offspring's lifetime. Lastly, technicians used the Western Blotting technique to measure protein expression in a target gene by transcription factors.

The Lab produced both negative and positive results in the maternal-BPA experimental system. Once, at weaning (3 weeks of age), there was no difference in basal plasma concentrations of glucose, insulin, or cholesterol for either male or female offspring. Additionally, there appeared to be no effect of BPA on maternal weight or food intake (appetite) during pregnancy and lactation. They did find, however, that BPA offspring had increased body fat mass at three weeks of age, resulting perhaps from enhanced adipogenesis and adipose tissue inflammation. As for metabolic abnormalities, females only showed an increase in triglycerides and altered fatty acid metabolism both of which are associated with increased risk of coronary heart disease. Male BPA offspring, on the other hand, showed altered glucose tolerance and insulin signaling relative to females. BPA appears to have sex-specific effects on offspring. Finally, both males and females had increased glucose-stimulated insulin secretion at six months of age. In sum, depending on sex, BPA offspring showed altered biological and physiological processes involved with a proliferation of fat cells. Whether the amount of fat tissue itself or the function of it matters most, is up for debate.

A wrinkle in these studies is the fact that control animals themselves had detectable levels of BPA in their plasma. This points to the degree to which even animals bred in highly controlled environments are already environmentally constituted by harmful exposure; something that is not a small problem for experimenters since “getting measured is not a trivial issue” (personal communication, May, 10, 2015). One PI informed me that “people get worried about the results” because there is no “standardized method of measuring BPA” (personal communication, May,

10, 2015). Nonetheless, the Lab attempted to minimize troubles by using BPA-free cages, glass water bottles, tinkering with the experimental dose of BPA, and even monitoring the rubber nubs on water stoppers to make sure rats were not nibbling on them. Still, as their results indicated, it is impossible to avoid exposure even in so-called control animals.

To summarize, when biomedical investigators, such as those at my research site, intervene on nonhuman biological actants, they do so from the position that fat cells are "living technologies" that can be harnessed, modified, and upgraded through the right diet and pharmaceuticals if only mechanisms that lead to increased adiposity are found (Landecker, 2007). For Landecker, understanding the history and practices of cell tissue culture provides an example of "how humans have come to regard and interact with living matter through the framework of life as technology" (p. 2). In her example, "techniques of plasticity and temporality" associated with biotechnology "changes what it is to be biological" (p. 233). By extension, in a maternal-fetal model of health and disease, the underlying "plasticity and temporality" of epigenetic and Developmental Origins logic points to how the environment changes what it means to be biological. Whether one defines the environment as internal (the womb) or external (food, BPA), the central biomedical site of "power-laden interventions" in these scientific models is in a gene-environment interaction between mother, fetus, and the broader nutritional and chemical landscape (Olson, 2010, p. 181).

In both of the Lab's experimental systems, I have shown that "what it is to be biological" at the Lab is to be environmentally constituted by diet and chemical exposure (Landecker, 2007, 2011). As a result, I have argued that animals are made to live life differently and their bodies are made to labor in particular ways as a direct consequence of nutritional and chemical exposure during "critical windows" of development (Burggren & Mueller, 2015, p. 91). The effects of

exposure on biological systems, and therefore embodied labor, include increases in BMI (excess adipose tissue), enhanced appetite (perturbations in the hypothalamus-pituitary-adrenal gland axis), altered glucose states associated with type-2 diabetes (insulin resistance), and changes in cardiovascular health (increased blood pressure and triglycerides). That animals are made to live and labor under these environmental conditions to produce an environmental, biomedical subject raises two questions I analyze below in conversation with interdisciplinary literature drawn from science and technology studies, feminist technoscience, gender, and sociology of work.

Implications for the Social Studies of Science: the Meaning of Biological Labor in the Production of Environmental Subjecthood and the Limitations of Biomedicalization in the Environmental Epigenetic Era of Health and Illness

In this section, I consider two questions: First, what does it mean to labor in an era in which biomedical capital accumulation depends on the molecular appropriation and extraction of use and exchange value (Marx, 1867) from *non*-human biomass? In other words, what are the contemporary labor dimensions of productivity in the Lab's experimental systems? Second, because the logic of epigenetics and Developmental Origins shifts the perimeters of disease causation including sources and timing of exposure, what does it mean to biomedicalize obesity (or health problems in general) in an environmental, developmental framework?

Capital is a defining theoretical construct in sociology. In science and technology studies, scholars have redefined core capital-related concepts such as "use-" and "exchange-value" to analyze vast shifts in the material conditions of capital accumulation specific to biotechnology and biomedicine in the 21st century (Marx, 1867). The "conjuncture of economic action and contemporary biotechnology" that characterizes the late 20th and early 21st century has been summarized by Stefan Helmreich (2008) as follows:

> Scholarship in the social and cultural study of biology has suggested that in the age of biotechnology, when the substances and promises of biological materials, particularly

> stem cells and genomes, are increasingly inserted into projects of product-making and profit-seeking, we are witnessing the rise of a novel kind of capital: biocapital. (p. 464)

In accordance with this revision of capital accumulation, science studies scholars today trace historical shifts in how the "appropriation of living nature" and the (re-)production of capital occurs at the molecular level (Helmreich, 2008, p. 463; Waldby, 2000, 2002; Waldby & Mitchell, 2006; Ritvo, 1995; Franklin & Lock, 2003; Rajan, 2006; Thompson, 2005). Further, Helmreich argues that a focus on biocapital also "fixes attention on the dynamics of labour and commoditization that characterize the making and marketing of such entities as industrial and pharmaceutical byproducts" (Helmreich, 2008, p. 464).

Aside from a few notable exceptions, however, the question of what constitutes labor continues to revolve around humans and human biological matter in a binary that remains structured by Karl Marx's 19th-century theories of surplus labor and capital accumulation. On first consideration, it seems impossible to argue that nonhumans "labor" because they cannot sell their labor power; a central proposition in Marx's theory of labor (1867) and what, for him, amounted to a key condition of what makes man, "man" (see also Herzig & Subramaniam, 2017). But, as Donna Haraway (2008) has argued, "the commodities of interest to those who live within the regime of Lively Capital [biotechnology, for example] cannot be understood within the categories of the natural and social that Marx…was finally unable to do under the goad of human exceptionalism" (Haraway, 2008, p. 46).

In this vein, the philosopher of science Eugene Thacker (2005) has reimagined Marx's "species being" and Michel Foucault's (1978, 1979) notion of "biopolitics." Thacker argues that the "biotech industry appropriates the species being at the molecular, genetic, and informatics levels, and doing so, it refashions human biological life activity or labor power as a form of nonhuman production" (p. 40). The traditional activity of selling and buying of labor power

exists differently in a biotechnological mode of production. There is a "continual transformation of biological value and medical value into economic value, a continual refashioning of the [molecular] species being as at once biological and economic, as a form of 'biomaterial labor'" (p. 47). For Thacker, therefore, "labor power is cellular, enzymatic, and genetic" in markets that are organized by the imperative to produce surplus value for scientific capital (p. 45; Bourdieu, 2004, p. 27). "Paradoxically," he writes, "in the biotech industry, what is valued is not human labor, but the specific labor power or life activity of cells, molecules, and genes" (p. 40).

In a second example of renewed thinking about lively labor, Catherine Waldby (2000) argues that stem cells and tissues for transplant markets become a source of "biovalue," which is defined by "the generative and transformative productivity of living entities [that] can be instrumentalized along lines which make them useful for human projects" (p. 33; Waldby & Mitchel, 2006). Additionally, "clinical labor" occurs at the "suborganismic level of the body" in, for example, human clinical trials and tissue and organ donation (Cooper & Waldby, 2014, p. 12). This form of labor, argues Melinda Cooper and Catherine Waldby (2014), "consists precisely in the endurance or risk and exposure to nonpredictable experimental effects that may be actively harmful, rather than therapeutic" for the participants (p. 8). Under these conditions, clinical trial participants are enrolled in a "process of valorization of a particular bioeconomic sector" in which their 'services' constitute a labor relation with capital "through either the production of experimental data or the transfer of tissue" (Cooper & Waldby, 2014, p. 1, 7; Waldby, 2000; Franklin & Lock, 2003; Petryna, 2009).

Building on these recent works, I argue that in a biomedical mode of production where "innovation value" amounts to the production of scientific data which is then converted into other forms of capital, the broader question of what constitutes labor emerges for *nonhumans* too

(Cooper & Waldby, 2004, p. 3). I argue that "fostering life" historically, and most often, centers *first* on harnessing the generative and value-producing capacities of nonhuman biological actants in the name of human health in accordance to Foucault (1978). Animal biomass provides a "living material base for contemporary life science" that is itself the infrastructure for modeling human health and disease (Helmreich, 2008, p. 463; Landecker, 2007, p. 2). The "appropriation of living nature," therefore, occurs through animal bodies at the Lab as much as it occurs through human labor at the Lab or human labor in experimental trials. Although there is no immediate market profitability for the embodied work products of animals and their constitutive elements at my specific research site, the "enrolled population" of experimental animals for basic science research is nevertheless a "form a resource that can be data mined" for disease patterns in much the same way as a business can "mine the everyday world of consumption patterns" (Mitchell, 2010, p. 8). Rather than selling scientific data on the obesity market as commercial labs do, data—the conversion of blood and flesh into alpha and numeric symbols—is exchanged and converted into other forms of capital that include economic (grant funding) and symbolic (reputational) capital in the scientific field (Bourdieu, 1986).

Further, even though Cooper and Waldby's (2014) characterization of "clinical labor" is based on humans and their biological matter, the emphasis placed on the "valorization" of capital through the production of goods (whether in the form of data or biological commodities) fits with the question provoked by my findings, as well as Donna Haraway's (2008) question as to whether "*human* labor power turns out to be only part of the story of lively capital" (Haraway, 2008, p. 46). I therefore extend Catherine Waldby's (2002) concept of "biovalue" defined by the "yield of vitality produced by the biotechnical reformulation of living processes" to include the labor of biomass at the Lab (Waldby, 2002, p. 310). Following Cooper & Waldby (2014), I argue

that the same production of "surplus fragmentary vitality" is similarly situated in *nonhuman* biological matter. The Lab derives "biovalue" from the reformulated vitality of rodent bodies (Waldby, 2000, 2002). Animals are made to live and labor in specific ways to create value for human health (use value) and knowledge for the obesity market (exchange value in the form of data that can be traded on to increase economic or symbolic capital) in as a consequence of *in vivo* experiments when they are alive, and *in vitro* experimentation in which elements of dead bodies are made to labor productively and reproductively in artificial cell cultures. After all, I argue that a "yield of vitality produced by the biotechnical reformulation of living processes" occurs not only through humans and human biological matter, but nonhumans and their biological matter too (Waldby, 2002, p. 310).

In sum, biological matter possesses its own generative capacity, i.e., its own capacity to labor and therefore co-produce new data while reproducing scientific and economic capital in the obesity market. And, animals' bodies perform "biological labor" differently as a result of environmental exposures (Thacker, 2005, p. 117). The outcome of their biological labor, in turn, is translated into "inscription devices" by technicians and PIs. That is, PIs appropriate congealed labor in the form of scientific data. Subsequently, new knowledge is circulated in the obesity market as data; a form of "scientific capital" that reproduces the Lab via publications, grants and grant renewals, conference presentations, editorial work, and other professional activities (Latour & Woolgar, 1979, p. 51; Bourdieu, 2004, p. 27). In response to the 'national threat' of obesity and metabolic syndrome, animals are not only made to live and labor under extreme environmental conditions in the process of constructing environmental, biomedical subjecthood, but they are also conditioned to be productive of environmental data that is itself structured by a

large precarious funding landscape and the NIH's focus on particular 'hot topics' in obesity research.

My ethnographic findings pointed to the argument presented above that nonhuman bodies and their labor counts too, specifically as a form of lively labor in a regime of "lively capital" (Haraway, 2008, p. 46). I also found that shifts in disease etiology (source and timing of exposure) within the epigenetic and Developmental Origins frameworks challenge what it means to be (bio-)medicalize obesity (or health problems in general) in an environmental framework. For instance, how does one medicalize an interaction effect between environment sources (ubiquitous chemicals or diet) and a medical subject (particularly reproductive women)? Also, how does one medicalize an interaction effect like this when, in some instances, the subject (the embryo, the fetus, the child) is not even yet a subject (as in the instance of pre-conception medicalization)? If "[b]iotechnologial change effectively produces new material conditions of possibility" for "relations between bodies, bodily fragments, human identities and social systems," then, how does one medicalize a post-industrial, chemical milieu that produces an environmentally contingent and epigenetically at-risk individual (Waldby, 2002, p. 308; Rose, 2000, p. 487)? Drawing on Wild's (2005, 2012) concept of the "exposome," are we witnessing an era of *exposomic medicalization?* Further, what are the implications of exposomic medicalization for the emergence of new forms of health inequality organized along social class, race, and ethnic dimensions? (Brown, 1995; Bell & Ebisu, 2012; Landecker & Panofsky, 2013; Almond & Curie, 2011).

The focus on maternal environments, or more precisely what goes on during pregnancy, points to the distinctly gendered nature of Developmental Origins and epigenetic studies. Because the language of 'programming' suffuses much of the Developmental Origins literature,

it is important to highlight the gender-specific and normative behaviors at stake in these models. Getting the conditions of pregnancy 'right' falls, once more, onto prospective and expectant mothers. In the course of aiming to produce *healthier babies*, we find a politics of risk reduction, and a new "regime of perceptibility" attuned to the maternal body as pathological environment (Murphy, 2006; Lappé, 2016; Lock, 2013; Richardson, 2015, 2017). This potentiality, which is not granted, creates the possibility for new modes of biomedical surveillance, intervention, and patient subjecthood for both mothers and their offspring. Under the theoretical framework of the Developmental Origins of Health and Disease, women are expressly charged with the epigenetic governance *of* their own bodies *for* the anticipated (and uncertain) sake of theirs and their children's children. But even as large babies replace small babies as a primary concern for some pediatric/neonatal researchers, environmental exposures -particularly to everyday household goods- are unwitting and cannot therefore be personally managed; producing a paradox for those interested in programming models of exposed inheritance. Moms cannot shop their way to safety. As ecologies are "inescapable", perhaps the same is true for an environmental politics of reproduction in which exposure resembles something akin to generations of nesting dolls, from which the immediate and sedimented effects of having lived life cannot be escaped (Nash, 2006; Ginsburg & Rapp, 1991).

For some social science observers of epigenetics, the gendered specificity of Developmental Origins presents a renewed risk for reductionist and deterministic models of biological development. According to Margaret Lock (2013), a new form of "somatic determinism" lurks in epigenetic and programming science. And, according to the historian of science Sarah Richardson (2017), mothers are "vectors" of "intergenerational epigenetic programming, [raising] worries of enhanced mother blaming and increased surveillance and

regulation of maternal bodies" (Richardson, 2017, p. 44). The nutrition researcher Jonathan Wells (2009) employs the concept of "the metabolic ghetto" to argue that there is an unequal distribution of health for women and that, therefore, "maternal capital" influences health outcomes for offspring (p. 11).

In general, the conceptual and material work being done through multiple research designs (e.g., clinical, epidemiological, basic science) has shifted how we understand obesity in relation to environments, and the effects of early life exposure on developing bodies and future states of health. There appears to be some consensus among those in social studies and history and those in biomedical research, that the social environment shapes us in profound and perhaps persistent ways, and that the reconstituted biology being built today is formed by an industrially constructed chemical environment specific to the mid-20th and early 21st century. From cancer to neurological disorders, obesity and diabetes, cardiovascular health, sexual development dysfunction, and infertility, scientists from many corners of biomedical research formulate these environmentally inflected illnesses as medical and economic burdens of the 21st century.

Conclusion: The Production of Environmental Biomedical Subjecthood

In this chapter, I have argued that basic science research at the Lab, which is representative of other environmental obesity work in labs around the world, is involved in producing an environmental model of health and illness in relation to obesity and metabolic syndrome. This research is accomplished in part through the labor of laboratory animals and their constituent parts. As a result of a molecular gaze on genes, proteins, and the maternal-uterine environment, obesity and metabolic dysregulation are recast as environmentally modulated phenotypes that are, by definition, gendered. The molecules at stake are interactively intrinsic (proteins and genes) and extrinsic (food and BPA), throwing into question precise

boundaries about what is *inside* and what is *outside* the development and regulation of bodies. Consequently, the epigenetics and programming of neurons, cells, tissues, and organs suggests that obesity and metabolic syndrome do not purely result from a lack of willpower. Rather, the central regulation of biology and physiology has been transformed by environmental exposures that affect epigenetic mechanisms. At stake, therefore, there is an environmental politics of reproduction, new modes of biomedical surveillance and therapeutic intervention for reproductive women in particular, and therefore a new era in which human and nonhuman bodies are subjected to and constitutive of harmful environmental exposures specific to the Anthropocene. Studying "how our bodies respond to environmental pressures, including epigenetic changes and mutations, as well as the complex chemistry resulting from the biochemical reactions that sustain our lives…*is not a challenge restricted to those interested in the environment, it is a critical question to all interested in biology*" (Miller & Jones, 2013, p. 2).

To be biological also means to be profitable for the obesity knowledge market, reifying the political economy of obesity and assuring the position of obesity and metabolism research at the forefront of funding agencies' priorities. Biomass produces value in the form of health data for the obesity market generally and an increase in "scientific capital" in the form of grant capital for investigators specifically. The central public health aim (use-value) is to apply new knowledge about the epigenetics of pregnancy to interventions that optimize human fetal growth, which may, in turn, produce a more metabolically fit population. Exchange value is realized through the transformation of biological processes into scientific data, but it is also evident in the accrual of reputational-symbolic and economic capital for principal investigators via grants, professional speaking invitations, NIH study panel participation, and editorial work. Accordingly, animal vitality labors "in the service of both health and wealth" (Rose, 2007, p. 3).

What are the social and medical implications of programmed obesity and metabolic syndrome? Developmental Origins and the epigenetics of pregnancy are not individualized models of the genetics of obesity but, instead, environmental and intergenerational models of biological and physiological dysfunction. Using the concept of "exposome" (Wild, 2005, 2012), I argue that Developmental Origins and epigenetics suggest a change in the conditions of medicalization towards *exposomic medicalization* because the social, or environmental, is seen to be biologically causative. What new forms of environmental politics of reproduction emerge in the Anthropocene era where the maternal environment, in particular, is the site of scientific interest and medical intervention?

Chapter 4

Principal Investigators and the Matter of Precarity

> I've been here more than 10 years, and of all the summer students that have gone through [the Lab], only one has gone through for a PhD to work in basic science. It speaks volumes that none of them want to do this. At the end of the day, after education, [they say] 'I want job security.'
>
> Dr. Moore, personal communication February 23, 2015.

> If everything blew up for me, I can always go back to take care of patients. But it is very precarious…for a PhD [investigator]. If Dr. Moore loses her grants, or the support of the institution, it is like 'What do I do'?
>
> Dr. Harris, personal communication, December, 2015.

In chapter 2, we saw the effects of funding precarity on University Lab's (Lab) technical workforce and how their positioning within the Lab's division of labor affects modes of anticipatory caretaking practices. In this chapter, I focus on principal investigators (PIs) to illustrate how a "frightening" landscape in obesity research funding affects investigators, their career paths, and, how, as a result, "experimental systems" are influenced by the experiences of financial precarity in academic basic science research (personal communication, March 16, 2015; Rheinberger, 1993). I found the cyclical nature of funding serves as a kind of metronome for the PIs, orienting the scope and rhythm of their work in pace with the demands of grant making institutions such as the National Institutes of Health (NIH).

In this chapter, I am interested in the effects of grant-based funding imperatives on PIs at the Lab, and how this produces epistemic effects for obesity knowledge. The political and economic circumstances under which basic scientists produce new laboratory knowledge raise questions about the specificity of what new obesity knowledge is produced, by whom, and how, overall, this affects research agendas and ultimately the kinds of knowledge produced. Within this

context, I ask two interrelated questions. First, how and in what ways does funding precarity shape the Lab's "experimental systems" as it addresses the nation's concern with obesity as identified and supported by the NIH's research funding priorities (Rheinberger, 1993)? Second, what does it mean to live the "scientific life" (Shapin, 2008) today in basic science research; what are the effects of funding precarity on investigators "forms of life" (i.e., no life is self-contained and can be understood apart from external influences) as scientists (Wittgenstein, 1953)?

In order to answer these questions, I first provide data gathered from several federal agencies and professional organizations that connect national trends in funding precarity in the biomedical workforce with the lived experiences of PIs at my research site. My findings complement data, gathered at the national level by multiple agencies, on the effects of precarity on basic science research in biomedicine. The effects of political and economic precarity felt and lived at the Lab are not idiosyncratic.

Second, I introduce the Lab's PIs, describing their typical work practices; all of which are firmly situated outside experimental spaces. They are compelled to work differently, i.e., their 'forms of life' as a scientist are structured by contemporary configurations of scientific support. Within this section I provide bibliographic detail for each of the PIs to trace out the evolution of research ideas and practices in relation to the intense funding pressures they face and how this intersects with different career stages.

Third, I describe the context of obesity funding at the Lab and the research institute's expectations for its PIs to illustrate how this bears on their research agendas and professional lives. Newer investigators have significantly less economic and symbolic capital relative to established investigators (Bourdieu, 1986). The ability to trade on either of these forms of capital influences the degree to which one leans toward more conservative empirical questions, or

towards greater novelty (Foster, Rzhetsky, & Evans, 2015).

Lastly, I trace the effects of funding sources on their experimental systems. The practical problems of the nation as defined and funneled through funding agencies like the NIH drive the Lab's hypotheses. As a consequence of the precarious landscape in basic science research, I detail the emergence of a new experimental 'brain model' of obesity at the Lab. The brain model is itself an evolutionary consequence of funding pressures on the Lab's experimental systems; having evolved out of a programming model of fetal thirst with female mammals to the programming of fetal adiposity via adipogenic pathways (altered physiology of fat cells), to the programming of fetal adiposity via hypothalamic pathways (altered physiology of neuronal cells). In the new brain model of obesity, senior PIs consider the possibility that a calorie restricted nutrition environment for newborn mice might remodel a region of the hypothalamus that regulates eating behaviors and, therefore, reverses deleterious effects of maternal obesity on the exposed offspring. In this new but tentative model of obesity, the causes of appetite and satiety are reimagined through specific neurons working differently in the brain as a consequence of changes in the timing of food exposure. By tinkering with the timing of food-as-exposure, obesity and its corollaries - namely appetite and satiety- are given a lifespan through the lens of the developmental origins of disease (Landecker, 2011; Lappé & Landecker, 2015). A remodeling model of appetite and obesity positions the PIs to argue for medical intervention during neonatal life that, theoretically, can undo programming effects from fetal life.

In sum, I argue that the Lab's "experimental systems" are bound to national health priorities and the precarity of grant funding in obesity studies. As a result, PI's "forms of life" reveal, when put in historical perspective, a particular "form of [scientific] life" that entails working at a distance from the living things about which they hypothesize, occupational precarity,

and anticipatory writing practices specific to the Lab's division of caretaking labor.

Situating the Funding Landscape of Biomedical Basic Science Research in Academia

Financial support has always been a concern for scientific practitioners. Among 17th and 18th century natural philosophers, it was a relatively simple affair: people (men usually) generally relied on family inheritance or received money from someone they knew, and patrons of the arts and sciences, including individuals and organizations such as churches, supported those they wanted to (Shapin, 1989). According to the historian of science Steve Shapin, American scientists in the early in the 20th century were suspicious of federal money, believing that it would lead to control over their work. During this timeframe, roughly "[b]etween 1900 and 1940, the primary sources of financing for medical research were private" and included foundations, university endowments, pharmaceutical companies, life insurance companies, medical associations, and "voluntary health agencies" such as National Foundation for Infantile Paralysis (Shapin, 1982, pp. 338-347). By the mid-20th century, the federal government, as a consequence of war, "drove the fastest expansion of higher education in American history (if not the world)" with regard to funding for research and the build-up of technical infrastructure to carry out that research (Kaiser, 2011, p. 31; Shapin 2008). Within this context of war, the NIH Biomedical Research Workforce Working Group (2012) describes the initial intent of federal funding for medical research as follows:

> Originally the conduct of federally-funded research at universities and other extramural institutions was based on an understanding that institutions would provide the bulk of facilities and salaries to the researchers and the NIH would provide the majority of funds for conducting research. Over the past decades, this distinction has become increasingly blurred, with NIH providing an increasing proportion of faculty salary support and the institutions covering a larger percentage of research costs. This is especially true during the start-up period, which has become significantly longer as young investigators struggle to receive their first R01 grants. The growth of 'soft money' positions in academic medical schools, in which investigators are required to raise 100% of their salaries and research funds, has contributed to the negative views of a career in biomedical science,

> and has had the additional consequence of encouraging institutions to expand their physical space [financial leveraging that becomes troublesome when the NIH budget plateaus or contracts] without making additional long term commitments to faculty. (p. 11)

At the center of this chapter, then, is an argument about how academic labor in basic biomedical science, and the experimental laboratory knowledge it produces, are shaped by a precarious and temporal landscape of soft money grants on which PIs increasingly depend. Basic science investigators -those who study the fundamentals of phenomena- whether they are affiliated with universities, research institutes, or medical schools, are increasingly dependent on extramural support, i.e., soft money, via grants to pay for their salaries, the salaries of support staff, and their laboratory's infrastructure. Soft-money positions are not only increasing in academia, but the grants themselves have also become more uncertain particularly in highly competitive spaces like obesity research. For example, "[o]ver the last decade, with budget deficits and cutbacks, funding levels at many government agencies have dropped to around 10 percent or fewer of [grant] applications. Yet pressure on faculty to continue supporting themselves remains relentless…" (Stein, 2007, p. 2). While soft-money is a financial boon to universities who can recover faculty and technical staff salary costs and expenses from federal grants, working and living the scientific life as a PI in a soft-money position, however, is filled with precarity and anxiety. " 'Second-class citizen' is how researchers on soft-money, who have to raise their salaries from grants, describe their position. It can be fraught with financial insecurity, disrespect, and poor facilities," according to the molecular biologist and journalist Marcia Barinaga (2000).

Given an awareness of these challenges, the Advisory Committee to the NIH Director for Extramural Research proposed the creation of the Biomedical Research Workforce Working Group (mentioned above) in 2011 to develop "a model for sustainable and diverse U.S.

biomedical research workforce" (National Institutes of Health, 2012, p. 7).[24] According to a survey of the Working Group's own committee, members were most concerned with funding, specifically with "uncertainty and lack of funding, distribution of funding, restricted paylines, success rates, and excessive competition" in light of an over-supply of biomedical PhDs relative to the availability of traditional tenure-track jobs (p. 49). Having found that "[t]he proportion of PhDs that move into tenured or tenure-track faculty positions has declined from ~34 percent in 1993 to ~26 percent today," the Group recommended policy shifts to help "offer opportunities for students to explore a variety of options while in graduate school without adding to the length of training" (p. 7-8)[25]. From 1993-2006, they argue,

> [As] the percentage of tenure or tenure-track positions decreased steadily…colleges and universities chose to increase their staff by increasing the number of non-tenure-track positions. These positions often are dependent on obtaining outside funding (mainly from the NIH) to cover 100% of salary. (p. 25)

According to data provided by the NIH's Extramural Research section, the number of principal investigators, including contact and multiple principal investigators on a research project, has risen from approximately 15,000 in 1980 to slightly more than 27,000 in 2014, the high of which was roughly 32,000 in 2012.[26]

Across federal agencies and professional organizations -including the National Science Foundation's Survey of Doctorate Recipients, the National Center for Education Statistics in

---

[24] See also https://biomedicalresearchworkforce.nih.gov/index.htm

[25] Within this report, in bold lettering, the Working Group recommends that the "NIH should require institutions that receive NIH funding to participate in programs that collect such data" (p. 40). This would be helpful to academic labor market research, including this chapter. The report layouts the shortcomings of "comprehensive data regarding biomedical researchers." (pp. 42-44)

[26] See Barinaga, 2000, pp. 2024-2028; Benderly, 2010; https://nexus.od.nih.gov/all/wp-content/uploads/2015/06/Graph21.jpg

collaboration with the Department of Education, the American Association of University Professors, and the Bureau of Labor Statistics, academic labor market researchers have found a marked increase in "contingent" faculty in academia since 1975, including a rise in both non-tenure-track faculty and part-time faculty; many of whom would be grant-dependent.[27] Likewise, among basic science faculty researchers at medical schools, the Association of American Medical Colleges' management researchers Mandy Liu and William Mallon (2004) have found a growth in non-tenure track appointments, identifying shifts in "the external environment surrounding the basic sciences" that have led "institutional leaders and policymakers to reexamine basic science faculty appointment policies" in the direction of greater flexibility for the employer (p. 206). Further, they write,

> [T]he biomedical research enterprise is no longer a genteel academic pursuit but an underdeveloped strategic market opportunity. Biomedical research is considered a vast potential driver of economic development, entrepreneurial opportunities, and increased income streams…*As a result of increased extramural funding and changing institutional priorities, basic science faculty are under greater pressure to spend more time on research, win more extramural support, and recover higher percentages of their salaries.* (p. 2, emphasis added)

In response to these professional funding realities, Donald Stein (2007), a professor and member of an advisory council to one of the NIH, writes of the "concern that so many biomedical scientists have to follow the money rather than their passions and interests, or even the logic of their research trajectory." "What does it mean," he asks, "to have a large population of researchers

[27] See Barinaga, 2999, pp. 2024-2028; Benderly, 2010; Kalleberg, 2009; and the following webpages: https://www.aaup.org/sites/default/files/Faculty_Trends_0.pdf; https://www.bls.gov/opub/mlr/2015/article/stem-crisis-or-stem-surplus-yes-and-yes.htm; https://nsf.gov/statistics/srvydoctoratework/; https://www.aaup.org/sites/default/files/Academic%20Labor%20Force%20Trends%201975-2015.pdf; http://journals.lww.com/academicmedicine/pages/articleviewer.aspx?year=2004&issue=03000&article=00003&type=fulltext; https://www.aaup.org/sites/default/files/files/AAUP-InstrStaff2011-April2014.pdf

who are not connected to the intellectual life of the community" because they sit outside the functions and professional duties of their tenure-track colleagues? (p. 3). According to Ron Hira, assistant professor of public policy at Rochester Institute of Technology, " 'Almost no one in Washington' recognizes the 'glut' of scientists, nor the damage that lack of opportunity is doing to the incentives that formerly attracted so many of America's most gifted young people to seek scientific and engineering careers" (as cited in Benderly, 2010, para. 15). At the core of these conversations among PIs and academic labor market experts is the concern for a destabilized academic market in biomedical basic science that creates disincentives for new generations of scientists and further affected is the pace and content of scientific discovery.

In addition to the overall precarity of biomedical research funding in terms of budget and competitiveness in obesity research, an analysis of grants awarded by the National Institute of Environmental Health Sciences (NIEHS) for Developmental Origins research shows a change over time (1991-2011) in what exposures garner the most funding opportunities, the specific health (disease) endpoints of interests, and what counts as significant timing of exposure during the lifespan (Haugen et al., 2014). So, not only is the funding landscape precarious, but research calls for particular exposures on specific developmental trajectories within a certain time-frame further shape what kinds of knowledge about the environmental determinants of health are produced.

My findings complement data collected by multiple national agencies as to the constitution of and challenges for the biomedical workforce. National data indicate that academic PIs in basic biomedical sciences are increasingly beholden to short-term grants rather than long-term tenure-track faculty commitments to universities and medical schools. The growth of soft-money positions in academia generates precarious work conditions and worker insecurity. As a

result, the pressure of soft-money funding affects "a scientist's choice of research problem," which itself also varies in accordance to what the grant agencies are interested in funding. The NIH, for example, defines what "disease burdens" exist and are worthy of being funded, nudging PIs to shape their careers around "hot topics" that are presumed to be more fundable (National Institutes of Health, 2017c). To help investigators navigate this terrain, the NIH even offers a matchmaking service so that applicants can take into account its *Funding Opportunities Announcement* as they strategize potential research plans that they hope will be funded.

In conjunction with the argument above that precarious funding pressure and the availability of funding for national "diseases burdens" shape research designs, I also argue that there is a temporal dimension to this outcome. One's career position in basic science bears on choice of research questions. Early stage and new investigators, who are not yet independent, compete with established investigators for large grants such as the R01, but they can also choose from a selection of smaller grants with shorter time commitments because, in the words of one new investigator at my research site, "no one will give you money without a proven track record" (personal communication, February 25, 2015). In relation to new and established investigators, Michael Lauer, the Deputy Director of the NIH's Extramural Research, has provided data on funding trends from 1995 to 2015 according to career stage. In general, "Early Stage Investigators," those with less than ten years since their terminal degree, and "New Investigators," those beyond ten years since receiving their terminal degree, "do not have many active awards" (Lauer, 2017, para. 6). Further, this data indicates that established investigators, those who have received competing R01 and equivalent awards, "may have an easier time staying in the system. Established Investigators not only make up a greater proportion of awardees, they also secure, to an even greater extent, a larger proportion of competing award dollars" (para. 8).

Additionally, "the current reliance [by the NIH] on preliminary results further discourages branching out into high-risk, high-reward areas" for early career investigators who utilize K99/R00 grants that are designed specifically for new investigators (National Research Council, 2005, para. 10). On the other hand, senior PIs have accumulated credibility, or symbolic capital, in the knowledge market (as well as greater economic capital) and are therefore less constrained in their research questions. They can pursue riskier, or more novel, conceptual projects via R01 grants, the NIH "gold-standard," because they can trade on accumulated symbolic capital typically documented by frequency of publications in their grant applications (Bourdieu, 1975, 2004). However, Nobel Laureate Roger Kornberg argues that, for both new and senior investigators alike, "[i]f the work you propose to do isn't virtually certain of success, then it won't be funded" (Lee, 2007, para. 5).

In sum, dependence on external grants (soft money) that is organized by the NIH in accordance with specific disease burdens helps define what questions *can* be asked. Additionally, one's career position (and therefore symbolic and economic capital) largely shapes the *types* of grants one can apply for. Thus, PIs -early and senior- are differently constrained in their research choices. Given the precarious context in which PIs increasingly carry out their work, I take the position in this chapter that internal details of the individual investigator, her or his stage of career, and the institutional context in which PIs work cannot be cleaved off from the external pressures of soft-money funding and the specificity of funding for national health priorities when decisions about research questions and experimental designs are made.

Principals and Precarity at the Lab

Based on my own reading of the literature and my initial "anthropological strangeness" prior to entering the Lab, I was surprised to find that PIs did not work at the bench (Latour &

Woolgar, 1979). Their primary workspaces were offices located in a separate building, a short walk away from the sociotechnical sites of knowledge production. The most senior PI, Dr. Harris, was a tenured MD-PhD, but Dr. Moore, the one who run the Lab's daily affairs, had a more precarious assistant professor position with the Institute and was reliant on extramural support for her funding in spite of her status in the field. Because Dr. Harris was a medical doctor, he maintained a clinical practice as well as teaching duties with the affiliated university hospital and, in general, enjoyed much greater employment security as a result. While Dr. Harris spent most of his time outside of their office space, Dr. Moore managed the Lab's everyday business affairs from her office, including directing the scope and pace of experimental work performed by the technical staff in order to meet imminent grant, conference, and publication deadlines. Both senior investigators had editorial roles with academic journals, which involved review of paper submissions, administrative work such as identifying associate editors, soliciting papers, advertising the journal, working with the publisher, and representing the journal at professional society meetings. Their professional practices also included volunteering for NIH Study Groups, presenting results at multiple conferences each year, giving talks at local (as well as international) universities, serving on graduate student committees, and running undergraduate programs for a local high school and a university that allow students to gain everyday laboratory experience. Lastly, both PIs had ongoing empirical work with other researchers at universities and labs outside of the United States.

According to François Jacob, winner of the Nobel Prize in Medicine for discovering how genes are regulated, experimental systems are "machines for making the future" and "everything depends on this choice" (Jacob, 1988, p. 9). At my site, however, experimental and therefore human futures are not open. Hypothesizing at the Lab was somewhat inverted; meaning that for

PIs, the certainty of financial precarity and the intensity of competition for obesity funding had produced a climate that distanced them from everyday scientific practice in the wet lab; a reality echoed at labs across the country. It is in external spaces, from medical libraries to private meeting rooms, online exchanges and large professional conferences, that PIs help shape new knowledge about the Developmental Origins of obesity and metabolic syndrome. Rather than an interactive tinkering process between physical and intellectual labor, emergent ideas for PIs often develop out of literature reviews that are highly strategic because hypothesizing is done in relation to the NIH's Requests for Applications (RFA) and the Lab's existing experimental systems. In a sense, therefore, experimental futures for PIs are not open. They are constrained by national health priorities and therefore funding allocation that, in turn, structures the kind of obesity knowledge that is produced and by whom.

This way of doing science stands in contrast to the more purist notion of science that Rheinberger (1992) presents in which scientists follow results wherever they go. PIs today cannot. They are constrained by funding priorities, and the precarity of soft-money funding. In the words of Dr. Moore, "in a way, we kind of sell our research in terms of putting it out there at meetings, conferences, in papers…so that people [can then] say, well, that is rubbish, or that is cool…[in order to help you see] what sells, what will get you funding" (personal communication, January 14, 2016). PIs work through the empirical data, but do it at the level of inscription, not inscribing raw data (Latour & Woolgar, 1979). That work is for technicians who have been trained and earned the trust of PIs who, in their early years, performed the embodied work of inscribing data. As we saw in chapter 2, technicians produce the raw date on which PIs depend, and this finding also complements nationally based data. It also raises questions at my research site, and in national data, about what it means to be a scientist today.

Dr. Harris. Dr. Harris was the most senior PI and the Lab was often referred to as "Dr. Harris' Lab," even though Dr. Moore managed its daily activities. Notably, Dr. Harris is the only man in the Lab, which struck me at first. After spending time in the Lab and learning more about the demographic shifts in education, I learned that this particular structure is not uncommon. Dr. Harris established himself decades ago during a time when men dominated the fields of biology, physiology, and genetics. Today, however, there is a gender balance in medical science and biology for example. This Lab, therefore, shows a generational structure that mirrors the changing demographics of science over the last thirty years.

While the Lab's conceptual apparatus is grounded in the Developmental Origins perspective on fetal origins of adult health, Dr. Harris did not begin his career having a particular interest in obesity and questions around appetite, adiposity, and health-related sequelae. Instead, early in his career doing hands-on experimentation, Dr. Harris studied osmoregulation (homeostasis of water and minerals in cells) in levels of amniotic fluid in a mammal model of maternal-fetal (de-)hydration in order to elucidate mechanisms of fetal swallowing and esophageal functioning. Two pregnancy conditions, polyhydramnios (high amniotic fluid level) and oligohydramnios (low amniotic fluid level), that are rare but extremely dangerous for both the mother and fetus. In order to measure fetal swallowing, the research team altered amounts of salt mothers received in their drinking water and food during gestation. His team found that changes in salt content in the maternal diet affected rates of fetal swallowing. Surprisingly, it raised the possibility that swallowing itself might signal fetal thirst. Prior to this, it was believed that both thirst and appetite developed only after birth, marking this as a consequential finding to the field of maternal-fetal medicine and others research areas concerned with developmental biology and physiology.

As a result of these findings, Dr. Harris surmised that, if thirst could be induced *in utero*, it would be possible to "make the baby swallow [as a result of] appetite stimulation. Would the baby *in utero* be hungry" (personal communication, December 9, 2015)? Further, he imagined how such early environmental "insults" on the developing organism might affect "thirst and fluid [and hunger] status *after* birth," giving new shape -and an orientation toward thinking about disease within the lifespan- to his research interests that would, over many years, coalesce into experimental systems centered on the long-term health effects of early life exposures (personal communication, December 9, 2015).

Importantly, during this time as a junior researcher, Dr. Harris also learned that he needed to be "constantly thinking ahead about how he is going to renew the grant" (personal communication, December 9, 2015). During an interview with him, he described a "moment of joy" in which he was notified of an NIH grant acceptance and recalled that he "went running up" to his mentor's office to share the good news. Although Dr. Harris fondly described his mentor as not being "known for his warmth or anything," his mentor responded, "Well, that is very good, Steve. What are you going to do for the renewal?" Deflated by his mentor's response, Dr. Harris nonetheless repeated to me the hard realities of basic science research funding:

> Okay, so here I got a grant funded. This is my moment of joy, and it's a five-year award. And the question [now] is how am I going to renew the grant? And it really is an important thought. You have to be constantly thinking ahead, you know, [about] how to get preliminary data and that takes you a year or two depending on what you are doing. With the NIH you have two tries (and at that time you had three tries to get a grant) and a nine-month turnaround. So you are always thinking, okay I've got this funding now, by this time [a date of submission], I need to write a grant. (Personal communication, December 9, 2015)

Although he laughed about it now, it took several years before he pursued programming models of adult health in this Lab. Importantly, when he finally did, he studied programmed appetite rather than thirst—his initial experimental system—because, as he reasoned, even

though “dehydration is a big factor in the developing world, the outcome of the consequences of programmed appetite versus programmed fluid status is probably [that] appetite is far more important [than thirst].” He understood that obesity was gaining prominence as a scientific object worthy of care and attention (Fortun & Fortun, 2005) and that it was more likely to be supported by the NIH and other national agencies such as the American Diabetes Association than the more relatively marginal medical problems of too little or too much amniotic fluid However, in spite of his growing recognition, Dr. Harris still needed persuading.

Despite his own early work on maternal-fetal health and the overall growth of so-called “fetal origins” research during the early 2000s, Dr. Harris “dismissed” epidemiological findings in particular (the area of most growth). From his perspective, they were “nonsense” because David “Barker was all human data, [and there is] lots of potential for confounding issues” in correlational studies (personal communication, December, 9, 2015). It was not until Dr. Harris visited a colleague’s lab in Europe, where an experimental diabetic rat model was being tested for maternal-environmental effects on the dams’ offspring, that he began to shift his own perspective. As Dr. Harris recounted his experiences to me, he said that the maternal-fetal animal food models “were no social cues [from parents],” and that, for him, the results were “fascinating” because the experimental animals indeed got fat because their mothers were obese while they were *in utero*.

Dr. Moore. While Dr. Harris needed to be persuaded of the power of programming models, Dr. Moore was hired by him in the early 2000s as a junior faculty member for her graduate and post-doctoral experimental work with animal models of programmed type-2 diabetes, a component of the metabolic syndrome. Dr. Moore developed an interest in environmental effects on children’s growth rates as a result of experiences during her

undergraduate studies in Southeast Asia. Traveling from high-to-low socioeconomic hospitals to conduct research, she found that children born with low birth weight, or premature, were "growth dependent on stimulation in the post-natal environment"; in other words, "how good your nutrition was after birth, or how much environmental maternal stimulation you got" affected their outcomes (personal communication, January 16, 2016). Dr. Moore was also influenced by the literature, specifically the "brilliant work done by two nutritionists," Elsie Widdowson and Ronald McCance in the UK, whose research on poor nourishment among people in Nazi Germany occupied territories (along with many other laboratory experimental studies on the chemical composition of food) gave shape to the burgeoning idea in the 1940s and 1950s that "there are long term consequences of compromised development" (personal communication, January 16, 2016).[28] These findings, of course, were similar to the ones that fellow Briton David Barker would formalize, several decades later, as the "fetal origins hypothesis" on the basis of population studies linking low birth weight to morbidity and early mortality.

Once accepted to a prominent university in Europe because of her undergraduate training in nutrition and biochemistry, Dr. Moore was mentored by intellectual descendants of Widdowson and McCance. While at university, she helped establish a protein restriction model of type-2 diabetes using an animal model. After several years of postdoctoral work, Dr. Moore joined Dr. Harris' Lab because, as she noted in our conversation, "not many labs were doing

---

[28] Widdowson and McCance were something of a national treasure to Britain in the 1940s, having established (among many other things) a rations diet for wartime Britons, and encouraged the use of vitamin supplementation in industrial foods, notably calcium, in everyday food such as bread. Their book colloquially referred to as "McCance and Widdowson" was considered the 'dietician's bible' and became the foundation for modern nutrition. Widdowson was awarded the Order of the Companions of Honour in 1993 for her achievements in nutritional sciences.

[programming work]" at the time (personal communication, January 16, 2016). She was also dependent on her own immigrant visa status and required an American citizen and mentor in order to work in an American lab such as this. Given his prior work on osmoregulation, which she characterized as "maternal and imprinting," Dr. Moore hoped "there was a potential [to do programming work] in his Lab". In spite of Dr. Harris' initial resistance, Dr. Moore won a five-year NIH mentoring grant, which is a career development award for junior faculty (among others) meant to help young investigators establish independence as principal investigators. Wanting to set herself apart from the work she had previously done on diabetes, and to build on Dr. Harris' existing experimental models, Dr. Moore articulated her new experimental system as follows:

> If you water-restrict the mothers, you also nutrient restrict. If water is not there, they reduce food intake…[and so] it became a model of nutrient restriction, [which] set me apart from my PhD work on diabetes. *Obesity was a problem* so I said, 'Okay, if the mother is undernourished, and you have low birth weight, do you have increased susceptibility to obesity? (Personal communication, 2016)

In order to craft a competitive grant application with the NIH, Dr. Moore built on Dr. Harris' previous experimental systems, as well as her own studies, to propose a new experimental system that would suggest some novelty while fitting neatly in with the NIH's health priorities around obesity. It is in this way, perhaps, that we find experimental systems come to "have a life of their own" (Rheinberger, 1993, p. 444). What began at the Lab as a maternal-fetal water restriction model of a relatively rare but extremely dangerous pregnancy condition became a maternal-fetal nutrition restriction model of a costly, widespread, and yet contested epidemic around fat, which was comparatively better funded.

Experimental systems are shaped by the funding landscape. To provide another example of "living and dying by grants," Dr. Harris told me that he and Dr. Moore were recently

approached by the NIH to inquire about their (the PIs) possible interest in programming models of addiction (personal communication, December 9, 2015). Addiction, like obesity, has entered the national dialogue in terms of an "epidemic," particularly from prescription opioids (Kolodny et al., 2015). "They have a funding interest," he said, and "they are just trying to get interest going, [which] is sort of their job" (December 9, 2015). In response to the query, Dr. Harris explained to me how an opportunity that the NIH initiated might be transformed into a new experimental system:

> They asked about programming and addiction and we had explored that a couple of years ago so I took another look at that and said Oh, should we do programming and addiction, and what would it take? So I went back to the old data that we have on undernutrition and effects on the reward pathway in the brain, and then [reviewed] some recent studies that we are doing with a group in Colombia, South America on undernutrition and food preference, which is also the reward pathway. And I went to the literature and said, 'Gee, can I put this together mechanistically? How would I do that?' (Personal communication, December 9, 2015)

In turning to the literature to help him "get this topic down," he elaborated on the process by adding:

> I just started saying, "How do I do this?" And [I] ended up stumbling on to some evidence that an enzyme, SIRT, which is an energy sensor that is also a factor that works epigenetically [and] is associated with changes in the dopamine and opioid receptors—potentially--in the reward pathway. It turns out that we've looked at that same factor, SIRT, in the appetite center, and so suddenly it starts to gel. Here is what we found, here is the pathway, right, and [now] this can be the model. We can make a case that it is probably reduced throughout the brain or increased. Other papers have shown that it acts to affect the reward pathway, and so we can [study whether] undernutrition in utero may be associated with an increase potential for addiction as an offspring. We have a little bit of data on food preferences which is sort of an addiction like property, and we collaborated with this guy from Cincinnati as well as Colombia, and we can put together a story, so let's do that. As a matter of fact, there are papers here [points to his briefcase] to try to put that together. So it is looking at an opportunity and saying can you go with the flow? (Personal communication, December 9, 2015)

"Going with the flow" means reproducing basic science knowledge markets while extending one's existing experimental systems, and it is how investigators survive in this

precarious climate. Working in the literature is as important as working in the laboratory properly, but PIs' "feeling for the organism" (Keller, 1983) in this case is the literature and specific molecular pathways directly and indirectly associated (at this time) with obesity; they treat "literature as an informant" in order to evolve and compete in the grant-based landscape (Kelty & Landecker, 2009, p. 177).

Dr. Jackson. The Lab's junior PI, Dr. Jackson, met Dr. Harris at a professional conference and he showed interest in her conference posters. Dr. Jackson was interested in developmental physiology and metabolic function, specifically glucocorticoids (steroid hormones). As with other young investigators, her career development hinged on an increase in "positive" findings and authorship (personal communication, February 25, 2015).[29] To that end, she helped Drs. Harris and Moore's research in exchange for a salary, use of the Lab's facilities - including technical support, and co-authorship on articles with them. At the same time, she had to separately apply for and raised her own grant money, generated data, and increasingly published articles as first author. In this liminal state in which Dr. Jackson "work[s] for them, but kind of not," she studied the hypothalamus-pituitary-adrenal axis (HPA) to ascertain how bisphenol-A (BPA) affects estrogen responsive tissues including fat, the hypothalamus, liver, and skeletal muscle (personal communication, February 2015). Studying the renin angiotensin system helped her characterize and explain how hormones control blood pressure at the level of liver and kidneys. It also gave her the opportunity to pursue her own specific interests in glucocorticoids while at the same time hitching onto an established system at the Lab. And she had to do it because "no one gives you money to do your own thing until you've proven

[29] This is not bias. This is pressure on everyone to produce positive findings. See Belluz, Plumer, & Resnick (2016).

yourself" (personal communication, February 2015). Dr. Jackson therefore used "some of their money to do some of their stuff [in order] to show that I can do my own thing," a kind of catch twenty-two with grant funding in basic science that frustrated her (personal communication, February 2015).

When Dr. Jackson and I first met in 2015, she already had ten years of experience but was still not an independent investigator. Dr. Jackson frequently lamented the difficulties of establishing an independent career and a tenure-track job in academic research. She identified money, and how to go about getting it, as the key problem. In a conversation we had about her work one day, Dr. Jackson became nearly resigned on the matter. She said:

> I thought if I had a really interesting research question and good data to support it, that I'd be able to get it funded. And that is not true. I think there used to be much more scope to say, okay, we will fund these things that we need to do and [then] all these other people with great ideas [too]. And those people with great ideas don't get money right now because they [NIH] is only funding 10 percent [of applications]. They have to focus on their [national health] priorities. (Personal communication, February 2015)

As a result of this, like others in basic science, Dr. Jackson found herself approaching daily work in very practical, less ideational, terms. She read literature to see "what's out there," and to see "what the concept is" in order to compare "the literature" to her own findings. As with Drs. Harris and Moore, the literature served as an "informant" that was used to "integrate what is known and not known" so that she could "relate all of it to funding" and increased her chances of becoming an independent investigator (personal communication, February 2015; Kelty & Landecker, 2009). Dr. Jackson said she was not "free" to "choose" whether to follow a particular topic or even change direction if she didn't "think [the results are] going where I thought it was" going to (personal communication, February 2015). She concluded, "I have an investment in this, and it is, I don't know, it is not necessarily what I want to continue…[I]t is a real pain in the ass that we like our jobs…it's really annoying" (personal communication, February 2015).

Although Dr. Jackson found her work very fulfilling, when I first approached her about shadowing to help me get acclimated to the Lab, she laughed and said it would be "boring" for me because she would be at her desk, reading and writing. Because, as she explained, "that is what you do. You either write for publication or for grants" (personal communication, February 2015). And which journal she submits to matters a great deal. As we discussed results from the BPA model and glucose tolerance tests they had recently completed, Dr. Jackson worried that her ideal journal would "hold us to a much higher standard…and we might have to go for a more developmental origins journal where people haven't seen this data in the same way, who are willing to see it at the first level [descriptive as opposed to causal], and go from there" (personal communication, February 2015). In terms of publishing their results, she said it "depends on what community you come from, and what the reviewer(s) decide". Dr. Jackson recognized that "it is not necessarily what I want to continue investigating, but I have to get something going" (personal communication, February 2015). "I have pressure," she said, "and I need to get funded, and this is the data I've got, and I have to have something interesting about it that is worth working on because my career depends on it" (personal communication, February 2015).

In sum, financial precarity molds investigators' careers as much as it historicizes and structures emergent experimental systems at the Lab. Precarity shapes both research questions and division of laboratory labor in academic science. And, based on workforce analyses by the NIH, NSF, and other federal agencies as to the impact of available funding on science and scientists (which was observed at my research site and noted elsewhere at professional conferences), these findings -that research questions are shaped by individual history as much as external career pressure- are not idiosyncratic. These findings are typical of laboratory life today. In my interpretation of Dr. Harris' explanation about how a programming model of addiction

was born, his motives are an inescapable blend of curiosity and practicality. With technicians at the bench (chapter 2), PIs are quite literally distanced from experimental practices. Although PIs have a history of experimentation at the bench, it is confined to their very early careers, tapering off as they finish their post-doctorate fellowships. As a consequence, PIs are better understood as lab managers in which their primary job is to work in relation to the literature and to "sell" the Lab's data to funding agencies and other obesity labs so that they can reproduce their own careers and that of others in the Lab. Because, in the end,

> In academic medicine doing research, there is always funding pressure….and so you say, 'what do I like to do? What am I good at doing, or what is my lab good at doing? And what are they [NIH] interested in funding?' I might be fascinated by fetal physiology, but if they [the grant reviewers] say, 'You know it's interesting, but what does that have to do with health?' So you have to sell it. Anyway, it is that whole combination of events. It is how to package it. (Personal communication, December 2015)

Funding Precarity in Basic Science Obesity Research at the Lab

Relying on grants rather than the greater certainty of regularized employment through a university as tenure-track faculty is a risky proposition for those in soft-money academic positions. To illustrate this ethnographically, during a weekly Lab meeting, Dr. Moore explained to a visiting scholar that the NIH is "the gold standard. If you are an NIH funded investigator, you have made it. That is why [Dr. Harris] and I just tear each other apart. [We are] helping each other out with our experiences as we debate what to study and how to sell it" (personal communication, March 2015). This is because even though the NIH has an approximately 30 billion dollar budget each year, the overall success rates in 2013 were at a "historic low" and generally part of a downward trend that began in 2003 (Rockey, 2015).[30] In 2014, for example,

[30] Moreover, NIH appropriations are subject to the vicissitudes of the nation's economic and political situation. For example, although the American Recovery and Reinvestment Act of 2009 shored up economic support for NIH applications for a time, a political sequestration in fiscal year in 2013 saw a $1.55 billion or 5% reduction in the NIH budget. Since then, the overall

only 15.9% of all new grants were successful, and R01 applications, the "original and historically oldest grant mechanism used by NIH" were similarly funded at 15.4% (2017d).[31] In contrast, approximately twenty years ago, if an applicant for an NIH grant was ranked in the 25th percentile (the lower the percentile, the better the review score), he or she was very likely funded. Today, though, "if you are in the tenth percentile, [you] may not get funded. That's "frightening", Dr. Moore exclaimed (personal communication, March 16, 2015). She added,

> The whole process is shifting. It is a wake-up call that there are not many young investigators being trained, and no one now gets funded before forty years of age. No one has an R01 before forty, so that has been a wake-up call [for everyone]. (Personal communication, March 2015)

Indeed, according to the National Academies of Sciences (2005), the average age for an R01 recipient increased to 42; while in 1980, it was 37. It is a rather long wait, then, to become an independent PI.

In contrast, as Berly Lieff Benderly (2010), an analyst of the scientific labor market and Fellow of the American Association for the Advancement of Science, notes "scientists of previous generations, such as Albert Einstein, Marshall Nirenberg and Thomas Cech, were winning their Nobel Prizes for work done in their twenties," (para. 29) but today investigators struggle to secure the necessary funding. Understanding these pressures, Dr. Moore reflected upon Dr. Harris' acknowledgement that *what matters is what the NIH is interested in* saying

---

budget for fiscal year 2015-2016 has grown again. In addition to the field of obesity research becoming more crowded, the NIH budget itself is also subject to expansion and contraction over time; thus linking precarity of scientific work not only to an explosion of obesity researchers in the field, but to the perceived health of the national economy and political environment because it is connected to Congressional appropriations. See https://nexus.od.nih.gov/all/2014/01/10/fy2013-by-the-numbers/ and for more fiscal year 2015 budget information, see https://officeofbudget.od.nih.gov/pdfs/FY15/FY2015_Overview.pdf.

[31] See also https://report.nih.gov/success_rates/Success_ByIC.cfm; https://grants.nih.gov/grants/funding/r01.htm

"You have to have the right topic which NIH is interested in funding," as well as, "and that is why it was a smart move when I came here—to move into obesity because it is a problem, right?" (personal communication, March 16, 2015).). Indeed, obesity was introduced as a new health endpoint in grants managed by the National Institute of Environmental Health Sciences (Haugen et al., 2014).

Dr. Jackson's sense of anxiety about launching independence is a common thread among early career investigators in particular, but it is also prevalent among senior investigators, including those at the Lab, because they work in a so-called "soft-money" institution (Sigl, 2002; Muller & Kenney, 2015). "This is the strange thing…we've got two [employer] components : it is a research institute and it heads the grants," explained Dr. Jackson (personal communication, February 2015). That is, although the Lab is affiliated with an academic institution -what I call Biomedical Research Complex (the Institute), there is no guarantee of funding for PIs. Rather, the Institute is a soft-money academic research center (i.e., PIs must raise their own money to carry out research), including support for everyone's salaries. On a separate occasion, Dr. Moore explained to me that the Institute "can't keep paying benefits, give [us] office space, a lab space without [us] bringing in an income, and that is a wake up for [us all]. If I don't bring in money, I have nothing" (personal communication, March 16, 2015). "[The Institute] doesn't pay [our] salary at all. They just provide the infrastructure for us to do our experiments, so there [is] minimal support" (personal communication January 9, 2016).

For Dr. Jackson, the Institute had recently agreed to employ her since she received her own external funding, a two-year R03 grant from the NIH. As with Drs. Harris and Moore, the Institute would help provide some salary support, office space and, in general, the *infrastructure* needed to work on their studies. Additionally, there was a tenure-track appointment, which was

organized and managed by the affiliated university. Dr. Jackson described some of the process and what was at stake for her:

> [The University will] collect the recommendation letters, and it has to go to the Academic Senate. It is a strange place. I think a lot of people would probably be happy to come here to work as a researcher at the Institute. My prefered position would probably be in a physiology department at a university, and I do like teaching. I am from a family of teachers and so I don't want to find myself having done this for five years, eight years, and then we move because my husand is on a five year contract right now. I don't want to have to go back to square one and say I want to start my tenure-track process even though I have been working on it for years. So, the affiliation with the Institute and Lab is kind of important to me in that sense.
>
> I am only here because of Dr. Moore. She does exactly what I want to do in many ways, and the person I worked with before knew her. That person said go to the Institute so you have someone whose work you can tag onto until you get your stuff up and running. Because you really need that at this level. They just won't give me 250K with no proven track record in the country. Everything I did in the past was in Europe, and it is a whole different system. I am not an MD, I am a scientist physiologist. For now, I have to remember we've just got some data, and I want to get it out. (Personal communication, February 25, 2015)

In this context, Dr. Moore had described their professional lot as an "uphill battle, and many times we feel like giving up but you don't because that is the end of you and your career." Dr. Jackson added to the conversation, "and yet lots of people do. Of all the people I did my PhD with, I am the only person still in science" (personal communication, February 23, 2015). Many, she said, go on to teach, including at community colleges simply because they could not get funding. Reflecting further on the situation, Dr. Moore added:

> The scary part is that the young generation does not want to go into PhD. And who then is going to be teaching at university level? That is, especially in basic science, we are losing a lot of people and, in fact, we are importing PhDs….It's sad. We got one person to do a PhD, and we were like –wooa—that is something. I've been here more than 10 years, and [of] all the summer students that have gone through, only one has gone through for a PhD to work in basic science. It speaks volumes that none of them want to do this. At the end of the day, after education, [they say] I want job security. (Personal communication, February 23, 2015)
>
> During a casual conversation I had with another young investigator outside of the Lab, I

was told that anyone wanting to enter a career in basic science research "needs a talking to" (personal communication, June 9, 2015). The apparent difficulties of a life in basic science research are felt outside of the Lab too. Both Dr. Harris and career technician, Eleanor, relayed to me that neither of their children wanted a career in basic research in part because of the tremendous precarity of the job. PIs in academic basic science research have reached the apex of formal education and training and, yet, they are required to be self-supporting professionals. Not only are PIs identifying the inherent precarity of research funding, but they are also pointing out its implications for who exactly is at work in the American basic science workforce.

Based on the "hard realities of soft-money" in basic science research today, some people opt out of a science career altogether. For others, it extends the period of apprenticeship to ever-greater lengths of time, and it also affects the general composition of the scientific workforce. These pressures also affect decisions about what experimental systems to pursue. A constrained choice is, therefore, made as to whether and how to proceed in basic science research. No wonder, I thought, that weekly meetings were often organized around funding. The pace of work at the Lab is largely, though not entirely, determined by funding cycles and the need for new data; all of which is designed to increase credibility of their findings in the field of developmental origins of programmed obesity. Dr. Jackson, explained,

> That is why these things [models and experiments] are on-going. We have all the projects [experimental systems], different levels of funding, and so [on any given day], this one [model] needs new data, this one [another model] needs preliminary data for a grant, and we…stagger…the schedule. The experiments don't run from start to finish per se. (Personal communication, February 25, 2015)

At University Lab, a highly competitive funding landscape shaped the way in which PIs mobilize values about obesity (i.e., it is a global health crisis) in order to raise the translational profile and utility of their own research. During the Lab's meetings, the team was frequently

reminded by Drs. Harris and Moore that, "it takes a long time to get a story," and "what matters is what the grant renewal is" (personal communication, January 1, 2014). Job security, professional recognition, and personal livelihood cannot be "decoupled" from rhythms of scientific productivity and the view of science as precarious labor (Bourdieu, 1999, 2004; Foster, Rzhetsky, & Evans, 2015, p. 900; Wouters et al., 2008). By situating the Lab in its larger political and economic context of funding for an urgent epidemic, we can attest to the "mediated character of science" and discover what it becomes productive of in this time and place, namely an environmental problematizing of obesity through women's reproductive lives (Wouters et al., 2008, p. 326).

"A Somewhat Suspect But Novel Theory of Remodeling" in the Arcuate Nucleus

"Don't chase phenotypes," Dr. Harris' reminded the team during a meeting. "For the grant renewal, what is the epigenetic mechanism" we are searching for (personal communication, November 21, 2016)? Although recording animals' phenotypes (e.g., their weight, blood pressure, and insulin levels among other traits) was where the *story* begins of how the Lab pieces together research questions and a new experimental system, Dr. Harris emphasized the need to move quickly because a R01 renewal application was due in the next few months. In light of this pressure, he wanted to explore "a somewhat suspect but novel theory of remodeling" that involves a ratio of appetite to satiety neurons in the arcuate nucleus region of the hypothalamus. His decision to pursue an appetite-satiety programming/re-modeling model of obesity via appetite was driven by the need for a novel yet familiar paradigm that the NIH would likely be receptive to in their renewal. What I will call the Lab's *brain model* was frequently described in meetings and at the bench in terms of human health correlates with obesity rates, type-2 diabetes, and hypertension. For example, Dr. Jackson once explained:

> We are interested in learning how appetite is regulated in the brain. In regard to type-2 diabetes, we are interested in changes to glucose in the blood, or insulin insensitivity and how glucose gets out of the blood and into storage tissues such as fat, liver, and skeletal muscle. (Personal communication, January 2015)

I was fortunate to begin my fieldwork at an early stage of this new experimental system, and, as a result, in addition to observing technical staff at the bench, I was also able to observe interactions between PIs and technicians during weekly Lab meetings about how to establish this new experimental system. During those meetings, sometimes tense conversations laid bare why and how the Lab would "grope around" adult, young, and newborn mouse brains to figure out how the regulation of appetite and satiety might be re-modeled due to changes in newborn chow. A working assumption is that if one can molecularly regulate appetite, one can manage eating and therefore weight and, in the case of excessive weight, co-morbidities of adiposity. Armed with the 'right' input, or balance of hormones and receptors (which can cause changes in gene expression), appetite -the output- might be differently regulated in a socially desirable way. In the following pages, therefore, I illustrate the PI's experimental brain model situated within the arcuate nucleus (ARC) region of the hypothalamus.

What is the arcuate nucleus (ARC)? The ARC is a hormone-regulating gland located within a region of the hypothalamus. Within the ARC, there are two main populations of neurons that work antagonistically in relation to one another. Together, they regulate an organism's appetite, i.e., its sense of hunger or fullness. Orexigenic neuropeptide Y (NPY) and agouti-related peptide (AgRP) are neurons that stimulate appetite, whereas anorexigenic neuropeptides called proopiomelanocortin (POMC) and cocaine and amphetamine-regulated transcript (CART) stimulate satiety. Because these sets of neurons are "first-order neurons where peripheral metabolic signals including leptin, insulin, ghrelin, and nutrients are primarily transferred" (Yu & Kim, 2012, p. 392) from the gut and adipose tissue (among other organs) to the hypothalamus

and brainstem, understanding how they form and function have become a mater of care and concern for appetite and obesity programming researchers, not only at my research site but also at labs across the world (Minor, Chang, & Cabo, 2010; Puig de la Bellacasa, 2017).[32] To demonstrate the degree to which researchers value the ARC's function and its control over feeding behavior (which was initially based on brain trauma injuries), one French lab recently described "[d]isruption of this regulation" between satiety and appetite neurons in the ARC as giving "rise to life-threatening conditions that include anorexia nervosa at one extreme and metabolic syndrome at the other" (Cansell, Denis, Joly-Amado, Castel, & Luquet, 2012, para. 2). From traumatic brain injuries, to epigenetic causes of obesity, the ARC develops its own history as an epistemic thing.

At my research site, PIs were interested in how neurons in the ARC form during embryogenesis (first eight weeks of development), become programmed during fetal stage of life, and possibly even re-modeled after birth due to altered newborn food environments. For example, the Lab had previously found in its food-restricted (FR) gestational model that control pups are "more normal" in terms of birth weight, but they eat more *after* they are born. The PIs "want to know what happens to the neurons that trigger hunger and satiety" among both the control and experimental groups (personal communication, February 11, 2015). Further, in a complementary experimental system, the PIs study a high-fat maternal obesity (HF) model and have found that "pups have higher amounts of appetite neurons relative to satiety ones" (personal communication, February 11, 2015). How is it that two very different gestational exposures -

[32] The ARC is not only a regulatory control site of appetite and satiety, it is also a key factor in reproduction and other areas of research, such as cancer, that relates to diseases associated with increased lifespans.

food restriction and high-fat diet- both generate similar increased appetites in the pups? Dr. Harris explained part of this empirical puzzle to the technical team and Dr. Jackson as follows:

> Stem cells migrate from the periventricular nucleus to the arcuate region, and that is where they differentiate into appetite kinds of neurons such as AgRP, NPY and POMC. This occurs during embryogenesis and early postnatal life. We have already shown that stem cells [from animals], whether they are food restricted or maternal obesity, have more of the AgRP, NPY neurons [appetite] and less of POMC [satiety]. So what we are trying to do is figure out why this is happening. There are transcription factors like Hes1, which is involved in proliferation; and Mash1 and Ngn3 are neurogenetic transcription factors that are involved in this, and we want to show the mechanisms of how this works. (Personal communication, January 2015)

This kind of convergence of two different experimental models on a similar outcome has parallels in human epidemiological research too. According to some PIs I observed, this result suggested to them a U-shaped curve between low-birth weight offspring on one end, and high birth weight on the other. Both extremes are believed to be linked to an increased risk for adult obesity and metabolic syndrome because of an increase in appetite neurons. In the food-restricted model, the thrifty-phenotype hypothesis (presented in chapter 1) asserts that small for gestational age (SGA) babies (possibly a result of maternal undernutrition or malnutrition) go through a "catch up" period during neonatal life. As a result, low birth weight babies, or uterine growth restricted babies, and those whose mothers had malnutrition show a greater risk for later life ill-health. Today, obesity researchers at my research site, and in labs across the world, employ the same logic of developmental programming and environmental plasticity of *small* babies to now study *large* babies. According to a PI at a Society for Reproductive Investigation conference I attended,

> Although there has been a huge amount of interest in the small babies, these large babies are also at risk of later life ill health, and I think this is increasingly important because we know that mean birth weight is increasing, and the instances of excess birth weight is increasing in part due to the obesity epidemic. As a contributor to later ill-health, these larger babies are increasing in importance [to the medical research community].

> If we are thinking about what an [in utero] insult could be, I think we have to postulate an insult that is happening *in utero* that impacts on birth weight and then later life ill-health. And there is increasing evidence that that insult can be under- or over- nutrition. [Studies on birth weight and BMI in adulthood] suggest to me, and it has suggested to others, that it is maternal obesity that has caused adult obesity and that birth weight has just been a mediator. Again, this is really important because maternal obesity is increasing almost exponentially. (Anonymous, personal communication, June 20, 2015)

Drawing on findings by Dr. Flier, former Dean of Faculty of Medicine at Harvard University, Dr. Harris discussed the possibility of studying apoptosis (programmed cell death that occurs naturally) in the ARC because it is thought to influence the ratio of satiety to hunger neurons (McNay, Briançon, Kokoeva, Maratos-Flier, & Flier, 2012). According to research at other labs,

> [S]tudies have showed that consumption of dietary fats promotes hypothalamic resistance to the main anorexigenic [satiety neurons] hormones, leptin and insulin, leading to the progressive loss of the balance between food intake and thermogenesis [caloric use and balance in the body] and, therefore, resulting body mass gain. (Moraes, et al., 2009, p. 1)

According to these same authors, it is the consumption of "dietary fat [not calories *per se*] that induces apoptosis of neurons and a reduction of synaptic inputs in the arcuate nucleus and lateral hypothalamus" (p. 1).

In addition to these questions about how satiety and hunger neurons are programmed during embryogenesis and fetal life, the Lab was also interested in studying another timeframe of development: the neonatal period. In one of the weekly meetings I regularly attended, Dr. Harris discussed the theory of remodeling with the other PIs and technicians, as follows:

> We have this theory about remodeling…we talked about remodeling as occurring sometime up until 12 weeks, so if we took samples [newborn mice] at birth, 4, and 8 weeks (I am just picking what we picked for the grant) and said 'Is there any apoptosis, both intrinsic and extrinsic pathways—I don't know where-- is [there] a common pathway?...One question is, 'If this remodeling is occurring, how is that happening?' There's gotta be some apoptosis you would think. This [model] is all post-natal. [We could] start with regular mice, day 1, 4weeks, 8 weeks, and staining to see whether there is anything in the arcuate? If we do, then it opens up a whole area, which means going intrinsic, extrinsic, which means comparing it to the FR [food restricted model], finding

> other [critical] dates [in the lifespan], figure out what's driving it, all of that. (Personal communication, March 16, 2015)

Dr. Harris continued:

> Some of the papers in the literature are showing that if you do a fetal injection of BrdU [a color-staining technique used to identify proliferation and cellular death that passes the placental barrier] in pregnant dams, that those cells look like they are gone by 12 weeks of age. It is still not clear to me how that happens, how you remodel [the ARC].

Dr. Moore agreed and suggested that they worked with control mice first to inject pregnant dams with BrdU, which would cross the placenta to the fetus' DNA. They would then test for cellular apoptosis at birth, 4, and 8 weeks. As the PIs mused further on this emergent system, Dr. Harris discussed part of the puzzle even further:

> But these cells, once you form a neuron, it supposedly doesn't divide so you're not going to dilute it out. And yet if you remodel, just sort of teleologically, if your neuron in the ARC already has a synaptic connection and it dies off and something else takes its place, how does it reform a connection? Then you're getting quite complicated.

Dr. Jackson quipped, "That is why we get dementia!" and Dr. Moore continued their discussion of the remodeling theory:

> So Dr. Flier's work was during fetal life. You inject BrdU [bromodeoxyuridine is a chemical compound that stains cells proliferating in a brain] to the mom and it crosses to the baby, it gets incorporated into the DNA, and it stains that cell forever. And then you go [look] at 1 day of age, 1 week of age and you say [study] how many BrdU cells are in the arcuate, and if I injected at day 10—he doesn't actually explain this well—and it is floating around, then at least to me let's say 50% of the cells in the arcuate are stained. On the other hand, that would mean 50% of the cells were born, or had their final DNA prior to day 10. He finds by one day of age that there are all of these stained cells and you can say, okay, they all came from E[embryonic day]12 to 14 or so. How you got to that conclusion is a little uncertain because did it stay in the progenitor when it was dividing into two daughter cells, or, I don't know? Let's just assume that for a moment. And then he rechecks those at 12 weeks of age and the same number of cells are there in the arcuate, at least the POMC [satiety] and NPY [appetite], but the BrdU is gone. So, oh look!, [he says,] there must be all this remodeling that is going on because how do you lose a POMC [satiety] cells that had been stained with BrdU?

"Right," Dr. Harris said, "neurons don't divide, so if you…."

"But even if they divide," Dr. Harris continued:

> Where are they coming from is one of the questions? Because usually they migrate from the periventricular to the ARC, not the other way around, so if they are reduced by end of embryonic [age], where are the others coming from? Maybe it is not the end of the embryonic [age, after all], and so is there continuing migration, or.. What's happening? We don't know.

As the PIs grappled with this new theory, they raised many questions as to the epigenetics of pregnancy, fetal programming, plasticity of critical windows of development, and definitions of the lifespan; all of which helped them assert "a sort of novel approach, a novel twist on the data" that could be used in imminent grant renewals. Dr. Harris said,

> Remember we always talked about that food restriction is the one that becomes fat, but the FRFR [gestational and neonatal food restricted] doesn't and yet both [were initially] food restricted. So one of the things we found in the literature was that in these papers by Flier [and others] was that there is more remodeling of NPY [appetite] cells (more than 95% and 90% of the POMC) but therefore there are twice as many NPYs that are not remodeled as POMCs and so that if you if mess up the NPY remodeling (I can't remember the balance of NPY and POMC)…so that was the approach we took. And now we are working on the diabetes project, which is a fat mouse and doing the same thing. *The whole grant is going to be on this somewhat suspect but novel theory of remodeling.*

"That's not going to be an easy one to substantiate," Dr. Jackson added, but the Lab already had a sense it might be possible. Dr. Harris noted that:

> It is interesting in the sense that when you look at our FR [food restricted] catch-up group, they become obese [during neonatal life as they eat in abundance]. But [mice] that were food restrict[ed] during [both] pregnancy *and* lactation, they DO NOT become obese so there is something happening because BOTH are born small. [The difference is chow during lactation period.] So there is something about neonatal nutrition that is causing…you know… they [the FR] don't eat more. They don't become obese and yet both of them were small to begin with. So something is going on in the neonatal period. What the story is, we don't know. (her emphasis)
>
> If you look at the high fat model, if you're just HF in utero, and you're nursed by a HF mom, you become fat. If you are nursed by a control mom, you don't.

Within these conversations, a point of contention was raised by Dr. Jackson and fielded by both Drs. Harris and Moore around the weight of cross-fostered HF pups. Dr. Harris said, "Yeah we kind of…in the adults…at three weeks they are still normal and have normal body

fat." But, Dr. Harris noted that even though "[t]heir body weight is not higher, their body composition shifts. Their body weights are the same, but they really reduce their lean body mass" (personal communication, March 16, 2015). This is an important detail because even if the cross-fostered pups have a "normal weight," the fact that they have less lean body mass than controls presumably affects their overall health. For example, loss of less lean body mass is associated with aging and the elderly, not a typically healthy newborn.[33] In spite of Dr. Jackson's pointed clarification, and perhaps inconvenient finding, Dr. Harris pushed past the caveat to say,

> Okay, so just staying on body weight, so they're not getting obese and the question is, they're not eating more, so what's happening now in lactation? And how is that controlling their arcuate nucleus? So now we are going to focus this diabetes grant sort of purely on the post-natal period and say remodeling must be impacted.

Throughout these conversations, PIs were thinking ahead in anticipation of how the Lab would be funded and sustained over the next several years. In the course of their everyday work, PIs had to learn to care for obesity, or "see" obesity in new ways because of their dependence on grants. Thus, drawing on previous experimental systems, and intriguing but "suspect" theories and findings in the literature, they focused on epigenetic mechanisms within the ARC both during pre- and post-natal timeframes. This allowed them to consider several new conceptual directions: within fetal programming models of disease, the thrifty-phenotype hypothesis may be less deterministic than originally believed; the epigenetics of disease may be refined through more definitive widows of exposure; and, lastly, the lifespan of obesity itself may be re-shaped due to specific nutritional exposures. For each path, there is a transformation in how biological objects (such as appetite and satiety neurons) in this instance, move from being a "neglected thing" to being a "matter of care" and "concern," and, therefore, ultimately an "epistemic thing"

[33] This points to a larger discussion in the politics of fat and obesity science around the contested meanings of weight and body-mass-index. See Saguy (2013) and Campos (2004) on this.

in the work of producing new laboratory knowledge about obesity (Puig de la Bellacasa, 2010, 2017). If the Lab could show that the effects (appetite and weight) of maternal obesity on newborn pups can be reversed via caloric restriction during lactation, it would be a significant though not unproblematic achievement for obesity sciences because body mass index would continue to trump other considerations such as skeletal muscle and fat distribution.

In order to begin advancing the new ARC re-modeling experimental system, Dr. Moore organized the pace of laying out the model. She reminded me, that "it takes a long time to get a story out of what is happening and getting a handle on what's driving this" (personal communications, February 2, 2015). "When we have a concept," Dr. Moore elaborated, "we put [together]..the study designs…It's not like we get the answer as you know in a day or two. It takes sometimes even six months to address or a year. But there are multiple studies going on to address. So one may finish this week, one may finish after two months, and then it's when we get the whole data that the whole story comes through" for us (personal communications, February 2, 2015).

Conclusion

In this chapter, I have argued that academic PIs in basic science work under particular "conditions of possibility" unique to this era of funding (Shapin, 2008, p. 229). PIs are obliged to do more communication work than experimental work. Their anticipatory caretaking labor practices are therefore at the level of inscription, not inscribing. Because of their dependence on external support, the pace of their work is largely dictated by grant-funding cycles. Established PIs, while under pressure to secure their own funds, have more intellectual freedom to pursue research questions than their subordinates.

Faced with competition for scarce tenure-track academic positions, and in light of

uncertainty in grant-based funding, younger investigators in particular face a singularly embattled plight as they try over many years to gain independence. A dichotomy exists for these new investigators where they must build on existing systems in a laboratory, but in order to secure their own funding they have to show senior or first authorship on papers. Yet, senior PIs also need senior authorship for their grants. This creates a tense situation where senior PIs must make sacrifices in order to help young investigators establish themselves.

Established PIs, while under mounting pressure to secure their own funds as well, have more intellectual freedom to pursue research questions than their subordinates. Nevertheless, for both dependent and established PIs, a precarious funding terrain affects knowledge production. "What's hot" is what is funded, and this reality not only reinscribes the medicalization of popular concerns, such as obesity and diabetes, but also siphons off money that theoretically could be spent on other empirical questions; affecting the overall scope of basic science research.

We can never know which questions go unasked in a laboratory. What I have observed is that certain aspects of a very complex biological field are made legible because the national mood is fixated on obesity as an imminent health threat to the nation's security in terms of medical costs and labor force productivity. While I am not an expert in the Lab's field of study and have not offered a critique of the science from within, I have mapped out a "regime of perceptibility" (Murphy, 2006) in which certain biological things rise to significance in the Lab. The ARC, for example, illustrates how the NIH's interest in addiction intersects with a medical problem to induce a course of action in the Lab, e.g., to study how neurons can be programmed and possibly reprogrammed to have the 'right' ratio of satiety to hunger neurons. We see, then, that by making certain biological things legible, while others remain boxed, definitions and relations between bodies and the environment shift in their meaning and significance.

Chapter 5
Conclusions

This dissertation has described the effects of economic and political precarity on a laboratory's division of labor and showed how this impacts the content and pace of laboratory fact making in obesity research. It is a case study of how the political economy of laboratory fact making shapes new obesity knowledge (Ragin & Becker, 1992). Specifically, I showed how political and economic precarity affected technicians' care for experimental practices (chapter 2). I drew on ethnographic observations of technicians' labor to illustrate variation in their *anticipatory caretaking practices*, and how this affected knowledge production. I then used observations with the handling of animal biomass to argue that animal biology was made to labor differently as a result of harmful exposures (chapter 3). Because the target laboratory studied environmental and developmental causes of obesity and metabolic syndrome, I described likewise how biomedical subjecthood became, in the context of fat, environmentally constituted. Through this process, I argued that biological labor of animals was as vital to the creation of scientific capital as human labor. Drawing on interviews and observations, I also showed how grant-based economic precarity impacted principal investigators in terms of their own *anticipatory caretaking practices* in relation to experimental trajectories (chapter 4) and what this means for knowledge production specific to obesity and basic science in general.

I defined anticipatory caretaking practices as a form of labor characterized by a persistent sense of anxiety among technicians and investigators in terms of precarity in grant renewals, publications, and visa status for the Lab's biomedical workforce. Caretaking practices, I argued, have situated meanings that vary by one's social location in the Lab's division of caretaking labor. These practices, in turn, produce epistemic effects through the Lab's experimental systems,

creating specific models of environmental and maternal-fetal models of obesity. Anticipatory caretaking practices reveal *what* kind of obesity knowledge is produced and by *whom*. They call attention to underlying social and political mechanisms that seep into laboratory "experimental systems" (Rheinberger, 1997). They help show how scientific "forms of life" are shaped by larger trends in funding and shifts in the political landscape, especially for recent immigrant scientists who are dependent on visas for their professional careers as well as their livelihood in this country (Wittgenstein, 1953). The labor they perform is gendered in that it is precarious, relatively poorly compensated, and has relatively little social status in contrast to principal investigators.

"Science," according to the late scientist Paul Kalanithi, "is as political, competitive, and fierce a career as you can find, full of the temptation to find easy paths" (as cited in Belluz, Plumer, & Resnick, 2016, introduction). As we saw, this "frightening" landscape compelled junior investigators to pursue less risky experimental projects. Senior investigators were also subject to the conservatizing force of grant funding, but their store of capital allowed them greater flexibility in choice of research questions and experimental designs. For the research site, the process of knowledge making involved a turn away from questions of fetal thirst, toward an environmental explanation of fat and appetite because of a shift in federal funding opportunities.

In addition to showing how precarity enters into experimental systems, I have also argued that the specificity of this Lab's work produces an environmental turn in obesity studies. As a consequence, new questions are raised as to what it means to medicalize conditions in developmental and environmental epigenetic models of health and disease. Also, within the frameworks of the Developmental Origins perspective and epigenetics, reproductive women are targeted for medical intervention, creating an environmental politics of reproduction. This

dissertation, then, offers a window onto how basic science research is organized today and how this affects knowledge production with consequences for the health of human (and non-human) futures. Looking through that very window we find that specific laboratory facts for one of the nation's preeminent health threats are located in maternal-fetal bodies via exposure to nutritional and chemical environments.

Contributions to the Literature

This dissertation treats theoretical scholarship on science in action (Latour, 1987), biovalue (Waldby, 2000), biopolitics (Foucault, 1997), biomedicalization (Clarke et al., 2010), ecobiopolitics (Olson, 2010), and politics of reproduction (Ginsburg & Rapp, 199) as central "sensitizing concepts" (Blumer, 1954). As a result of following technicians in action, I illustrated how anticipatory caretaking practices, as a form of laboratory labor, and variation within that labor, entered into the Lab's experimental systems (chapter 2). I then showed how the effects of caretaking labor and experimental exposures reconstituted the biology and physiology of laboratory model animal organisms. In the course of being made to live life differently due to chemical and nutritional exposure, I argued that animal biomass was productive of environmental biomedical subjecthood (chapter 3). Further, I extended Waldby's (2000) concept of biovalue to include nonhuman biological labor in the larger production of scientific capital in the form of valuable obesity data. I connected these forms of labor (anticipatory caretaking practices and biomass labor) to investigators (chapter 4); braiding together the larger national funding scene and values about health and disease in chapter 4 with the local lives and practices of PIs at the Lab. Together, these findings provide insights into concepts in science and technology studies, sociology of health and illness, gender, and work and occupations. They include: a) the study of contemporary effects of political-economy on laboratory division of

labor and its epistemic effects on knowledge production; b) the production of scientific value through the labor of animal biomass; c) and the creation of environmental biomedical subjecthood in obesity research and studies of metabolic syndrome. I illustrate these contributions below according to the order of the dissertation chapters.

Technicians and Anticipatory Caretaking Practices

In chapter 2 "Laboratory Technicians and Anticipatory Caretaking Practices," I detailed the Lab's division of caretaking labor; highlighting the embodied work of technicians at the bench. I argued that because of funding precarity (which trickled down from PIs) technicians performed experimental labor rather than PIs. In particular, I also showed how technicians' immigration status helped shape the division of caretaking labor at the Lab. I used the notion of *anticipatory caretaking practices* within this division of labor to describe the effects of different career trajectories, as either a career or temporary technician, and how this affected the kinds of experimental questions they helped pose. I argued that career technicians were able to extend the Lab's existing experimental systems in new directions whereas temporary technicians helped reproduce "normal science" because they had to work within established paradigms to quickly produce positive results for publications necessary to their professional timelines as international medical graduates. As a result, I argued that the effects of funding and immigration precarity enter into how science is done, including what questions about obesity and metabolic syndrome are posed.

The concept of anticipatory caretaking practices contributes to science, technology, and medicine studies and scientific work and occupations. I illustrated how "care" for an experimental system was shaped by technicians' own temporal positioning in the Lab. Caretaking, I have argued, is not a static concept. Rather, it varies by one's social location. In

this specific study, the effects of immigration status in particular had a conservatizing effect over temporary technicians who needed to produce "a happy story" and "positive" results in order to fulfill their occupational requirements as international medical graduates. Career technicians, on the other hand, had a long-term commitment to the Lab and were in a better position to suggest and pursue new experimental questions that would help reproduce the Lab's economic and reputational capital in the field of Developmental Origins. In both instances, the notion of care had different meanings and, therefore, created different outcomes for knowledge production.

Biological Labor and the Production of Environmental Biomedical Subjecthood

In chapter 3 "Biological Labor and the Biomedical Production of Environmental Subjecthood," I traced technicians' practices in handling biomass to show how a biomedical subject in animal models of human health were environmentally constituted. I traced two experimental systems, including the effects of maternal-high fat diet and the effects of maternal-BPA on offspring obesity and altered metabolic profiles. In doing so, I argued that animals, i.e., biomass, were environmentally constituted by nutritional and chemical exposures at different stages of life including uterine and neonatal life. As part of this argument, I showed how what "it means to be biological" is specific to environmental milieu of the Lab, which was intentionally designed to translate to human health (Landecker, 2007, 2010; Olson, 2010). I also argued in this chapter that nonhuman biomass serves national questions of health and wealth and, as a result, is productive of value for the Lab and the market for obesity facts in general. Because of this, I contended that nonhuman biological matter labors alongside human counterparts in the buildup of "lively capital" in contemporary modes of value extraction that occur at the molecular level (Haraway, 2008).

Findings in this chapter contribute to medical sociology and anthropology on the politics of reproduction. The trajectory of this scholarship has evolved to now include an environmental politics of women's (and men's) reproductive lives in relation to the rise of epigenetics and theories of developmental programming, as exemplified by Developmental Origins of Health and Disease. Biomonitoring data collected by the Centers for Disease Control suggests that humans are environmentally constituted by chemical exposures; some of which are known to produce harm such as BPA and many more which are unknown because have not been studied. How laboratory knowledge might translate to human health care practices is an emergent area of inquiry.

Findings in chapter 3 also contribute to scholarship that analyzes how value is extracted via bodies working in modes of production that are biotechnological and biomedical. I have extended a feminist approach, particularly that of Donna Haraway, that seeks to highlight less visible forms of labor to include the biological labor of biomass in the production of value, or scientific capital, in laboratory research. If data implies value and value is extracted at the molecular level and appropriated for the reproduction of a knowledge market, then the labor of animal biomass, as much if not more, is central to scientific capital accumulation; in fact, it depends on it.

Principal Investigators and Anticipatory Caretaking Practices

In chapter 4 "Principal Investigators and the Matter of Precarity," I highlighted the precarity and competitive nature of the contemporary funding landscape in biomedical and obesity research. I detailed national statistics that complemented ethnographic observations. I then argued that this particular landscape affects the investigators' "forms of [scientific] life" (Wittgenstein, 1953), producing what is seen as a "frightening" climate for investigators who

rely on grant-based funding to not only support their laboratory but their own salaries. PIs work, I argued, is communication-based rather than experimental. As such, PIs are engaged in anticipatory writing practices at the level of inscription, not inscribing. I also claimed that in meeting the NIH's demand for new obesity knowledge, the Lab (among others), reproduces the obesity market and the problematic of obesity and metabolic syndrome.

Findings from this chapter dovetail with those of chapter 2 in that funding precarity enters into how principal investigators (PIs) pursue specific research questions and experimental designs. For PIs, however, precarity varies in accordance with whether one is an established PI or a junior investigator trying to establish her independence. For both junior and senior investigators, there is an overall conservatizing impact on the creation of experimental systems because they are bound by what the NIH considers "hot" topics. However, senior investigators are able to trade on established reputational capital to pursue topics that fit within the NIH's calls for proposals but which push boundaries. The example of linking obesity to appetite in the framework of programmed addiction in hypothalamic neurons illustrates the bridging of national epidemics in order to be more competitive in grant applications. As with technicians, the role of precarity enters into decision-making and therefore affects the overall course and content of obesity knowledge production.

Study Limitations and Future Directions

This dissertation is an ethnographic study of a basic science biomedical laboratory that studies a burgeoning area in the environmental causes of obesity and metabolic syndrome. Work at my fieldsite (and elsewhere) represents what I have viewed as the physiological functioning of social nature in obesity studies. Findings from my study appear to reflect national trends in terms of grant-based funding, the composition of the biomedical workforce, and the environmental turn

in obesity studies. In that way, the ethnographic account helps provide detailed descriptions of how phenomena at the macro-level inform the lived experiences and practical realities of contemporary lab life at the micro-level.

When I entered the research site, I did not know I would find how strong the effects of funding and immigration status were on the Lab's personnel. I entered as a novice, aware only of the basic outlines and tenets of epigenetics and Developmental Origins of disease. Over time, I drew connections between the competitiveness and precarity of grant-based funding in obesity research, publication cycles, and the effects of immigration precarity on Lab personnel and how this affected experimental questions. Once I was able to understand the significance of this broader landscape on their everyday lives and practices, it became apparent how experimental questions were not only guided by personal interests but, more accurately, what everyone thought would get funded and published. The interdependence of so many connected actants and external forces that molded their careers to experimental systems raised a multitude of additional questions some of which I have outlined above. In some ways, the political economy of bench work entails perhaps intractable questions about how and in what ways we, as a nation, want to support scientific research.

We appear to be witnessing an environmental politics of reproduction. There are several implications for governance in terms of environmental and/or molecular regulation of health concerns that are situated in pregnant bodies (Shostak, 2013; Landecker & Panofsky, 2013). Further, research suggests that bodies are not equally exposed. The sciences of Developmental Origins and epigenetics reflect a "regime of perceptibility" (Murphy, 2006) that reveals to us heritable and stratified health outcomes along the lines of race and class in addition to gender. These findings, I have argued, raise questions about how and when (or if) to medicalize a

concern such as obesity in the epigenetic era. As Latour notes, "[t]he laboratory does not exist just to train students, but it is producing the means to deeply modify the practice of medicine" (1992, p. 299). What, then, does it mean to medicalize obesity (or other diseases) in an epigenetic framework in which social forces are seen as biologically causative in ways that they were not before, and when the mode of proof rests on a triangulation of evidence between epidemiology, clinical, and laboratory work (Landecker & Panofsky, 2013)? In short, what are the changing conditions of medicalization as explanations of causation shift with epigenetics? For medical sociologists, can exposomic medicalization provide a framework to account for shifts in how gene-environment interactions are studied and acted on by biomedical researchers and medical professionals?

I have been able to show how larger social forces affect obesity knowledge production and highlighted their effects on science, the scientific pipeline, and medicalization, but there are also methodological limitations to this study. First, ethnographic accounts of laboratory life are not generalizable, but in this study the findings are, in fact, supported by government data on national trends with respect to the biomedical workforce in academia. Second, my data would nonetheless have benefited from a comparison with another obesity laboratory either in academia or at a commercial site that specifically looks for developmental and environmental epigenetic answers to obesity and metabolic syndrome. Third, the analysis would have benefited from spending more time with PIs relative to technicians; although weekly meetings in particular were very revealing. Lab meetings revealed tensions about the interpretation of raw data, ideas to explore for new grants, data needed for publications, paper reviews, and how technicians' work should be organized. Lastly, one might wish that this dissertation had been able to contribute to an analysis of the regulation of ambient chemicals that are of such potential harm to human

futures. The paradox, however, is that although the threat of harm is of such importance in the shaping of my dissertation's narrative, issues around regulation were not central in the data itself because scientists—who have no reward structures for being politically engaged—say that policy making is not their job, that it is not their area of expertise. While investigators and technicians very much hope that their results will contribute to change (or that they even find negative results), they do not see advocating for regulation as part of their job.

While those at my fieldsite studied the developmental and environmental epigenetics of obesity through maternal-fetal bodies, there is also research being done on the paternal epigenetics of obesity. In studies of infertility and obesity, for example, men are increasingly subjects of investigation in relation to environmental factors that are thought to affect their fertility. A comparative case could be drawn along the lines of gender, environmental biomedical research in obesity studies, infertility, or many other areas of neurological and physiological research such as autism and disorders of sex development. The implications of an environmental politics of reproduction, therefore, extend beyond the reproductive female body, and this would be a fruitful area of future research.

Another possible future research direction incudes following PIs in the Lab who are OB/GYN doctors into their clinics. It would be useful to know how, if at all, lessons from laboratory work bleed into clinical interactions or vice-versa, i.e., how information from doctor-patient interactions with pregnant (and often overweight) women entered into their thoughts about how to create experimental designs. My sense though, from talking with PIs/MDs is that they care more about making sure patients return for regular care than to unintentionally shame or scare pregnant women by discussing potential programming effects of what they eat and how this might impact their unborn children. What matters most in the clinical interaction is to

support mothers in order that they will return for standardized pregnancy care. Lastly, in the context of clinical care with these same PIs/MDs, it would also be interesting to know how race, ethnicity, and social class impact medicalization of pregnant women in terms of clinical obesity care.

Afterward

Based on scholarship in science and technology studies, sociology of health and illness, medicine, gender, and work and occupations, I have illustrated an environmental turn in obesity that is predicated on a precarious division of caretaking labor between principal investigators and technicians in the Lab. In highlighting *anticipatory caretaking practices*, I have shown the effects of funding and political precarity on the production of new laboratory facts. While scientists, unless they were personally wealthy, have always experienced political and economic precarity (the risks of religious heresy and of requiring a noble patron are historical examples), the forms of precarity have changed. In this setting, I have articulated the ways in which various forms of labor, from humans to nonhuman biomass, braid together to produce environmental subjecthood in obesity studies through female bodies and therefore an environmental politics of reproduction. I hope my study will lead to further dialogue and investigation into the impacts that precarity has on contemporary science and the creation of facts as well as into immigrant scientists themselves, who constitute a vulnerable population within the scientific pipeline. Finally, this discussion is not limited to obesity or biomedical studies since other areas of science are implicated.

References

Adams, V., Murphy, M., & Clarke, A. (2009). Anticipation: Technoscience, life, affect, temporality. *Subjectivity, 28*(1), 246-265.

Al-Gailani, S. (2014). Making birth defects 'preventable': Pre-conceptional vitamin supplements and the politics of risk reduction. *Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Sciences, 47*, 278-289.

Almond, D., & Currie, J. (2011). Killing me softly: The fetal origins hypothesis. *The Journal of Economic Perspectives, 25*(3), 153-172. doi:10.1257/jep.25.3.153

American Diabetes Association (2015). *We are research leaders*. Retrieved from http://www.diabetes.org/research-and-practice/we-are-research-leaders/?utm_source=Offline&utm_medium=Print&utm_content=aerobic&utm_campaign=monthly

American Diabetes Association (2017). *What kind of research does the American Diabetes Association support?* Retrieved from http://www.diabetes.org/research-and-practice/we-are-research-leaders/how-we-fund-research.html

American Society for Metabolic and Bariatric Surgery (2016). *Estimate of bariatric surgery numbers, 2011-2016*. Retrieved from https://asmbs.org/resources/estimate-of-bariatric-surgery-numbers

Åsberg, C., & Lykke, N. (2010). Feminist technoscience studies. *European Journal of Women's Studies, 17*(4), 299-305. doi:10.1177/1350506810377692

Association of American Medical Colleges (2015). *2015 State physician workforce data book*. Retrieved from https://members.aamc.org/eweb/upload/2015StateDataBook%20(revised).pdf

Astwood, E. B. (1962). The heritage of corpulence. *Endocrinology, 71*(2), 337-341. doi:10.1210/endo-71-2-337

Baird, J., Jacob, C., Barker, M., Fall, H. C., Hanson, M., Harvey, C. N., . . . Cooper, C. (2017). Developmental origins of health and disease: A lifecourse approach to the prevention of non-communicable diseases. *Healthcare, 5*(1). doi:10.3390/healthcare5010014

Baele, S. (2008). *An interview with Thomas Lemke: Michel Foucault today*. Retrieved from http://www.revue-emulations.net/interviews/interviews2/thomaslemke-foucaulttoday

Balcombe, J.P. (2006). Laboratory environments and rodents' behavioural needs: A review. *Laboratory Animals, 40*(3), 217-235.

Barad, K. (2003). Posthumanist performativity: Toward an understanding of how matter comes to matter. *Signs, 28*(3), 801-831. doi:10.1086/345321

Barker, D. J. (1990). The fetal and infant origins of adult disease. *BMJ: British Medical Journal, 301*(6761), 1111.

Barker, D. J. (1995). Fetal origins of coronary heart disease. *The BMJ, 311*(6998), 171-174.

Barker, D.J. (1998). In utero programming of chronic disease. *Clinical Science*, 95(2), 115-128.

Barker, D.J., & Osmond, C. (1986). Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *Lancet, 1*(8489), 1077-1081. doi:10.1016/S0140-6736(86)91340-1

Barley, S.R., & Bechky, B.A. (1994). In the backrooms of science: The work of technicians in science labs. *Work and Occupations, 21*, 85–126.

Barley, S.R., & Orr, J. (1997). *Between craft and science: Technical work in the United States*. Ithaca, NY: ILR Press.

Barinaga, M. (2000). Soft money's hard realities. *Science, 289*(5487), 2024-2028. doi:10.1126/science.289.5487.2024

Bateson, P., Gluckman, P., & Hanson, M. (2014). The biology of developmental plasticity and the predictive adaptive response hypothesis. *The Journal of Physiology, 592*(11), 2357-2368. doi:10.1113/jphysiol.2014.271460

Batista, T. M., Alonso-Magdalena, P., Vieira, E., Amaral, M. E. C., Cederroth, C. R., Nef, S., . . . Nadal, A. (2012). Short-term treatment with bisphenol-A leads to metabolic abnormalities in adult male mice. *PLOS ONE, 7*(3), e33814. doi:10.1371/journal.pone.0033814

Becker, H. (1998). *Tricks of the trade: How to think about your research while you're doing it*. Chicago, Il: The University of Chicago Press.

Bell, M. L., & Ebisu, K. (2012). Environmental inequality in exposures to airborne particulate matter components in the United States. *Environmental Health Perspectives, 120*(12), 1699-1704. doi:10.1289/ehp.1205201

Bell, S.E., & Figert, A.E. (Eds.). (2015). *Reimagining (bio)medicalization, pharmaceuticals and genetics: Old critiques and new engagements*. New York, NY: Routledge.

Belluz, J., Plumer, B., & Resnick, B. (2016). *The 7 biggest problems facing science, according to 270 scientists*. Retrieved from https://www.vox.com/2016/7/14/12016710/science-challeges-research-funding-peer-review-process

Belsky, D. (2014). Appetite for prevention: Genetics and developmental epidemiology join forces in obesity research. *JAMA Pediatrics, 168*(4), 309-311. doi:doi:10.1001/jamapediatrics.2013.5291

Benderley, B.L. (2010). *Does the U.S. produce too many scientists?* Retrieved from https://www.scientificamerican.com/article/does-the-us-produce-too-m/

Bennett, J. (2010). *Vibrant matter: A political ecology of things*. Durham, NC: Duke University Press.

Bliss, C. (2015). Defining health justice in the postgenomic era. In S. Richardson & H. Stevens (Eds.), *Postgenomics: Perspectives on biology after the genome.* Durham, NC: Duke University Press.

Blumer, H. (1954). What is wrong with social theory? *American Sociological Review, 19*(1), 3-10. doi:10.2307/2088165

Blumer, H. (1986). *Symbolic interactionism: Perspective and method*. Oakland, CA: University of California Press.

Bourdieu, P. (1975). The specificity of the scientific field and the social conditions of the progress of reason. *Social Science Information, 14*(6), 19-47.

Bourdieu, P. (1986). The forms of capital. In J. Richardson (Ed.), *Handbook of theory and research for the sociology of education* (pp. 241-258). Westport, CT: Greenwood.

Bourdieu, P. (1998). *Acts of resistance: Against the tyranny of the market* (R. Nice, Trans.). New York, NY: The New Press and Polity Press.

Bourdieu, P. (1999). *The weight of the world: Social suffering in contemporary society*. Stanford, CA: Stanford University Press.

Bourdieu, P. (2004) *Science of science and reflexivity*. Chicago, Il: University of Chicago Press.

Bourdieu, P. (2013). Symbolic capital and social classes. *Journal of Classical Sociology*, *13*(2), 292-302.

Boutant, M., & Cantó, C. (2014). SIRT1 metabolic actions: Integrating recent advances from mouse models. *Molecular Metabolism, 3*(1), 5-18.

Bray, G. A. (2015). Why obesity? *The Annual Review of Nutrition, 35*, 1-31.

Brodwin, P. (2000). *Biotechnology and culture : Bodies, anxieties, ethics*. Bloomington, IN: Indiana University Press.

Brown, P. (1995). Race, class, and environmental health: A review and systematization of the literature. *Environmental Research, 69*(1), 15-30. doi:https://doi.org/10.1006/enrs.1995.1021

Buettner, R., Parhofer, K. G., Woenckhaus, M., Wrede, C. E., Kunz-Schughart, L. A., Schölmerich, J., & Bollheimer, L. C. (2006). Defining high-fat-diet rat models: Metabolic and molecular effects of different fat types. *Journal of Molecular Endocrinology, 36*(3), 485-501. doi:10.1677/jme.1.01909

Buklijas, T. (2013). Food, growth and time: Elsie Widdowson's and Robert McCance's research into prenatal and early postnatal growth. *Studies in History and Philosophy of Biological and Biomedical Sciences, 47*, 267-277.

Burawoy, M. (1998). The extended case method. *Sociological Theory, 16*(1), 4-33. doi:10.1111/0735-2751.00040

Burger, M., Drummond, J. C., Sandstead, H. R., & Netherlands Committee on Malnutrition (1948). *Malnutrition and starvation in Western Netherlands : September 1944 - July 1945 Pt. 1*. The Hague, Netherlands: General State Print Office.

Burggren, W. W., & Mueller, C. A. (2015). Developmental critical windows and sensitive periods as three-dimensional constructs in time and space. *Physiological and Biochemical Zoology, 88*(2), 91-102. doi:10.1086/679906

Callon, M. (1984). Some elements of a sociology of translation: Domestication of the scallops and the fishermen of St Brieuc Bay. *The Sociological Review, 32*(1_suppl), 196-233. doi:10.1111/j.1467-954X.1984.tb00113.x

Campos, P. (2004) *The obesity myth: Why America's obsession with weight is hazardous to your health*. New York, NY: Gotham Books.

Canguilhem, G. (2001). The living and its milieu. *Grey Room,* -(3), 7-31. doi:10.1162/152638101300138521

Cansell, C., Denis, R. G. P., Joly-Amado, A., Castel, J., & Luquet, S. (2012). Arcuate AgRP neurons and the regulation of energy balance. *Frontiers in Endocrinology, 3*, 169. doi:10.3389/fendo.2012.00169

Carmo-Silva, S., & Cavadas, C. (2017). Hypothalamic dysfunction in obesity and metabolic disorders. In L. Letra & R. Seiça (Eds.), *Obesity and brain function* (pp. 73-116). Cham, Switzerland: Springer International Publishing.

Cedikova, M., Kripnerová, M., Dvorakova, J., Pitule, P., Grundmanova, M., Babuska, …& Kuncova, J. (2016). Mitochondria in white, brown, and beige adipocytes. *Stem Cells International, 2016*, 6067349. doi:10.1155/2016/6067349

Charmaz, K. (1995). The body, identity, and self: Adapting to impairment. *The Sociological Quarterly, 36*(4), 657-680. doi:10.1111/j.1533-8525.1995.tb00459.x

Charmaz, K. (2003). Grounded theory: Objectivist and constructivist methods. In N. K. Denzin, & Y. S. Lincoln (Eds.), *Strategies for qualitative inquiry* (2nd ed., pp. 249-291). Thousand Oaks, CA: Sage Publications.

Charmaz, K. (2006) *Constructing grounded theory: A practical guide through qualitative analysis*. Thousand Oaks, CA: Sage Publications.

Chavatte-Palmer, P., Tarrade, A., & Rousseau-Ralliard, D. (2016). Diet before and during pregnancy and offspring health: The importance of animal models and what can be learned from them. *International Journal of Environmental Research and Public Health*, *13*(6), 586. MDPI AG. doi:10.3390/ijerph13060586

Centers for Disease Control and Prevention (2009). *Fourth national report on human exposure to environmental chemicals*. Retrieved from https://www.cdc.gov/exposurereport/pdf/FourthReport_ExecutiveSummary.pdf

Centers for Disease Control and Prevention (2012). *Obesity is common, serious, and costly*. Retrieved from https://www.cdc.gov/media/matte/2012/05_weight_of_nation.pdf

Centers for Disease Control and Prevention (2015). *Defining childhood obesity*. Retrieved from https://www.cdc.gov/obesity/childhood/defining.html

Centers for Disease Control and Prevention (2016). *Adult obesity facts*. Retrieved from http://www.cdc.gov/obesity/data/adult.html

Centers for Disease Control and Prevention (2017). *Adult obesity causes and consequences*. Retrieved from https://www.cdc.gov/obesity/adult/causes.html

Clarke, A. (2005). *Situational analysis: Grounded theory after the postmodern turn.* Thousand Oaks, CA: Sage Publications.

Clarke, A.E., Shim, J.K., Mamo, L., Fosket, J. R., & Fishman, J.R. (2003). Biomedicalization: Technoscientific transformations of health, illness, and U.S. biomedicine. *American Sociological Review, 68*(2), 161-194.

Clarke, A.E., Mamo, L., Fosket, J.R., Fishman, J.R. & Shim, J.K. (Eds.). (2010). *Biomedicalization: Technoscience, Health, and Illness in the U.S.* Durham, NC: Duke University Press.

Conrad, P. (1992). Medicalization and social control. *Annual Review of Sociology 18*, 209-232.

Conrad, P. (2005). The shifting engines of medicalization. *Journal of Health and Social Behavior, 46*(1), 3-14.

Conrad, P., & Schneider, J. (1992). *Deviance and medicalization: From badness to sickness* (Exp. ed.). Philadelphia, PA: Temple University Press.

Cooper, C. (2013). David Barker obituary. *The Guardian*. Retrieved from https://www.theguardian.com/society/2013/sep/11/david-barker

Cooper, M., & Waldby, C. (2014). *Clinical labor: Tissue donors and research subjects in the global bioeconomy*. Durham, NC: Duke University Press.

Corton, J. C., Peters, J. M., & Klaunig, J. E. (2017). The PPARα-dependent rodent liver tumor response is not relevant to humans: Addressing misconceptions. *Archives of Toxicology*, *92*(1), 83-119. doi:10.1007/s00204-017-2094-7

Crutzen, P.J., & Stoermer, E.F. (2000). The "anthropocene". *Global Change Newsletter*, *41*, 17.

Curley, J. P., Mashoodh, R., & Champagne, F. A. (2011). Epigenetics and the origins of paternal effects. *Hormones and Behavior, 59*(3), 306-314. doi:10.1016/j.yhbeh.2010.06.018

Day, J., Savani, S., Krempley, B. D., Nguyen, M., & Kitlinska, J. B. (2016). Influence of paternal preconception exposures on their offspring: Through epigenetics to phenotype. *American Journal of Stem Cells, 5*(1), 11-18.

Deans, C., & Maggert, K. A. (2015). What do you mean, "epigenetic"? *Genetics, 199*(4), 887-896. doi:10.1534/genetics.114.173492

Desai, M., Jellyman, J. K., & Ross, M. G. (2015). Epigenomics, gestational programming and risk of metabolic syndrome. *International Journal of Obesity, 39*(4), 633-641. doi:10.1038/ijo.2015.13

Diamanti-Kandarakis, E., Bourguignon, J.-P., Giudice, L. C., Hauser, R., Prins, G. S., Soto, A. M., . . . Gore, A. C. (2009). Endocrine-disrupting chemicals: An endocrine society scientific statement. *Endocrine Reviews, 30*(4), 293-342. doi:10.1210/er.2009-0002

Doing, P. (2008). Give me a laboratory and I will raise a discipline: The past, present, and future politics of laboratory studies in STS. In O. A. E.J. Hackett, M. Lynch & J. Wajcman (Eds.), *The handbook of science and technology studies* (3 ed., pp. 279). Boston, MA: Massachusetts Institute of Technology.

Donahue, W. (2017). Trust in technicians in paleontology laboratories. *Science, Technology, & Human Values, 43*(2), 324-348. doi:10.1177/0162243917722844

Dreger, A. D. (1998). *Hermaphrodites and the medical invention of sex*. Harvard, MA: Harvard University Press.

Eaton, S. A., Aiken, A. J., Young, P. E., Ho, J. W. K., Cropley, J. E., & Suter, C. M. (2017). Maternal obesity heritably perturbs offspring metabolism for three generations without serial programming. *International Journal of Obesity*. doi:10.1038/ijo.2017.247

Edwards, J., Harvey, P., & Wade, P. (2010). *Technologized images, technologized bodies*. New York, NY: Berghahn Books.

Engels, F. (1845). *The condition of the working class in England*. Leipzig, Germany: Otto Wigand.

Faloia, E., De Robertis, M., Luconi, M. P., Furlani, G., & Boscaro, M. (2012). Inflammation as a link between obesity and metabolic syndrome. *Journal of Nutrition and Metabolism,12*. doi:10.1155/2012/476380

Farmer, S. R. (2006). Transcriptional control of adipocyte formation. *Cell Metabolism, 4*(4), 263-273. doi:10.1016/j.cmet.2006.07.001

Farooqi, S., & O'Rahilly, S. (2017). The genetics of obesity in humans. In L.J. Groots, G. Chrousos, K. Dungan et al. (Eds.), *Endotext [Internet]*. Retrieved from https://www.ncbi.nlm.nih.gov/books/NBK279064/

Faubion, J. D., & Marcus, G. E. (2009). *Fieldwork is not what it used to be : Learning anthropology's method in a time of transition*. Ithaca, NY: Cornell University Press.

Fortun, K., & Fortun, M. (2005). Scientific imaginaries and ethical plateaus in contemporary U.S. toxicology. *American Anthropology, 107*(1), 43-54. doi:10.1525/aa.2005.107.1.043

Fortun, M. (2015) What toll pursuit: Affective assemblages in genomics and postgenomics. In S. S. Richardson & H. Stevens (Eds.), *Postgenomics: Perspectives on biology after the genome* (pp. 32-55). Durham, NC: Duke University Press.

Fothergill, E. Guo, J., Howard, L., Kerns, J,C., Knuth,N.D,…& Hall, K.D. (2016). *Persistent metabolic adaptation 6 years after "The Biggest Loser" competition*. Retrieved from https://onlinelibrary.wiley.com/doi/full/10.1002/oby.21538

Foucault, M. (1997). The birth of biopolitics. In P. Rabinow & J.D. Faubion (Eds.) *Ethics, subjectivity and truth: Essential works of Foucault* (Vol. 1, pp 73-79). New York, NY: New Press.

Foucault, M. (1978). *The history of sexuality*. New York, NY: Pantheon Books.

Foucault, M. (2003). *Society must be defended: Lectures at the Collège de France 1975-76* (D. Macey, Trans.). New York, NY: Picador.

Foucault, M. (2008). *The birth of biopolitics: Lectures at the Collège of France 1978-79* (G. Burchell, Trans.). New York, NY: Palgrave Macmillan.

Franklin, S. (2006). The cyborg embryo: Our path to transbiology. *Theory, Culture & Society, 23*(7-8), 167-187. doi:10.1177/0263276406069230

Friese, C. (2013). *Cloning wild life. Zoos, captivity, and the future of endangered animals*. New York, NY: NYU Press.

Garrison, L. (2013). *Knowledge, resistance and labor*. Retrieved from https://anthrobiopolitics.wordpress.com/

Ghosal, S., Nunley, A., Mahbod, P., Lewis, A. G., Smith, E. P., Tong, J., . . . Herman, J. P. (2015). Mouse handling limits the impact of stress on metabolic endpoints. *Physiology & Behavior, 150*, 31-37. doi:10.1016/j.physbeh.2015.06.021

Ginsburg, F., & Rapp, R. (1991). The politics of reproduction. *Annual Review of Anthropology, 20*(1), 311-343. doi:10.1146/annurev.an.20.100191.001523

Glaser, B. G., & Strauss, A. L. (1999). *Discovery of grounded theory*. New York, NY: Routledge.

Gluckman, P. D., & Hanson, M. A. (2004). Developmental origins of disease paradigm: A mechanistic and evolutionary perspective. *Pediatric Research, 56*(3), 311-317.

Gluckman, P. D., Hanson, M. A., & Beedle, A. S. (2007). Early life events and their consequences for later disease: A life history and evolutionary perspective. *American Journal of Human Biology, 19*(1), 1-19. doi:10.1002/ajhb.20590

Gluckman, P. D., Hanson, M. A., & Buklijas, T. (2009). A conceptual framework for the developmental origins of health and disease. *Journal of Developmental Origins of Health and Disease, 1*(1), 6-18. doi:10.1017/S2040174409990171

Gluckman, P. D., Hanson, M. A., Cooper, C., & Thornburg, K. L. (2008). Effect of in utero and early-life conditions on adult health and disease. *The New England Journal of Medicine, 359*(1), 61-73. doi:10.1056/NEJMra0708473

Gooday, B. G. (2008). Placing or replacing the laboratory in the history of science? *Isis, 99*(4), 783-795. doi:10.1086/595772

Gore, A. C., Chappell, V.A., Fenton, S.E., Flaws, J.A., Nadal, A., Prins, G.S., …, Zoeller, R.T. (2015). EDC-2: The Endocrine Society's second scientific statement on endocrine-disrupting chemicals. *Endocrine Reviews, 36*(6), E1–E150. doi: 10.1210/er.2015-1010

Grün, F. & Blumberg, B.(2009a). Endocrine disrupters as obesogens. *Molecular and Cellular Endocrinology, 304*, 19–29

Grün, F., & Blumberg, B. (2009b). Minireview: The case for obesogens. *Molecular Endocrinology, 23*(8), 1127-1134. doi:10.1210/me.2008-0485

Guthman, J. (2011). *Weighing in: Obesity, food justice, and the limits of capitalism*. Los Angeles, CA: University of California Press.

Guthman, J. (2012). *Plastic bodies: Biopolitical implications of the new environmental epigenetics*. Los Angeles, CA: University of Southern California.

Guthman, J., & Mansfield, B. (2012). The implications of environmental epigenetics: A new direction for geographic inquiry on health, space, and nature-society relations. *Progress in Human Geography, 37*(4), 486-504.

Hales, C. N., & Barker, D. J. P. (1992). Type 2 (non-insulin-dependent) diabetes mellitus: The thrifty phenotype hypothesis. *Diabetologia, 35*(7), 595-601. doi:10.1007/bf00400248

Hales, C. N., & Barker, D. J. P. (2001). The thrifty phenotype hypothesis: Type 2 diabetes. *British Medical Bulletin, 60*(1), 5-20. doi:10.1093/bmb/60.1.5

Hanson, M. A., & Gluckman, P. D. (2014). Early developmental conditioning of later health and disease: Physiology or pathophysiology? *Physiological Reviews*, *94*(4), 1027–1076. http://doi.org/10.1152/physrev.00029.2013

Haraway, D. (1988). Situated knowledges: The science question in feminism and the privilege of partial perspective. *Feminist Studies, 14*(3), 575-599. doi:10.2307/3178066

Haraway, D. (1990). A manifesto for cyborgs: Science, technology, and socialist feminism in the 1980s. In L. Nicholson (Ed.) *Feminism/Postmodernism* (pp.190-233). New York, NY: Routledge.

Haraway, D. (1997). *Modest_witness@second_millennium. Femaleman@ _ meets_ oncomouse TM: Feminism and technoscience*. New York, NY: Routledge.

Haraway, D. (2008). *When species meet*. Minneapolis, MN: University of Minnesota Press.

Hardt, M. (1999). Affective labor. *Boundary 2, 26*(2), 89-100.

Hardt, M., & Negri, A. (2000). *Empire*. Cambridge, MA: Harvard University Press.

Haugen, A. C., Schug, T. T., Collman, G., & Heindel, J. (2014). Evolution of DOHaD: The impact of environmental health sciences. *Journal of Developmental Origins of Health and Disease, 6*(2), 55-64. doi: 10.1017/S2040174414000580

Heindel, J. J., Newbold, R., & Schug, T. T. (2015). Endocrine disruptors and obesity. *Nature Reviews Endocrinology, 11*, 653. doi:10.1038/nrendo.2015.163

Helmreich, S. (2008). Species of biocapital. *Science as Culture, 17*(4), 463-478. doi:10.1080/09505430802519256

Hendryx, M., & Luo, J. (2018). Children's environmental chemical exposures in the USA, NHANES 2003–2012. *Environmental Science and Pollution Research, 25*(6), 5336-5343. doi:10.1007/s11356-017-0874-5

Herzig, R., & Subramanian, B. (2017). Labor in the age of "bio-everything". *Radical History Review, 127*, 103-124. doi:10.1215/01636545-3690894

Hess, D. (2001). Ethnography and the development of science and technology studies. In P. Atkinson, A. Coffey, S. Delamont, J. Lofland, & L. Lofland (Eds.) *Sage Handbook of Ethnography* (pp. 234-245). Thousand Oaks, CA: Sage Publications.

Holtcamp, W. (2012). Obesogens: An environmental link to obesity. *Environmental Health Perspectives, 120*(2), 62-68. doi: http://doi.org/10.1289/ehp.120-a62

Howard Hughes Medical Institute (2008). *Discovering the obesity genes*. Retrieved from https://www.cdc.gov/obesity/adult/causes.html

Huang, P. L. (2009). A comprehensive definition for metabolic syndrome. *Disease Models & Mechanisms, 2*(5-6), 231-237. doi:10.1242/dmm.001180

Iliffe, R. (2008). Technicians. *Notes and Records of the Royal Society, 62*, 3-16.

Ingalls, A. M., Dickie, M. M., & Snell, G. D. (1950). Obese, a new mutation in the house mouse. *Journal of Heredity, 41*(12), 317-318.

Jacob, F. (1988). *The statue within: An autobiography: F. Jacob* (F. Philip, Trans.). London, UK: Unwin Hyman.

Janesick, A., & Blumberg, B. (2011). Endocrine disrupting chemicals and the developmental programming of adipogenesis and obesity. *Birth Defects Research. Part C: Embryo Today: Reviews, 93*(1), 34-50. doi:10.1002/bdrc.20197

Janesick, A. S., & Blumberg, B. (2016). Obesogens: An emerging threat to public health. *American Journal of Obstetrics and Gynecology, 214*(5), 559-565. doi:10.1016/j.ajog.2016.01.182

Janvier Labs (2018). *Sprague Dawley*. Retrieved from https://www.janvier-labs.com/rodent-research-models-services/research-models/per-species/outbred-rats/product/sprague-dawley.html

Johnson, L. (2016, October). *Get off your duff! Sitting is the 'new smoking'*. Retrieved from http://www1.cbn.com/cbnnews/healthscience/2015/December/Scam-Tracker-Helps-Shoppers-During-Christmas-Season

Jungheim, E. S., Travieso, J. L., & Hopeman, M. M. (2013). Weighing the impact of obesity on female reproductive function and fertility. *Nutrition Reviews, 71*(0 1), 10.1111/nure.12056. doi:10.1111/nure.12056

Kaiser, D. (2011). The search for clean cash. *Nature, 472*(7341), 30-31.

Kamakura, R., Kovalainen, M., Leppäluoto, J., Herzig, K. H., & Mäkelä, K. A. (2016). The effects of group and single housing and automated animal monitoring on urinary corticosterone levels in male C57BL/6 mice. *Physiological Reports, 4*(3), e12703. doi:10.14814/phy2.12703

Kang, M. (2010). *The managed hand: Race, gender, and the body in beauty service work*. Berkeley, CA: University of California Press.

Karkazis, K. (2008). *Fixing sex: Intersex, medical authority and lived experience*. Stanford, CA: Stanford University Press.

Kava, R., Greenwood, M. R. C., & Johnson, P. R. (1990). Zucker (fa/fa) rat. *ILAR Journal, 32*(3), 4-8. doi:10.1093/ilar.32.3.4

Keller, E.F. (1983). *A Feeling for the organism: The life and work of Barbara McClintock*. New York, NY: W.H. Freeman and Company.

Keller, E. F. (2014). From gene action to reactive genomes. *The Journal of Physiology, 592*(11), 2423-2429. doi:10.1113/jphysiol.2014.270991

Kelty, C., & Landecker, H. (2009). Ten thousand journal articles later: Ethnography of «the literature» in science. *Empiria, 18*, 173-192.

Khullar, D., Blumenthal, D. M., Olenski, A. R., & Jena, A. B. (2017). U.S. immigration policy and American medical research: The scientific contributions of foreign medical graduates. *Annals of Internal Medicine, 167*(8), 584-586. doi:10.7326/M17-1304

Kirk, R.G.W. (2014). The invention of the "stressed animal" and the development of a science of animal welfare, 1947–86. In D. Cantor & E. Ramsden (Eds.) *Stress, shock, and adaptation in the twentieth century* (pp. 241-263). Rochester, NY: University of Rochester Press.

Klimentidis, Y. C., Beasley, T. M., Lin, H.Y., Murati, G., Glass, G. E., Guyton, M., . . . Allison, D. B. (2011). Canaries in the coal mine: A cross-species analysis of the plurality of obesity epidemics. *Proceedings of the Royal Society B: Biological Sciences, 278*(1712), 1626-1632. doi:10.1098/rspb.2010.1890

Knorr Cetina, K. (1999). *Epistemic cultures: How the sciences make knowledge*. Cambridge, MA: Harvard University Press.

Kolata, G. (2016). *After 'the biggest loser,' their bodies fought to regain weight*. Retrieved from https://www.nytimes.com/2016/05/02/health/biggest-loser-weight-loss.html

Kolodny, A., Courtwright, D. T., Hwang, C. S., Kreiner, P., Eadie, J. L., Clark, T. W., & Alexander, G. C. (2015). The prescription opioid and heroin crisis: A public health approach to an epidemic of addiction. *Annual Review of Public Health, 36*(1), 559-574. doi:10.1146/annurev-publhealth-031914-122957

Kuhn, T.S. (2012). *The structure of scientific revolutions: 50th anniversary edition*. Chicago, Il: University of Chicago Press.

Lajonchere, C. (2011). *The womb as environment*. Retrieved from https://www.autismspeaks.org/science/science-news/womb-environment

Landecker, H. (2007). *Culturing life: How cells became technologies*. Cambridge, MA: Harvard University Press.

Landecker, H. (2010). Living differently in time: Plasticity, temporality and cellular biotechnologies. In J. Edwards, P. Harvey, & P. Wade (Eds.) *Technologized images, technologized bodies* (pp. 211-233). New York, NY: Berghahn Books.

Landecker, H. (2011). Food as exposure: Nutritional epigenetics and the new metabolism. *BioSocieties, 6*(2), 167-194. doi:10.1057/biosoc.2011.1

Landecker, H., & Panofsky, A. (2013). From social structure to gene regulation, and back: A critical introduction to environmental epigenetics for sociology. *Annual Review of Sociology, 39*, 333-357.

Landrigan, P. J., Sonawane, B., Mattison, D., McCally, M., & Garg, A. (2002). Chemical contaminants in breast milk and their impacts on children's health: An overview. *Environmental Health Perspectives, 110*(6), A313-A315.

Lappé, M. (2014). Taking care: Anticipation, extraction and the politics of temporality in autism science. *BioSocieties, 9*, 304-328.

Lappé, M. (2016). The maternal body as environment in autism science. *Social Studies of Science, 46*(5), 1-26.

Lappé, M., & Landecker, H. . (2015). How the genome got a lifespan. *New Genetics and Society, 34*(2), 152-176.

Latimer, J., & Puig de la Bellacasa, M. (2013). Re-thinking the ethical: Everyday shifts of care in biogerontology. In N. M. Priaulx & A.Wrigley (Eds.), *Ethics, law and society* (pp.153-174). Surrey, UK: Ashgate.

Latour, B. (1983). *Give me a laboratory and I will raise the world*. Retrieved from http://www.bruno-latour.fr/sites/default/files/12-GIVE-ME-A-LAB-GB.pdf

Latour, B. (1987). *Science in action: How to follow scientists and engineers through society*. Cambridge, MA: Harvard University Press.

Latour, B. (1992). The costly ghastly kitchen. In A. Cunningham & P. Williams (Eds.), *The laboratory revolution in medicine*. Cambridge, UK: Cambridge University Press.

Latour, B., & Woolgar, S. (1979). *Laboratory life: The construction of scientific facts*. Princeton, NJ: Princeton University Press.

Lauer, M. (2017). *Data on trends according to career stage*. Retrieved from https://nexus.od.nih.gov/all/2017/08/31/data-on-trends-according-to-career-stage/

Lee, C. (2007, May 28). Slump in NIH funding is taking toll on research. *The Washington Post*. Retrieved from http://www.washingtonpost.com/wp-dyn/content/article/2007/05/27/AR2007052700794.html

Lee, R. C. (2014). *The exquisite corpse of Asian America: Biopolitics, biosociality, and posthuman ecologies*. New York, NY: NYU Press.

Lemke, T. (2011). *Biopolitics: An advanced introduction*. New York, NY: New York University Press.

Li, X. (2013). SIRT1 and energy metabolism. *Acta Biochimica et Biophysica Sinica, 45*(1), 51-60. doi:10.1093/abbs/gms108

Liu, M., & Mallon, W.T. (2004). Tenure in transition: Trends in basic science faculty appointment policies at U.S. medical schools. *Academic Medicine, 79*(3), 205-213.

Lock, M. (2013). The lure of the epigenome. *The Lancet, 381*(9881), 1896-1897. doi:10.1016/S0140-6736(13)61149-6

Luquet, S., Perez, F. A., Hnasko, T. S., & Palmiter, R. D. (2005). NPY/AgRP neurons are essential for feeding in adult mice but can be ablated in neonates. *Science* 310, 683–685.

Lynch, M. (1985). *Art and artifact in laboratory science: A study of shop work and shop talk in a research laboratory*. London, UK: Routledge & Kegan Paul

Lynch, M., Cole, S.A., McNally, R., & Jordan, K. (2008). *Truth machine. The contentious history of DNA fingerprinting*. Chicago, Il: University of Chicago Press.

MacKay, H., Patterson, Z. R., Khazall, R., Patel, S., Tsirlin, D., & Abizaid, A. (2013). Organizational effects of perinatal exposure to bisphenol-A and diethylstilbestrol on

arcuate nucleus circuitry controlling food intake and energy expenditure in male and female CD-1 mice. *Endocrinology, 154*(4), 1465-1475. doi:10.1210/en.2012-2044

Marcus, G. E. (1995). Ethnography in/of the world system: The emergence of multi-sited ethnography. *Annual Review of Anthropology, 24*(1), 95-117. doi:10.1146/annurev.an.24.100195.000523

Martin, A., Myers, N., & Viseu, A. (2015). The politics of care in technoscience. *Social Studies of Science, 45*(5), 625-641. doi:10.1177/0306312715602073

Martin, B., Ji, S., Maudsley, S., & Mattson, M. P. (2010). "Control" laboratory rodents are metabolically morbid: Why it matters. *Proceedings of the National Academy of Sciences, 107*(14), 6127-6133. doi:10.1073/pnas.0912955107

McAllister, E., Dhurandhar N.V., Keith, S.W, Aronne, L. J., Barger, J., Baskin, M., …Allison, D. B. (2009). Ten putative contributors to the obesity epidemic. *Critical Review Food Science Nutrition, 49*(10), 868-913.

McCance, R. A., & Widdowson, E. M. (1974). Review lecture: The determinants of growth and form. *Proceedings of the Royal Society of London. Series B. Biological Sciences, 185*(1078), 1-17.

McDermott, M.T. (2016). *Readers respond: The biggest loser*. Retrieved from https://www.nytimes.com/2016/05/05/health/readers-respond-the-biggest-loser.html?action=click&contentCollection=Opinion&module=RelatedCoverage&region=%20Marginalia&pgtype=article

McNay, D.E.G., Briançon, N., Kokoeva, M.V., Maratos-Flier, E., & Flier, J.S. (2012). Remodeling of the arcuate nucleus energy-balance circuit is inhibited in obese mice. *The Journal of Clinical Investigation, 122*(1), 142-152. doi:10.1172/JCI43134.

Mead, M. N. (2008). Contaminants in human milk: Weighing the risks against the benefits of breastfeeding. *Environmental Health Perspectives, 116*(10), A426-A434.

Miller, G. W., & Jones, D. P. (2014). The nature of nurture: Refining the definition of the exposome. *Toxicological Sciences, 137*(1), 1-2. doi:10.1093/toxsci/kft251

Minor, R. K., Chang, J. W., & de Cabo, R. (2009). Hungry for life: How the arcuate nucleus and neuropeptide Y may play a critical role in mediating the benefits of calorie restriction. *Molecular and Cellular Endocrinology, 299*(1), 79-88. doi:10.1016/j.mce.2008.10.044

Mitchell, R. (2010). National biobanks: Clinical labor, risk production, and the creation of biovalue. *Science, Technology & Human Values, 35*(3), 330-355. doi:10.1177/0162243909340267

Mol, A. (2002) *The body multiple: Ontology in medical practice*. Durham, NC: Duke University Press.

Moraes, J. C., Coope, A., Morari, J., Cintra, D. E., Roman, E. A., Pauli, J. R.,… Velloso, L. A. (2009). High-fat diet induces apoptosis of hypothalamic neurons. *PLoS ONE 4*(4), e5045. doi:10.1371/journal.pone.0005045

Mullainadhan, V., Viswanathan, M. P., & Karundevi, B. (2017). Effect of bisphenol-A (BPA) on insulin signal transduction and GLUT4 translocation in gastrocnemius muscle of adult male albino rat. *The International Journal of Biochemistry & Cell Biology, 90*, 38-47. doi:https://doi.org/10.1016/j.biocel.2017.07.015

Müller, R., & Kenney, M. (2014). Agential conversations: Interviewing postdoctoral life scientists and the politics of mundane research practices. *Science as Culture, 23*(4), 537-559. doi:10.1080/09505431.2014.916670

Müller-Wille, S., & Rheinberger, H-J. (2012). *A cultural history of heredity*. Chicago, Il: The University of Chicago Press.

Murphy, M. (2006). *Sick building syndrome and the problem of uncertainty: Environmental politics, technoscience, and women workers*. Durhan, NC: Duke University Press.

Murphy M. (2011). Distributed reproduction. In M.J.Casper & P.Currah (Eds.), *Corpus: An interdisciplinary reader on bodies and knowledge*. New York, NY: Palgrave Macmillan.

Murphy, M. (2013). Distributed reproduction, chemical violence, and latency. *The Scholar and Feminist, 11*(3). Retrieved from http://sfonline.barnard.edu/life-un-ltd-feminism-bioscience-race/distributed-reproduction-chemical-violence-and-latency/2/

Murphy, M. (2017). *The economization of life*. Durham, NC: Duke University Press.

Murphy, M., Hendrich, S., Landgren, C., & Bryant, C. M. (2006). Food mycotoxins: An update. *Journal of Food Science*, *71,* R51-65. doi:10.1111/j.1750-3841.2006.00052.x

Muscogiuri, G., Barrea, L., Laudisio, D., Savastano, S., & Colao, A. (2017). Obesogenic endocrine disruptors and obesity: Myths and truths. *Archives of Toxicology*, *91*(11), 3469-3475. doi:10.1007/s00204-017-2071-1

Myers, N. (2008). Molecular embodiments and the body-work of modeling in protein crystallography. *Social Studies of Science, 38*(2), 163-199. doi:10.1177/0306312707082969

Nader, L. (1972). Up the anthropologist: Perspectives gained from studying up. In D.H. Hymes (Ed.), *Reinventing anthropology* (pp. 284-311). New York, NY: Pantheon Books.

Nash, L. (2006). *Inescapable ecologies: A history of environment, disease, and knowledge*. Berkeley, CA: University of California Press.

Nathanielsz, P. (1999). *Life in the womb: The origins of health and disease*. Ithaca, NY: Promethean Press.

National Academies of Sciences, Engineering, and Medicine (2005). The interplay between environmental chemical exposures and obesity. Proceedings of a workshop. *Proceedings of the Roundtable on Environmental Health Sciences, Research, and Medicine*, Washington, DC.

National Center for Health Statistics (US) (2016). *Health, United States, 2015: With special feature on racial and ethnic health disparities*. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/27308685

National Institute of Environmental Health Sciences (2017). *Bisphenol A (BPA)*. Retrieved from https://www.niehs.nih.gov/health/topics/agents/sya-bpa/index.cfm

National Institutes of Health (n.d.). *The human energy crisis*. Retrieved from https://irp.nih.gov/our-research/research-in-action/the-human-energy-crisis

National Institutes of Health (2012). *Biomedical Research Workforce Working Group Report*. Retrieved from https://acd.od.nih.gov/documents/reports/Biomedical_research_wgreport.pdf

National Institutes of Health (2014). *Funding facts*. Retrieved from https://report.nih.gov/fundingfacts/fundingfacts.aspx

National Institutes of Health (2015). *Grants: Applications, awards, success rates, and total funding by institute/center, mechanism, activity code, and funding source*. Retrieved from https://report.nih.gov/success_rates/index.aspx

National Institutes of Health (2016). *Stem cell information: Stem cell basics II* . Retrieved from https://stemcells.nih.gov/info/basics/2.htm

National Institutes of Health (2017a). *Estimates of funding for various research, condition, and disease categories (RCDC)*. Retrieved from http://report.nih.gov/categorical_spending.aspx

National Institutes of Health (2017b). *DNMT3A gene*. Retrieved from https://ghr.nlm.nih.gov/gene/DNMT3A

National Institutes of Health (2017c). *Report on NIH Funding vs. Global Burden of Disease*. Retrieved from https://report.nih.gov/info_disease_burden.aspx

National Institutes of Health (2017d). *Research project success rates by type and activity for 2017*. Retrieved from https://report.nih.gov/success_rates/Success_ByActivity.cfm

National Research Council (2005). *Bridges to independence: Fostering the independence of new investigators in biomedical research*. Washington, DC: National Academies Press.

Nathanielsz, P. (1999). *Life in the womb: The origins of health and disease*. Ithaca, NY: Promethean Press.

Nelson, N. (2013). Modeling mouse, human, and discipline: Epistemic scaffolds in animal behavior genetics. *Social Studies of Science, 43*(1), 3-29.

Ogawa, Y., Masuzaki, H., Isse, N., Okazaki, T., Mori, K., Shigemoto, M., . . . Yoshimasa, Y. (1995). Molecular cloning of rat obese cDNA and augmented gene expression in genetically obese Zucker fatty (fa/fa) rats. *Journal of Clinical Investigation, 96*(3), 1647-1652.

Ogden C. L., Carroll. M. D., & Curtin L. R. (2006). Prevalence of overweight and obesity in the United States, 1999-2004. *JAMA,295* (13), 1549-1555. doi:10.1001/jama.295.13.1549

Ogden C. L., Carroll, M., Kit, B. K., & Flegal, K. M. (2012). Prevalence of obesity and trends in body mass index among US children and adolescents, 1999-2010. *JAMA, 307*, 483-490. doi:10.1001/jama.2012.40

Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2014). Prevalence of childhood and adult obesity in the United States, 2011–2012. *JAMA, 311*(8), 806-814. doi:10.1001/jama.2014.732

Ogden, C.L., Carroll, M.D., Fryar, C.D., & Flegal, K.M. (2015). Prevalence of obesity among adults and youth: United States, 2011–2014. *NCHS Data Brief*, 219. Hyattsville, MD: National Center for Health Statistics. Retrieved from https://www.cdc.gov/nchs/data/databriefs/db219.pdf

Ogden, C. L., Carroll, M. D., Lawman, H. G., Fryar, C.D., Kruzson-Moran, D., Kit, B.K., & Flegal, K.M. (2016). Trends in obesity prevalence among children and adolescents in the united states, 1988-1994 through 2013-2014. *JAMA, 315*(21), 2292-2299. doi:10.1001/jama.2016.6361

Oksala, J. (2016). Affective labor and feminist politics. *Signs, 41*(2), 281-303.

Olson, V. A. (2010). The ecobiopolitics of space biomedicine. *Medical Anthropology, 29*(2), 170-193. doi:10.1080/01459741003715409

Panosfsky, A. (2009). Behavior genetics and the prospect of "personalized social policy." *Policy and Society*, 28(4), 327-340.

Park, A., Kim, W. K., & Bae, K.H. (2014). Distinction of white, beige and brown adipocytes derived from mesenchymal stem cells. *World Journal of Stem Cells, 6*(1), 33-42. doi:10.4252/wjsc.v6.i1.33

Paul, A. M. (2011). *Origins: How the nine months before birth shape the rest of our lives.* New York, NY: Free Press.

Perera, F., & Herbstman, J. (2011). Prenatal environmental exposures, epigenetics, and disease. *Reproductive Toxicology, 31*(3), 363-373. doi:10.1016/j.reprotox.2010.12.055

Powledge, T. M. (2011). Behavioral epigenetics: How nurture shapes nature. *BioScience, 61*(8), 588-592. doi:10.1525/bio.2011.61.8.4

Puig de la Bellacasa, M. (2012). Nothing comes without its world: Thinking with care. *The Sociological Review, 60*(2), 197-216.

Puig de la Bellacasa, M. (2017). *Matters of care: Speculative ethics in more than human worlds*. Minneapolis, MN: University of Minnesota Press.

Rabadán-Diehl, C., & Nathanielsz, P. (2013). From mice to men: Research models of developmental programming. *Journal of Developmental Origins of Health and Disease, 4*(1), 3-9. doi:10.1017/S2040174412000487

Rabinow, P., & Rose, N. (2006). Biopower today. *BioSocieties, 1*(2), 195-217. doi:10.1017/s1745855206040014

Ragin, C.C., & Becker, H. S. (1992). *What is a case?: Exploring the Foundations Social Inquiry*. Cambridge, UK: Cambridge University Press.

Ramaraju, G. A., Teppala, S., Prathigudupu, K., Kalagara, M., Thota, S., Kota, M., & Cheemakurthi, R. (2017). Association between obesity and sperm quality. *Andrologia, 50*(3), e12888. doi:doi:10.1111/and.12888

Rapp, R. (1999). *Testing women, testing the fetus. The social impact of Amniocentesis in America*. New York, NY: Routledge.

Ravelli, G. P., Stein, Z. A., & Susser, M. W. (1976). Obesity in young men after famine exposure in utero and early infancy. *New England Journal of Medicine, 295*(7), 349-353. doi:10.1056/NEJM197608122950701

Rheinberger, H-J. (1993). Experiment and orientation: Early systems of in vitro protein synthesis. *Journal of History of Biology, 26*(3), 443-471.

Rheinberger, H-J. (1997). *Toward a history of epistemic things. Synthesizing proteins in the test tube*. Redwood City, CA: Stanford University Press.

Rheinberger, H-J. (2012). Experimental systems: Difference, graphematicity, conjuncture. In F. Dombois, U. M. Bauer, C. Mareis, & M. Schwab (Eds.), *Intellectual birdhouse: Artistic practice as research* (pp. 89-99). Köln, Germany: König.

Ribaroff, G. A., Wastnedge, E., Drake, A. J., Sharpe, R. M., & Chambers, T. J. G. (2017). Animal models of maternal high fat diet exposure and effects on metabolism in offspring: A meta-regression analysis. *Obesity Reviews, 18*(6), 673-686. doi:10.1111/obr.12524

Richardson, S.S. (2015). Maternal bodies in the postgenomic order: Gender and the explanatory landscape of epigenetics. In S.S. Richardson & H. Stevens (Eds.), *Postgenomics: Perspectives on biology after the genome* (pp. 210-231). Durham, NC: Duke University Press.

Richardson, S. S. (2017). Plasticity and programming: Feminism and the epigenetic imaginary. *Signs: Journal of Women in Culture and Society, 43*(1), 29-52.

Richardson, S. S. (2018). *The maternal imprint*. Manuscript in preparation.

Richter, C. A., Birnbaum, L. S., Farabollini, F., Newbold, R. R., Rubin, B. S., Talsness, C. E., . . . vom Saal, F. S. (2007). In vivo effects of bisphenol A in laboratory rodent studies. *Reproductive Toxicology, 24*(2), 199-224. doi:10.1016/j.reprotox.2007.06.004

Riska, E. (2010). Gender and medicalization and biomedicalization theories. In A.E. Clarke, L. Mamo, J.R. Fosket, J.R. Fishman, & J.K. Shim (Eds.), *Biomedicalization: Technoscience, Health, and Illness in the U.S.* (pp. 147- 170). Durham, NC: Duke University Press,

Robert Wood Johnson Foundation (2016). *Declining childhood obesity rates: Where are we seeing signs of progress?* Retrieved from https://www.rwjf.org/content/dam/farm/reports/issue_briefs/2016/rwjf401163

Rockey, S. (2015). *What are the chances of getting funded?* Retrieved from http://nexus.od.nih.gov/all/2015/06/29/what-are-the-chances-of-getting-funded/

Rose, H. (1983). Hand, brain, and heart: A feminist epistemology for the natural sciences. *Signs, 9*(1), 73-90.

Rose, N. (2001). The politics of life itself. *Theory, Culture & Society, 18*(6), 1-30. doi:10.1177/02632760122052020

Rose, N. (2006). *The politics of life itself: Biomedicine, power, and subjectivity in the twenty-first century*. Princeton, NY: Princeton University Press.

Rosen, E. D., & MacDougald, O. A. (2006). Adipocyte differentiation from the inside out. *Nature Reviews Molecular Cell Biology, 7*, 885. doi:10.1038/nrm2066

Ross, D. A., Rao, P. K., & Kadesch, T. (2004). Dual roles for the notch target gene hes-1 in the differentiation of 3T3-L1 preadipocytes. *Molecular and Cellular Biology, 24*(8), 3505-3513. doi:10.1128/MCB.24.8.3505-3513.2004

Rothrock, T. (2017). *Sprague Dawley®: The workhorse of reproductive endocrinology research - part 2*. Retrieved from https://blog.envigo.com/sprague-dawley-the-workhorse-of-reproductive-endocrinology-research-part-2

Russell, N.C., Tansey, E.M., & Lear, P.V. (2000). Missing links in the history and practice of science: Teams, technicians, and technical work. *History of Science*, 38, 237-241.

Sacks, F. M., Bray, G. A., Carey, V. J., Smith, S. R., Ryan, D. H., Anton , S. D., . . . Williamson, D. A. (2009). Comparison of weight-loss diets with different compositions of fat, protein, and carbohydrates. *New England Journal of Medicine, 360*(9), 859-873. doi:10.1056/NEJMoa0804748

Saely, C. H., Geiger, K., & Drexel, H. (2012). Brown versus white adipose tissue: A mini-review. *Gerontology, 58*(1), 15-23.

Saguy, A. C. (2013). *What's wrong with fat?* Oxford, UK: Oxford University Press.

Schipper, L., Harvey, L., van der Beek, E. M., & van Dijk, G. (2018). Home alone: A systematic review and meta-analysis on the effects of individual housing on body weight, food intake and visceral fat mass in rodents. *Obesity Reviews*. Advanced online publication. doi:10.1111/obr.12663

Shanle, E. K., & Xu, W. (2011). Endocrine disrupting chemicals targeting estrogen receptor signaling: Identification and mechanisms of action. *Chemical Research in Toxicology, 24*(1), 6-19. doi:10.1021/tx100231n

Shapin, S. (1989). The invisible technician. *American Scientist, 77*, 554-563.

Shapin, S. (2008). *The scientific life*. Chicago, IL: University of Chicago Press.

Shapin, S., & Schaffer, S. (1985). *Leviathan and the air-pump: Hobbes, Boyle, and the experimental life*. Princeton, NJ: Princeton University Press.

Shostak, A., Husse, J., & Oster, H. (2013). Circadian regulation of adipose function. *Adipocyte*, 2(4), 201-2016. doi:10.4161/adip.26007

Shostak, S. (2013). *Exposed science: Genes, the environment, and the politics of population health*. Berkeley, CA: University of California Press.

Siroux, V., Agier, L., & Slama, R. (2016). The exposome concept: A challenge and a potential driver for environmental health research. *European Respiratory Review, 25*(140), 124-129. doi:10.1183/16000617.0034-2016

Shapin, S. (2010) *Never pure: Historical studies of science as if it was produced by people with bodies, situated in time, space, culture, and society, and struggling for credibility and authority*. Baltimore, MD: The Johns Hopkins University Press.

Speakman, J. R. (2006). Thrifty genes for obesity and the metabolic syndrome — time to call off the search? *Diabetes and Vascular Disease Research, 3*(1), 7-11. doi:10.3132/dvdr.2006.010

Standing, G. (2011). *The precariat: The new dangerous class*. London, UK: Bloomsbury Academic.

Standing, G. (2014). *A precariat charter: From denizens to citizens*. London, UK: Bloomsbury Academic.

Starr, P. (1982). *The social transformation of American medicine*. New York, NY: Basic Books.

Steensma, D. P., Kyle, R. A., & Shampo, M. A. (2010). Abbie Lathrop, the "mouse woman of Granby": Rodent fancier and accidental genetics pioneer. *Mayo Clinic Proceedings, 85*(11), e83-e83. doi:10.4065/mcp.2010.0647

Stein, D. (2007). Hard money/soft money: The two cultures. *The Academic Exchange, 9*(6). Retrieved from http://www.emory.edu/ACAD_EXCHANGE/2007/may/steinessay.html

Stephan, P. (2012). *How economics shapes science*. Cambridge, MA: Harvard University Press.

Straus, A. (1987). *Qualitative analysis for social scientists*. New York, NY: Cambridge University Press

Strauss, A., & Corbin, J. (1994). Grounded theory methodolog: An overview. In K.D. Norman & S.L.Y. Vannaeds (Eds.), *Handbook of qualitative research* (pp. 22-23). Thousand Oaks, CA: Sage Publications.

Szyf, M., Weaver, I. C. G., Champagne, F. A., Diorio, J., & Meaney, M. J. (2005). Maternal programming of steroid receptor expression and phenotype through DNA methylation in the rat. *Frontiers in Neuroendocrinology, 26*(3), 139-162. doi:10.1016/j.yfrne.2005.10.002

Taconic (n.d.). *Sprague Dawley® rat: Preferred for safety and efficacy, surgical modifications and reproductive studies*. Retrieved from https://www.taconic.com/pdfs/sprague-dawley-rat.pdf

Tansey, E. M. (2008). Keeping the culture alive: The laboratory technician in mid-twentieth-century British medical research. *Notes and records of the Royal Society of London, 62*(1), 77-95. doi:10.1098/rsnr.2007.0035

Taubes, G. (2002). *What if it's all been a big fat lie?* Retrieved from http://www.nytimes.com/2002/07/07/magazine/what-if-it-s-all-been-a-big-fat-lie.html

Tavory, I., & Timmermans, S. (2009). Two cases of ethnography: Grounded theory and the extended case method. *Ethnography, 10*(3), 243-263. doi:10.1177/1466138109339042

The Guardian (2013, September 11). *David Barker obituary*. Retrieved from https://www.theguardian.com/society/2013/sep/11/david-barker

Thigpen, J. E., Setchell, K. D., Kissling, G. E., Locklear, J., Caviness, G. F., Whiteside, T., . . . Grant, M. (2013). The estrogenic content of rodent diets, bedding, cages, and water bottles and its effect on bisphenol A studies. *Journal of the American Association for Laboratory Animal Science, 52*(2), 130-141.

Traweek, S. (1988). *Beamtimes and lifetimes*. Cambridge, MA: Harvard University Press.

Trust for America's Health & Robert Wood Johnson Foundation (n.d.). *Obesity rates and trends overview*. Retrieved from https://stateofobesity.org/obesity-rates-trends-overview/

Turner, B. S. (1982). The government of the body: Medical regimens and the rationalization of diet. *The British Journal of Sociology, 33*(2), 254-268.

Vickers, M. H., Breier, B. H., Cutfield, W. S., Hofman, P. L., & Gluckman, P. D. (2000). Fetal origins of hyperphagia, obesity, and hypertension and postnatal amplification by hypercaloric nutrition. *American Journal of Physiology - Endocrinology And Metabolism, 279*(1), E83-E87.

vom Saal, F.S. & Welshons, W.V. (2014). Evidence that bisphenol A (BPA) can be accurately measured without contamination in human serum and urine, and that BPA causes numerous hazards from multiple routes of exposure. *Molecular and Cellular Endocrinology, 398*(1-2), 101-113. doi: 10.1016/j.mce.2014.09.028

Waddington, C.H. (1942). Canalization of development and the inherence of acquired characters. *Nature, 150*, 563-565.

Waddington, C. H. (1956). Embryology, epigenetics and biogenetics. *Nature, 177*, 1241. doi:10.1038/1771241a0

Waddington, C. H. (2012). The epigenotype. 1942. *International Journal of Epidemiology*, *41*(1), 10-13. doi: 10.1093/ije/dyr184

Waggoner, M. (2015). Cultivating the maternal future: Public health and the pre-pregnant self. *Signs: Journal of Women in Culture and Society, 40*(4), 939-962.

Waldby, C. (2000) *The visible human project: Informatic bodies and posthuman medicine*, London, UK: Routledge.

Waldby, C. (2002). Stem cells, tissue cultures and the production of biovalue. *Health, 6*(3), 305-323. doi:10.1177/136345930200600304

Waldby, C., & Cooper, M. (2008). The biopolitics of reproduction. *Australian Feminist Studies, 23*(55), 57-73. doi:10.1080/08164640701816223

Wells, J. C. K. (2010). Maternal capital and the metabolic ghetto: An evolutionary perspective on the transgenerational basis of health inequalities. *American Journal of Human Biology, 22*(1), 1-17. doi:10.1002/ajhb.20994

Whalley, P., & Barley, S.R. (1997). Technical work in the division of labor: Stalking the wily anomaly. In S.R. Barley & J.E. Orr (Eds.), *Between craft and science: Technical work in US setting* (23-52). Ithaca, NY: Cornell University Press.

Wild, C. P. (2005). Complementing the genome with an "exposome": The outstanding challenge of environmental exposure measurement in molecular epidemiology. *Cancer Epidemiology Biomarkers and Prevention, 14*(8), 1847-1850. doi:10.1158/1055-9965.epi-05-0456

Wild, C. P. (2012). The exposome: From concept to utility. *International Journal of Epidemiology, 41*(1), 24-32. doi:10.1093/ije/dyr236

Wittgenstein, L. (1953). *Philosophical investigations* (G.E.M. Anscombe, Trans.). Oxford, UK: Blackwell.

Wylie, C.D. (2017). Trust in technicians in paleontology laboratories. *Science, Technology, & Human Values, 43*(2), 324-348. doi:10.1177/0162243917722844

Woodruff, T., Zota, A., & Schwartz, J. (2011). Environmental chemicals in pregnant women in the US: NHANES 2003–2004. *Environmental Health Perspectives, 119*(6), 878-885. doi: 10.1289/ehp.1002727

Wu, C.T., & Morris, J.R. (2001). Genes, genetics, and epigenetics: A correspondence. *Science, 293*(5532), 1103–1105.

Xiong, Y., Walker, K., Min, X., Hale, C., Tran, T., Komorowski, R., . . . Véniant, M. M. (2017). Long-acting MIC-1/GDF15 molecules to treat obesity: Evidence from mice to monkeys. *Science Translational Medicine, 9*(412), eaan8732. doi:10.1126/scitranslmed.aan8732

Xue, Y. X., & Larson, R.C.. (2015). STEM crisis or STEM surplus? Yes and yes. *Monthly Labor Review*. Retrieved from https://doi.org/10.21916/mlr.2015.14.

Yavory, I., & Timmermans, S. (2009). Two cases of ethnography: Grounded theory and the extended case method. *Ethnography, 10*(3), 243-263. doi:10.1177/1466138109339042

Yu, J. H., & Kim, M.-S. (2012). Molecular mechanisms of appetite regulation. *Diabetes & Metabolism Journal, 36*(6), 391-398. doi:10.4093/dmj.2012.36.6.391

Zelizer, V. A. (2002). Review essay [Review of The price of motherhood: Why the most important job in the world is still the least valued by Ann Crittenden; The invisible heart: Economics and family values by Nancy Folbre; Unbending gender: Why family and work conflict and what to do about it by Joan Williams]. *Contemporary Sociology, 31*(2), 115-119. doi:10.2307/3089463

Zenzen, M., & Restivo, S. (1982). The mysterious morphology of immiscible liquids: A study of scientific practice. *Information (International Social Science Council), 21*(3), 447-473. doi:10.1177/053901882021003004

Zieff, G., & Stoner, L. (2018). Re: The environmental impact of obesity: Longitudinal evidence from the United States. *Public Health, 152*, 182. doi:10.1016/j.puhe.2017.08.018

Zola, I. (1972). Medicine as an institution of social control. *Sociological Review, 20*, 487-504.

Zola, I. (1991). Bringing our bodies and ourselves back in: Reflections on a past, present, and future 'medical sociology'. *Journal of Health and Social Behavior, 32*(1), 1-16.

Zoeller, R. T., & Heindel, J. J. (2017). Do environmental chemicals make us fat? *Endocrinology, 158*(10), 3086-3087. doi:10.1210/en.2017-00715

Zucker, L. M., & Zucker, T. F. (1961). Fatty, a new mutation in the rat. *Journal of Heredity, 52*(6), 275-278.