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Philosophy, Phenomenology, and Neuroscience: The Groundwork for an Interdisciplinary Approach to a Comprehensive Understanding of Addiction

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DEDICATION

To my Mom and Dad, condiciones sine quibus non

ABSTRACT OF THE DISSERTATION

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by

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Biologists, psychologist, philosophers, sociologists, and even economists have offered explanations of addiction. These analyses range from the view that it is the result of a disorder of the reward and inhibitory centers of the brain to the contention that it is the product of a rational choice model. One philosophical theory holds that addiction is something which holds life affirming meaning for the addict.

My dissertation uses some of the (more promising) existing theories to formulate what I call the "hybrid" theory. Many theorists do not seem to attempt, and may not recognize the need for, an interdisciplinary approach to addiction. In my dissertation I begin –it seems unlikely that my theory could be considered complete- to construct such an approach.

My theory integrates neurobiological and phenomenological explanations. The neurobiological element of the theory centers on the claim that addiction can be understood as a *disordered appetite*. I offer evidence from the biological literature that

addiction and appetite have a great deal in common. I argue that the literature implies that addiction is a disordered variant of an acquired appetite. I also explain, in conceptual terms, what constitutes a disordering of appetite.

The second element of the hybrid theory takes account of the phenomenology of addiction. Specifically I claim that addiction can also be a *disordered passion*. Biological models explain a great deal, but they fail to capture the experience of the addict which accumulates over the years or decades of addiction. It is necessary to understand the emotional connection with an addictive good –from the addict's point of view- in order to gain a fuller understanding of addiction. I believe the phenomenology of addiction is especially important to explaining the problem of relapse.

The hybrid theory is not merely the juxtaposition of two different views of addiction. I believe addiction is best understood as being both things at once. I claim that either is sufficient for addiction, but each can explain aspects of addiction that the other cannot account for fully. My theory, if correct, shows that understanding addiction requires a balanced and integrated approach from multiple perspectives.

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Introduction

This dissertation has one major task. It is to develop the groundwork for a comprehensive theory of addiction, both at the conceptual and individual levels, that draws upon existing theories and evidence from neurobiology, psychology, philosophy, and phenomenology. Secondary to that, I wish consider some of the implications of the foundation of such a theory in the explaining the constituents, phenomena, and puzzles associated with addiction. This secondary goal is an effective means for determining whether the groundwork I hope to lay down will act as a solid foundation for a comprehensive theory of addiction. In order to accomplish this secondary task I present seven questions about addiction which I answer, at least in part, using my theory of addiction.

Prima facie one might think that it would be an easy task to understand addiction as a readily identifiable pattern of behavior associated with some drug or activity. However, I believe the most identifiable elements of addiction are only the geographic surface phenomena of a deeper and more difficult to understand structure. In reality, addiction is a puzzling, heterogeneous phenomenon that defies easy analysis. In its most familiar manifestation it involves excessive and detrimental ingestion, inhalation, or injection of substances of intoxication, such as alcohol, cocaine, or heroin. However, it also includes use of a substance that generally does not produce pronounced intoxication, nicotine. The array of things to which one may become addicted seems vast, encompassing processes or behaviors (both should be read as designating addictions which do not involve drug use), such as gambling, eating, and perhaps even shopping, in

addition to substances. In some cases, addiction lays the life of the addict to waste in only a few years (for social and physical reasons), while in other cases it seems more akin to a bad habit.[1]

In addition to this diversity there are other puzzling aspects of addiction. It can lay dormant for years and then, with only a fleeting interaction, pull the addict back into a cycle of addictive behavior.[2] Also puzzling is that addicts often seem to be divided against themselves, hating their addiction and making repeated attempts to quit. It is unclear whether they are unable or unwilling to be successful. Many addicts cling to their addictions in spite of repeated and recognized adverse consequences. Yet the addictive drive makes use of the same neurological pathways and reward feedback as -and bears a resemblance to- common non-addictive drives for food, drink, and sex. Understanding addiction requires an explanation of its diversity, its persistence, and its distinction from related phenomena.

I do not begin by dividing addiction into sub-categories, such as chemical and process addictions. That sort of approach would impede the goal of a unitary understanding. It does so in two ways: First, by requiring focus on the variation among

[1] Here I am thinking of the marketed diversity in apparent physical impact and strength of certain chemical addictions, such as heroin and nicotine. Although historically the rates of quitting are similar for the two I believe this says something about the strength of heroin vis-à-vis nicotine. The physical, financial, familial, legal, and personal inducements to quit using heroin seem stronger and yet are no more successful in deterring chronic use. However, cigarette use seems to be generally inhibited by something as minor as price fluctuations of a few dollars (see, Beck, Grossman and Murphy, 1992).

[2] E.M Jellinek,1960, relates an interesting story of an alcoholic who, after years of sobriety innocently had a drink with his lunch one day, only to find himself soon repeating the pattern of abuse he had engaged in years before (clinically this would be called a priming dose). The ease with which addictive behavior reoccurs after only a small or token use of a substance is also expressed by a saying common in Alcoholics Anonymous, “One drink is too many, 10 is never enough”.

addictions before first considering the unifying elements; second, by scattering, instead of focusing, those unifying elements. There are many important distinctions among substances and behaviors of addiction: many, but not all, chemical addictions can result in serious or even life threatening physical withdrawal, while behavioral addiction withdrawal is physically manifested to a limited degree and only in some cases.[3] However, accounting for such diversity will come later in the dissertation when I present my own theory. I start by introducing already developed general theories of addiction, and in examining them I hope to find a springboard from which to launch my own.

My hope is that a general theory of addiction, drawn from the theories and evidence of multiple disciplines, will provide an explanation of many of the core phenomena associated with addiction, such as relapse, loss of control, and inner conflict.[4] I will be successful if I am able to explain the core phenomena I select while leaving my theory open and able to absorb additions, consistent with the theory, that explain phenomena that I will not be able to address in this dissertation.[5]

Before introducing any general theories or specific questions, I begin with a

[3] See Elster "Gambling and Addiction" for some discussion of the types of withdrawal generally associated with process addiction . Elster notes that physical symptoms are often minor in most process addictions, if they occur at all. I take it to be general knowledge that there are serious physical side effects associated with withdrawal from many substances of dependence such as heroin, alcohol, methamphetamines. For a discussion of some of the dangers of withdrawal from alcohol see Trevisan, et al., 1998.

[4] This naturally leads one to wonder: How am I choosing the phenomena I will try to explain? Although virtually any choice of phenomena one might choose to explain (short of them all) would be arbitrary, I use the 7 criteria for the diagnosis of substance dependence in the DSM-IV to suggest the phenomena I target.

[5] Even if I were knowledgeable about all existing biological and psychological theories of addiction (which I am not) I do not think I could work them all into a definitive explanation of addiction. The amount of information is overwhelming for one dissertation. In addition, neurobiologists are continuing to find out new things about neural pathways in the brain and how they operate in addicts. In such circumstances it is a virtue of a theory that it explains what it can and leaves itself open to additions and perhaps some revisions.

general definition of addiction. In broad, clinical terms chemical dependence is defined by the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV) in this way:

> …a syndrome characterized by a maladaptive pattern of substance use, leading to clinically significant impairment or distress….[6]

Norman Miller, professor of psychiatry and medicine at Michigan State University, defines it this way:

> Addiction is defined by behaviors…which are the preoccupation with the acquisition of alcohol (drugs), compulsive use of alcohol (use in spite of adverse consequences), and a pattern of relapse to alcohol and drugs…Pervasive to the three criteria for addiction is a loss of control underlying the preoccupation…[7]

Jerzy Vetulani, Pharmacologist at the Polish Academy of Sciences, is more succinct:

> Dependence on a drug (and also on other rewarding stimuli) can be defined as a condition in which such changes in psyche have occurred that seeking a drug or other rewarding stimulus becomes the main focus of the addict's life.[8]

Finally, Ada Kahn and Jan Fawcett, editors of *The Encyclopedia of Mental Health*, define addiction in these terms:

> Psychological dependence can involve a loss of control of the substance use [or activity] and a tendency to orient behavior or life priorities toward obtaining the drug or pursuing the behavior.[9]

These definitions are generally representative of most I have read. My own

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[6] Schultz and Graham (eds.), 1998, p. 1287. The DSM-IV then goes on to list 7 ways this distress or impairment might be manifested, three or more of which must be true of the patient in the previous 12 months for a diagnosis of chemical dependence. In exceptionally abridged format the 7 criteria for determining chemical dependence are: tolerance; withdrawal; unintended degree of use; persistent desire; large amounts of time devoted to use, procurement, or recovery; decrease of previous involvement in social, occupational, or recreational activities; continued use despite recognized problems.

[7] Miller, 1991, p.298

[8] Vetulani, 2001, p.310

[9] Kahn and Fawcett, 1993, p.10

working definition of addiction draws from each of these definitions. However, I wish to avoid making adverse social and familial consequences an essential element of addiction. Doing so would make my working definition artificially narrow since what constitutes adverse social circumstances is somewhat variable across cultures. Even if there were invariance in this regard it seems to be the case that social and familial consequences are separable from the concept. I make this exclusion so that I do not preclude the possibility, for example, of alcoholism on a deserted island, or nicotine addiction in a society of nicotine addicts.[10] I also wish to avoid assuming that addiction entails compulsion or that it is a disease (the term disorder is used clinically and I will adopt that term). Drawing from the previous definitions I take any addiction to require the following: 1. some sort of acquired, deeply felt need or want (physical and/or mental) to use a drug or engage in a behavior; 2. difficulty in resisting or controlling -when one attempts to exert control- the extent and degree of drug use or behavioral engagement; 3. drug use or behavior which becomes a central focus around which other life activities revolve (particularly when other activities revolve around a particular drug or behavior in an ever increasing way); 4. the salience (i.e. the direction of attention) of the felt need or want for the behavior or drug of addiction continues even after a period of inactivity (with regard to use of the drug/engagement in the behavior).

The definition I offer is intended as a neutral starting point, consistent with addiction as a disease or a moral failing, a pattern of willful self-destructive choices or an

[10] Associating guidelines for defining or understanding addiction too closely with particular mores is a problem. It seems worth noting that in order to avoid this very problem the W.H.O uses the International Classification of Diseases -10 as its diagnostic criteria for addiction, as opposed to the DSM-IV.

irresistible compulsion. There is some question about whether addiction is a disease and a great deal of controversy about whether it entails compulsion.[11] My proposed theory will address the former question directly. As to whether addiction entails compulsion I will not be in a position to claim one way or another. The nuances of how best to understand compulsion are beyond the scope of what I examine, and without first having this understanding it is impossible to discover the relationship of addiction to compulsion. However, it would certainly be my hope that someone working with the notion of compulsion could use what I will argue to make some determination about addiction in that regard.

Chapter 1 is a survey of some helpful theories of addiction from the fields of economics, psychology, neurobiology, and philosophy. The ideas they contain serve a dual purpose: some help form the basis of my own theory and others serve as a helpful guide to what makes a theory of addiction fail. Chapter 1 further serves as an illustration of the need for an interdisciplinary approach to addiction. In chapter 2 I introduce and explain the concept of disordered appetite which is the first core constitutive element of my own interdisciplinary theory of addiction, which I call the "hybrid theory".[12] A great

[11] For an interesting, albeit minority, point of view with respect to the former controversy see Fingerette, 1988. The philosophical and psychological literature are replete with discussions of how exactly to categorize the strong impulses associated with addiction, for discussion of this see Watson 1999band Yaffe , 2001.

[12] The hybrid theory will be composed of two different branches, the hybrid concept and the hybrid instantiation branches. The hybrid theory is meant to answer conceptual questions about addiction as well as questions about the kinds of addiction that may be found in individual addicts. I would be jumping the gun if I were to say more so I will leave the details of the different branches for chapter 4.

deal of the inspiration for this element of the hybrid theory comes from Gary Watson.[13] However, Watson leaves much unexamined about precisely how to understand what makes an appetite disordered and I devote my focus to detailing what constitutes this disordering. In chapter 3 I introduce and explain the second constitutive element of my hybrid theory of addiction: disordered passion. In chapter 4 I present the hybrid theory in detail and show how these two distinct but related disorders underlie the concept of addiction, and how they act to explain some of the troubling phenomena associated with addiction. In this chapter I will also briefly consider some of the implications of my theory in both the treatment of addicts and their moral responsibility.

Anyone proposing a theory of addiction should make it clear to which questions his theory is addressing itself. The breadth and difficulty of those questions, as well as the consistency and specificity of the answers, provide the yard stick for measuring the strength of a theory. The questions I explore will surface throughout the dissertation. For now, I offer a list roughly in order of how they will be addressed:

1. What distinguishes addictive (disordered) appetites from non-addictive ones?
2. What mechanisms perpetuate the wanting and decision making that sustains addiction, often in the face of negative consequences, and how do they account for a disordered appetite?
3. How can one explain the diversity of substances and behaviors of addiction and the phenomena they produce? What unifies the different instantiations of addiction?

[13] Specifically his article "Disordered Appetites: Addiction, Compulsion, and Dependence," 1999a.

4. How can addicted agents be cross-temporally or simultaneously divided against themselves with respect to their addiction?[14]

5. What accounts for relapse and how does the concept of relapse inform our understanding of remission (or the boundaries) of addiction?

6. In what sense (if any) is addiction a disease?

7. How can one hope to overcome addiction?

I do not think I can provide exhaustive answers to all of these questions. Questions 2, 3, and 4 are largely the purview of the sciences and much empirical work remains to be done in providing a full answer. A good theory of addiction must consider the work done by other disciplines and incorporate that work as best it can. My own contribution will be most focused on the conceptual questions, and it is within my power to come closest to providing complete answers for questions 1, 5, 6 and 7. In answering these questions I wish to directly suggest a surer purchase from which to make an assessment of remission in addicts. Fully understanding and explaining addiction is a mammoth task, and much work will still need to be done when I have concluded, but I hope my reader will find the amount reduced by a worthwhile sum.

[14] In chapter 1 it will become apparent that explaining how and why addicts change their minds about using is important in explaining addiction. That a theory cannot do this is a fatal flaw, as I will show with Becker and Murphy's theory. However, I do not wish to imply that there are no uncomplicated willing addicts. There certainly could be. It is just that this seems one of the questions and account of addiction must address.

Chapter 1

Different Approaches to Understanding Addiction

There will be much more to do in creating a comprehensive theory of addiction even at the completion of this dissertation. It is because of the vastness and depth of the research on addiction that I cannot entertain any hope of constructing a comprehensive theory. But it is reasonable to believe that in addition to providing some answers to the questions brought out in the introduction a promising foundation for a comprehensive theory can be laid down. The goal of such a theory must be to create an intellectual space within which different disciplines may begin to approach one another, and to show how this might be done. By creating such a space the hope is that social theorists, economists, psychologists, neurobiologists, philosophers, and anyone else with an interest in addiction will see how particular findings in one discipline might be integrated into a larger view. The realization of a common and interdisciplinary goal of confirming, correcting, or even refuting a unified theory of addiction can only come when the ideas or evidence from one discipline are more easily embraced by other disciplines.

In constructing a unified theory one must begin with the various approaches to addiction taken by thinkers within the disciplines one wants to bring together. Thus, in this chapter I examine the ideas and theories of economists, psychologists, neuroscientists, and philosophers who hope to explain what constitutes addiction, or some element of it. Each author I have selected has some positive contribution to make to

the specific questions I attempt to answer and the interdisciplinary theory for which I want to lay the foundation. The psychological and philosophical theories I have chosen to examine are well known, with one exception, in part because I do not intend for my own theory to be especially radical and because there is usually something worth exploring in generally accepted ideas.

Integrating neurobiology into the discussion is more challenging. The literature is vast and technical, but my use of it is confined to recognized commonalities in the specific neural networks and parts of the brain that are affected by substances and behaviors of addiction. This is because at the greatest and most technical level of biological detail the neurobiological effects of one substance of abuse invariably differ from another.[15] However, there seems to be a great unifying thread among substances and behaviors associated with addiction call this the "dopamine connection." My examination of this will take place at the beginning of chapter 2. With this general framework in mind I now move into an examination of particular theories of addiction.

[15] *Principles of Addiction Medicine* , 1998, gives a nice illustration of the similarities and differences in the cellular and neural network operation between various substances of abuse. That there are differences should not be surprising since common substances of abuse fall into different drug classifications, such as alcohol (a sedative/hypnotic) and cocaine (a stimulant). But even within the same classification different drugs can have slightly different neural affects. For instance cocaine blocks dopamine's (a neurotransmitter) reuptake into neurons after it has been released, whereas amphetamines not only block its reuptake but also stimulate increased production. The details of how each drug operates are highly technical and distinct (particularly at the cellular level). However, this will not matter much in making my broader conceptual point.

§1. Delay-Discounting Theories: Modeling Impulsivity and Decision Making in Addiction

Central to some theorists' explanations of addiction is an account of why an addict makes the choice to sacrifice greater non-addictive goods (greater, because they are sustainable and long term) as a result of their engagement in addictive behavior. Colloquially this can be thought of as the impulsivity and recklessness associated with addiction. There are various reasons for impulsivity studied by psychologists. Madden and Johnson point out that, "Failures of attention, inability to inhibit pre-potent responses, and the failure of delayed events to control current choices have all received considerable attention."[16] It is the third of these reasons for impulsivity that is at the heart of delay-discounting.

Delay-discounting in simplest terms is how one takes into account the uncertainty that future goods will obtain and uses that to guide present behavior. Take a simple example: suppose one has a choice between a 1.00 chance of receiving 25 dollars now or a .70 chance of receiving 100 dollars (and an .30 chance of receiving nothing) 1 year from now from a risky investment of that 25 dollars. The rational thing to do to maximize gain (assuming no special circumstances, such as needing the money now to avoid starvation) would be to invest the money. This is because on average the return for making such an investment would be 70 dollars (as opposed to just 25 for the average non-investor). But sufficiently high uncertainty about the future can also countenance preferring near term rewards. One can easily imagine such a case using a similar

[16] Madden and Bickel, 2010, p.11

scenario. Suppose that instead of a .70 chance of a 100 return the chance was only .20 (in which case the average return would be just 20 dollars, and the 25 dollars would be preferable).

But scenarios in the real world are not so straightforward. The degree to which someone should discount a particular good, the way that discounting should take place over delays of variable lengths, and even what value the future good should be assigned (when it is non-monetary) are difficult problems. And yet, "a number of studies have established that individuals addicted to a variety of drugs have a strong tendency to devalue delayed gains and losses."[17] Delay-discounting, in fact, seems to be a general disposition that when used poorly has links to behavioral problems and impulsivity. A famous study, done by Mischel, et al., showed greater degrees of discipline and truancy problems in adolescence among elementary aged students who showed difficulty in delaying gratification.[18]

In this section I consider two delay-discounting theories which purport to explain the kind of decision making and impulsivity shown by the addict. The first, an exponential discounting model, is the work of Gary Becker and Kevin Murphy.[19] The

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17 Ibid. p.12

18 Mischel, Shoda, and Rodriguez, 1989. The study to which I am referring is the well know 'marshmallow' study in which young children were given a marshmallow by an experimenter and told that if they went 15 minutes without eating it they would receive a second marshmallow. The experimenter would then leave the room and observe the children. In a follow up study is was found that children who were unable to wait the 15 minutes for the experimenter to return suffered higher rates of disciplinary problems and truancy as adolescents.

19 Gary Becker received the 1992 Nobel Prize in economics in part for applying economic theories to social behaviors. This is precisely what he does in the theory I examine in this section.

second, a hyperbolic discounting model, has George Ainslie as one of its foremost proponents. The importance of these two theories is that between them they consider two elements critical to understanding addiction: how an addict comes to value her addiction and how that value conflicts with other non-addictive goods.

Prima facie addicts do not seem to care about maximizing their long term good when they choose to engage in their addiction. Generally addiction is thought to endanger the means of subsistence, personal life, and health of the addict. But Becker and Murphy offer a model that accounts for the addict's tendency to give up greater (in terms of absolute value) distant goods in favor of lesser (again in terms of absolute value) near-term goods within the confines of rational choice.

Becker and Murphy argue that addictions "are rational in the sense that they involve forward looking maximization [of goods] with stable preferences."[20] They propose that rational agents globally apply a discount coefficient to future goods, and that this coefficient is increased exponentially given greater distance in the futurity of a good. Consider an example: I exponentially discount the future and I find out I must choose between trying to get a car (worth seven thousand dollars) in three years or five thousand dollars in one year. Suppose I assign a value of 7 to the car and 5 to the money. Further suppose I discount the future at a rate of .9 per year. At the present time my preference can be determined with the following equation: $P = v * d^a$ (where 'P' = how much I presently prefer some choice; 'v' = the absolute value assigned; 'd' = is the factor by which I discount a future good per unit of delay; and 'a' = the number of units of

[20] Becker and Murphy, 1988, p.675

delay).[21] In this scenario, a year away from being given five thousand dollars and three years away from a new car I prefer the new car. A year later I still prefer the new car even when I could have the money immediately if I would just give up my claim to the new car (still two years off). Graphically representing my preferences would show an increase of my preference for both the money and the car as each draws nearer but my preferring one relative to the other would not change.

According to Becker and Murphy the addict simply uses a lower (more extreme) discount coefficient for future goods than does a non-addict.[22] This means the addict tends to see addictive behavior as more likely to maximize his good because it (invariably) is temporally nearer than non-addictive goods. This is true even when non-addictive goods have greater absolute value, because they become less preferred (have less relative value) once the discount coefficient is applied. Complicating the picture is that the value function (the absolute value) of an addictive substance or process increases artificially because the addict *values* the addictive behavior due to the phenomena of

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[21] In a slightly different form this equation occurs in Yaffe's , 2001, explanation of exponential discounting. The preference I have for the car at the time of my initial decision would be $p = 7 * .9^3$, giving p a value of 5.103. The value I assign to the money at the point of initial decision would be represented as $p = 5 * .9$, giving p in this case a value of 4.5. Thus I would prefer the car at the time of my initial decision, given these values. The important thing to notice is that my preferences do not change on this scheme of future discounting. A year later, when I fully value the money, such that my p for the money is now 5, I still prefer the car (which now has a p of 5.67). If I had valued the car less initially such that the 'v' for the car was 6 I would prefer the money at the time of my initial decision (with respective preferences of 4.5 as opposed to 4.37) and I would still prefer the money a year later (5 to 4.86). For a proof of the unchanging relationship between these preferences over a period of time consider the following: let f be the time delay for the most proximate good and let x be the additional time difference between the first good and the second (and let f and x be exponents). The equation for exponential discounting $p_1 = v_1 * d^{f}$ bears the same proportionality to $p_2 = v_2 * d^{f+x}$ as $p_1 = v_1 * d^{(f \ldots - f)}$ bears to $p_2 = v_2 * d^{(f \ldots - f)+x}$ (because the equations are balanced at all points on the timeline), thus $p_1 = v_1 * d$ bears the same proportionality to $p_2 = v_2 * d^{x}$ as the first equations bear to one another, meaning that p_1 and p_2 will remain proportionately distant from each other on a preference curve at all points along the curve.

[22] Suppose in the example my discount coefficient was .7, in that case I would prefer the money.

reinforcement and withdrawal. This further increases the relative value (the value after the coefficient is applied) of an addictive substance or behavior.[23] These conditions make the addict a rational decision maker who is acting to maximize her good in much the same way as any rational agent who realizes that the future is not assured and discounts the value of future goods. So what appears to be myopia or impulsivity on the part of the addict is, according to them, a decision model already used by rational decision makers.

Becker and Murphy are touching on something important in understanding addiction. Accounting for the addict's preferences and the reasons why an addict assigns the value she does to an addictive substance or behavior is an important element of addiction. However there seem to be both factual and logical errors in their account. Factually they seem unable to account for the preference shifts that addicts show during the course of their addiction. According to the DSM-IV (Diagnostics and Statistics Manual of Mental Health Disorders, 4th edition) one of seven generic *criteria* for recognizing substance dependence are a patient's repeated attempts at trying to control or reduce her substance use without success.[24] That such effort is recognized as a criterion for diagnosis implies the prevalence of a lack of a stable preference within the addicted population.

There are also subtle, but important, logical problems in trying to account for addiction as a manifestation of rational exponential discounting. If Becker and Murphy

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[23] They refer to tolerance and withdrawal as "consumption capital". They define this as the idea that current consumptive levels of a good required achieve a particular (stable) level of utility depend on past levels of consumption of that good.

[24] Morrison, 1995, p.69

are right, and it is a radical discount coefficient which sustains addiction, it is unclear how a rational person could justify it. If a discount coefficient is low enough to prefer near term goods with a consistency that leads to addiction it seems that the mechanism producing such consistency might be an irrational basis for one's choices in the first place. Minimally Becker and Murphy should concede that certain discount coefficients are too radical to be counted as rational, especially when they discount the future in a way which is not in keeping with reasonable expectations of the future.

They might also have difficulty in explaining why a radical delay-discounter's preferences would not constantly be trumped by a preference for even nearer term goods, such that the discounter's behavior would rarely be consistent enough to produce the physical or psychological dependence characteristic of addiction. It seems one should expect that a low discount coefficient would produce general impulsivity that would be inimical to the consistent and focused behavior that is required to produce addiction. Becker and Murphy might reply that the substance of addiction is initially given a greater absolute value such that this counteracts rampant impulsivity, allowing the agent to consistently use an addictive substance.

But such a reply invalidates their own focus on delay-discounting as being the *primary explainer* of addictive behavior and raises another logical problem. If the value of the addictive substance or behavior is what thwarts certain impulsive behavior that might trump the development of an addiction, then they must admit that there are factors other than withdrawal and reinforcement that contribute to the absolute value assigned an addictive substance of behavior. But to do this would be to open to flood gates to

irrational assignments of value to an addictive substance or behavior, which would mean that within their own delay-discounting model the values might be irrationally assigned and so addictive choices could be grounded in irrational choice.[25] But that would be to doom exponential discounting as a rational model of addictive choice and to shift focus to how the value of an addictive substance or behavior is assigned.

George Ainslie proposes an account of addictive choice that is intended to address the factual problem with the exponential discount model. Ainslie writes that Becker and Murphy "cannot deal with the common case in which an organism knows he will change his choice in the future and is still at pains to prevent this."[26] Ainslie proposes that *hyperbolic discounting* is a way to account for the ambivalence and cross-temporal division in attitude that addicts have toward addictive substances and behaviors. Ainslie believes that preference changes and the loss of control reported by addicts is the result of a preference shift that occurs as the availability of an addictive substance or behavior draws near.

The difference between these types of discounting can be rendered graphically (see figure 1.1). First consider exponential discounting. Two exponential discount curves for two different future goods will rise gradually as the goods grow nearer. In exponential

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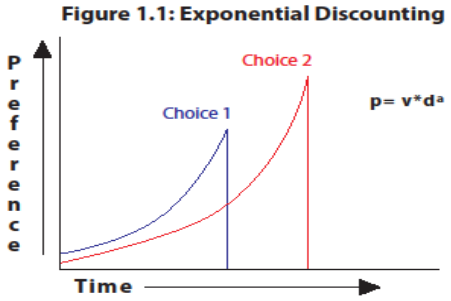
[25] By irrationally assigned value I mean something like the following: Suppose I start drinking to alleviate depression, despite evidence that alcohol worsens symptoms in the long run. In that case part of the value (v) I assign to alcohol in the preference equation $p_1 = v_1 * d$ is the value I assign it because it alleviates my symptoms of depression. Yet this an irrational assignment of value since I am might be willingly ignoring evidence that alcohol in fact has no such value. In short I can value things for irrational reasons and since value factors into the preference equation it follows that part of the reason I prefer what I do (part of why and how I make the decision I do) is rooted in irrationality.

[26] Ainslie, 1992, p.61

discounting the two curves, representing two distinct (mutually exclusive) goods occurring at different times, will not cross over one another. Meaning preference for one over the other will not change. Hyperbolic discounting is different. It is termed "hyperbolic" because graphically lines representing the preference of a hyperbolic discounter take the shape of the conic section. The ends of the curves (which represent preference) rise steeply, toward the tail end of the curve, as a good becomes imminent.[27] So, even with fixed futurity and unchanging absolute value, preferences over time represented by hyperbolic curves tend to cross over one another, and the person experiences a change in preference.[28] This means the good that was not initially preferred may become preferred as the discounter draws closer to its arrival. This is interesting because it allows hyperbolic discounting to capture the impulsivity and change in preference generally associated with addiction (see figure 1.1).

[27] Ainslie and Haslam,1999, use this description.

[28] See Yaffe, 2001, for a succinct explanation of hyperbolic discounting. It can be represented with the following equation: p = v / 1+a, where 'p' is a current preference, 'v' is the absolute value assigned a good, and 'a' is the number of temporal units of delay by which one is discounting the future. To see how the change of preference works take two mutually exclusive choices. Choice 1 occurs first and has values of 10 and 1 assigned to 'v' and 'a' respectively. Choice 2 occurs second and has values of 40 and 5. In that case choice 2 would be preferred (with a preference of 5 for choice one and 6.67 for choice two). But as the choices draw nearer and the value of 'a' is reduced choice 1 becomes the preferred choice.



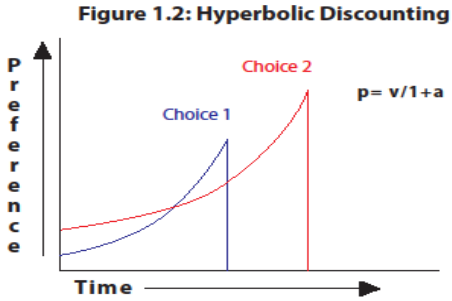


Figure 1.1

While Ainslie accounts for the phenomenon of the addict being divided against herself and the psychic struggle this can cause in overcoming addiction there are limits to the merit of his explanation. In some ways the problem is similar to the logical objection against Becker and Murphy. If hyperbolic discounting is a global disposition, then this suggests that an addictive behavior would routinely be trumped by other addictive behaviors or non-addictive behaviors since some number of goods will always be

available prior to the addictive good. But this does not seem to be the case with addiction, since it is the consistency of addictive choices that are essential to addiction. A further problem arises when one realizes that in claiming that such discounting is global, Ainslie must also claim that addicts use this sort of discounting in all or most other areas of their lives. While there is evidence that the impulsive are more liable to become addicts it is certainly not true that only those who behave in a generally impulsive way become addicts.

Perhaps hyperbolic discounting is a local disposition. If Ainslie makes this claim, then he must account for why it gets applied only to particular substances or behaviors.[29] But if there is an independent reason for such a localized application of delay-discounting then it would seem that that reason itself is equally important in an explanation of addiction.

One might also object that Ainslie's model of addictive impulsivity is somewhat artificial. Such a model assumes that it is by virtue of a large enough temporal gap between addictive and non-addictive goods that an addict behaves impulsively. But the impulsivity associated with addiction might come from the fact that addictive behavior is tempting and available not at set points in the future, with fixed relations to non-addictive goods, but looms continually.[30] It would seem the drive for the addict to use increases not

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[29] I am grateful to Agnieszka Jaworska for drawing my attention to this problem.

[30] It seems Ainslie misses this by generating an algorithm meant to capture *when* 'impulsive' preference reversals will occur. But why can it not be the case that addicts spontaneously come to prefer an addictive good, not because it is more immediate, but because the addict spontaneously comes to prefer that good and acts on that preference? This seems to be a more natural account of impulsivity. In particular it can account for multiple preference reversals over a short period of time when the addict has to choose between two goods (one addictive, one not). Anslie does not take account of repeated preference reversals.

simply because of an approaching decision point on a time line but also as a result of internal forces which operate independently of outside opportunity. Finally, one might object that Ainslie's theory cannot explain extended simultaneous self-division or uncertainty within the addict (or at least a constant shifting of preference over a short period of time).[31]

This is not to say that delay-discounting should play no role in a general theory of addiction. There is good evidence from the psychological community that addicts and non-addicts alike use it to make decisions. But it should play a limited role. Delay-discounting, be it exponential or hyperbolic, is too weak by itself to provide a foundation for a robust interdisciplinary theory of addiction. I believe the fundamental problem with such theories is that they give superficial gloss behavior which involves emotions, intentions, and beliefs with mathematical models that do a poor job accounting for such complexity. A more naturalistic approach might bring us closer to a good general theory of addiction and it is to that sort of approach I now turn.

§ 2. The Matching Law and the Melioration Theory of Addiction

Another type of theory is provided by Richard Herrnstein and Drazen Prelec. The theory, known as "melioration", is based on the pioneering work done by Herrnstein at the end of the 1960s. This work produced an observation in psychology so well supported

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[31] I am grateful to John Martin Fischer for pointing this last objection out to me.

that it came to be known as the "matching law".[32] The matching law maintains that subjects (human and animal) will tend to pursue the more immediately rewarding of two behaviors until that behavior has, due to overindulgence, ceased to be as rewarding as some alternative. The subject will then switch to the other behavior until the originally more rewarding behavior has 'rejuvenated' itself and again become more pleasurable than the alternative. This cycle of tending to overindulge in the more immediately rewarding behavior, until a marked diminishment in utility is recognized, followed by a switch to the initially less rewarding behavior, will continue until the *average* level of utility derived from each pursuit matches.

The problem, Herrnstein and others have found, is that this strategy can be sub-optimal in the long run.[33] This is especially true when one of the behaviors, which is initially more rewarding but whose utility diminishes over repeated engagement in the behavior, is paired with a behavior which is initially less rewarding, but whose utility increases over repeated engagement. As an example consider practicing the piano and watching TV. The matching law predicts that a person will initially choose to watch TV, as opposed to practicing piano, because it yields higher immediate utility. But overindulgence in TV gets old and as a break the person might take up piano practice, until the desire for TV rebounds and the next session of TV watching is better than the next session of practicing piano. The matching law posits that equilibrium will be set up

[32] Herrnstein first advanced the matching law in 1970. The form in which I describe it is discussed by Herrnstein and Drazen Prelec, 1992a. It roughly forms the psychological basis for the future discounting discussed by Becker, Murphy, and Ainslie.

[33] Herrnstein and Prelec, 1992a.

so that, in the long term, *each* experience with TV is as rewarding as *each* session of practicing the piano.

However, it would be more optimal (in terms of overall utility) to practice the piano more than one watches TV. The more one practices the piano (assuming one gets better) the higher the levels of utility from each session of playing.[34] Optimized behavior in this case dictates that one limit TV watching in order to ensure that each instance returns a consistently high level of utility by avoiding the overindulgence which diminishes returns. By engaging more frequently in the behavior of practicing piano, which returns progressively higher rewards (because one is getting better), one optimizes behavior so that cumulatively one is maximizing utility. When choice is optimal returns from piano practicing consistently rise while returns from instances of television watching are not diminished by overindulgence. Optimization sometimes requires the avoidance of matching.[35]

But matching not optimization was found to be a common behavior among Herrnstein's test subjects and he theorizes that matching is a product of evolutionary adaptation.[36] According to Herrnstein and Prelec addiction is the consequence of the

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[34] This is true even if the most enjoyable session of TV watching outstrips the most enjoyable session of playing the piano, because unlike the piano (or other acquired or improvable skills) TV watching tends to diminish in the value of its return over time.

[35] It is worth noting that each instance of TV watching could be valued more than each instance of practicing within optimized behavior. But what is at issue is the total utility derived from the two behaviors in combination over the long term.

[36] Herrnstein and Prelec cite several studies in their1992a article to show that matching is a common phenomenon. Herrnstein theorizes that given the uncertain nature of future events (especially in primitive environments) taking nearer, higher rewards is more conducive to survival that seeking longer term rewards

matching law being applied to behaviors that tend to produce withdrawal, reinforcement, and tolerance. The practical difference between matching non-addictive goods and matching an addictive good(s) with a non-addictive good(s) is that addictive goods in general not only decrease their value with repeated use but they push the value of alternatives down as well.[37] This means that even though repeated use of an addictive good offers a diminishing amount of utility per use, non-addictive alternatives have been driven down as well and the 'matching' point for the addict (between addictive and non-addictive goods) gets stuck around an equilibrium that is well below where it would be for two non-addictive goods. This significantly drives down the addict's overall utility.

The melioration theory asserts that this low level of utility continues because in using the matching law the addict gets caught in a vicious cycle. The one behavior that will return the highest rate of utility (at a given decision point) is, due to withdrawal and reinforcement, usually the behavior that keeps them in the cycle. At the heart of Herrnstein and Prelec's analysis is their observation meant to explain the value of an addictive substance or behavior relative to other choices, "value functions depend on levels of allocation…and one cannot readily sample from other points across the range of allocations".[38] Like Ainslie, they believe that increasing levels of indulgence have great relative or near term value, but that it is difficult to recognize the diminishment in long

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that are initially less rewarding. The theory is well respected and George Lowenstein, Jon Elster, George Ainslie and others discuss it at various points in the literature.

[37] This relies partly on the opponent process theory of addiction, a theory of addiction that will be addressed later in this chapter.

[38] Herrnstein and Prelec, 1992, p.55.

term value that comes from these near term choices (i.e. they suppose value assessment is made in terms of greatest immediate utility). Herrnstein and Prelec can account for the seeming impulsivity and changing inclinations of the addict.

Herrnstein and Prelec take a more naturalistic approach to addiction than does Ainslie. They do not think of the addict as a delay-discounter so much as the subject of immediate pressure on her well-being. There is little emphasis in their model on considerations other than immediate utility selections from a range of contemporaneous alternatives. This addresses one of the objections I raised against Ainslie. On Ainslie's (Becker and Murphy's, also) view the motivating influence to use addictive substances comes largely from the approach of an opportunity to use. But it seems reasonable to think that often it is not so much the approach of an opportunity as it is internal considerations which motivate the addict to make the decision to use. Herrnstein and Prelec are better able to account for this since melioration, in part, is a response to diminished levels of general utility and the need to address undesirable feelings such as withdrawal.

But there are problems with their theory. First, it seems to give parameters to addiction that it should not be thought to have. The melioration theory implies that addiction is the vicious cycle of behavior that results from repeated use of certain kinds of substances or engagements in certain kinds of behaviors. This means Herrnstein and Prelec's theory runs out of explanatory force at a certain point. Once the utility of non-addictive choices return to normal addiction would seem to be over and the addict recovered or sober. But this is in tension with other thinking that even after withdrawal

and a return to normal levels of utility for non-addictive goods that addiction has not necessarily ended. The DSM-IV, "defines recovery as the absence of a current substance use disorder", and lists time indexed criteria for full, partial, early, and sustained remission.[39] But, as James Morrison notes," This is not entirely satisfactory. For one thing a large number of experts believe "Once an addict, always an addict." A considerable body of experience supports this impression."[40]

Other sources also demonstrate the problems in clarifying the boundaries of addiction. The American Society of Addiction Medicine defines recovery as, "A process of overcoming both physical and psychological dependence…with a commitment to sobriety"; and sobriety as, "A state of complete abstinence…in conjunction with a satisfactory life".[41] While the waters here are murky it is critical for any comprehensive theory of addiction to either justify an endpoint for addiction, explain why that endpoint is hazy, or to explain why it does not have one. However, the melioration theory stops short of meeting its obligation in this regard.

Nor does melioration theory explain relapse that is the reacquisition of addictive behavior. Why, after the addict breaks out of the vicious cycle of addiction, is she at a risk -often a high one- of returning to that cycle weeks, months, or even years later? It seems *overly simple* to suppose that the addict repeats the cycle by again incautiously

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[39] Morrison, p.77, 1995, the criteria for early full remission require that a patient meets no criteria for substance abuse for 2-12 months. So up to 1 year after the patient last experienced problems the DSM-IV places the recovering addict in a category below full sustained remission. This implies that addiction, fully understood, has fuzzy boundaries.

[40] Ibid. p.78.

[41] Graham and Schultz, 1998, p.1303

applying the matching law to the same (or a similar) addictive good. It is problematic for the same mechanism to explain both relapse and the initial acquisition of addiction (i.e. matching behavior). If this were the case, then one would expect addicts to be roughly as susceptible to relapse as non-addicts are to becoming addicted (since the matching law is not particular to addicts), but this is not the case. Relapse rates for addicts are often orders of magnitude higher than rates of addiction in the general population, implying something other than the matching law is at work in relapse.[42]

It is because Herrnstein and Prelec do not offer an account for why, as is pointed out in the work of the American Society of Addiction Medicine, "compliance and relapse in addictive disease are comparable to rates of relapse in other illnesses such as diabetes and hypertension," that I think they give an incomplete theory.[43] The theory must be supplemented so that the boundaries of addiction and the problem of relapse can be given a better account.

There is also a sharpness problem for the melioration theory. Melioration, like hyperbolic discounting, assumes that an addict's choice is the result of the failure of non-addictive alternatives to be competitive with addictive ones in terms of utility. This is not incorrect, but it emphasizes addictive substances or behaviors are preferable largely

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[42] Prochaska, et al, 1992, pp. 1102-1114. According to these authors rates of relapse for alcoholics (use occurring after the end of withdrawal symptoms) reaches roughly 60% within 6 months after beginning abstinence (the rate of abstinence then remains fairly stable). Rates of alcoholism are significantly lower than this in the general population. According to a 2001-2002 study by the National Institute on Alcohol Abuse and Alcoholism 3.81% of the total population (not controlling for gender, age, or ethnicity) would meet DSM-IV criteria for alcohol dependence. While this does not definitively undercut the melioration theory's ability to address relapse it certainly calls it into question.

[43] Kasser, et al, 1998, p.425. I caution the reader that I do not at this point want to make any claim about whether addiction is a disease. The authors use that term and I found it difficult to omit the word.

because of a depression in the value of non-addictive goods. Such emphasis misses the emotional returns on utility that addictive substances can have even in later stages of addiction.[44] Melioration does not account for the fact that an addictive substance (or behavior), more than merely being the best of a set of alternatives, can in some ways be seen as a good alternative in and of itself, as an answer to some deep emotional of psychic problem. If this is true, then the melioration theory seems to ignore an element of complexity in the picture of addiction. The complex wrinkle it misses is that an addict can deeply emotionally value her addiction and that that deep value, at times, can drive her choice.

The melioration theory accounts for the problems of impulsivity and poor long term decision making in the addict as did hyperbolic discounting. Ultimately it is an improvement over Ainslie's theory (which is an improvement over Becker and Murphy's). Melioration better accounts for the mechanisms of an addict's choice to use, not as a decision point toward which the addict is pulled by the passage of time, but by accounting for them as the product of utility considerations confined to immediate wants. It can also explain, in a way that Ainslie would find difficult, not only why an addict might feel divided against herself at different times, but at the same time. The strengths of the melioration theory foreshadow some of the elements of my own hybrid theory, just as its shortcomings portend the points on which I will expand in the coming chapters.

[44] In chapter 3 I will discuss the emotional or passionate appeal of addictive substances. In such cases the choice to use is not seen in stark relief with alternative sources of utility, but is seen as a sine qua non for the achievement of any happiness whatever.

§ 3. The Visceral Factor and Opponent Process Theories

The visceral factor and opponent process theories of addiction are even further away, than is melioration, from the cognitively based delay-discounting theories. The two theories are together in this section because they are similar in that they both emphasize visceral non-cognitive factors as driving addiction. Unlike delay-discounting, and even melioration, visceral factor and opponent process theories do not centralize cognitive judgments about utility. Instead, they offer a model of addiction as a behavioral response to strong and primitive motivational forces, much like those that drive the desire for hunger and sleep.

George Loewenstein advances the visceral factor theory of addiction. In it he gives craving (especially cue-conditioned craving) a central role in explaining addiction.[45] According to Loewenstein craving is a visceral factor, and visceral factors in general are, "associated with regulatory mechanisms that are essential for survival…[and] are also associated with behavior disorders."[46] Visceral factors exist for basic needs such as food and drink and are capable of driving behavior by themselves, sometimes in ways contrary to one's better judgment or without any particular higher order judgment at all.

Loewenstein points to several virtues of his account of craving as constitutive of addiction. First, visceral factors directly motivate behavior, without a need for comparing

[45] The main proponent of a 'visceral' or 'craving' based account of addiction that I will examine is Loewenstein, but a great deal of the evidence that he cites in support of his position comes from various sources. Avram Goldstein (1994), Drazen Prelec (in his work with Loewenstein (1996)), and the work of various other authors who have studied the relationship of the brain's reward system and memory on self-control and addiction is used as evidence by Loewenstein, so his representation of visceral factors is not singular.

[46] Loewenstein, 1999, p.235

or considering a range of choices, because of their immediate and direct effects. It is the nature of visceral factors that they are given to quick changes and tend to increase and decrease in strength in relatively short periods of time. That is, craving can arise fluidly and at unexpected times, thereby accounting for shifts in the preference shown by an addict.[47]

Loewenstein can also account for the divided-self of the addict. He contends that addicts (and others) generally have a poor ability to predict how visceral factors will effect their future decisions. He has found that predictions about future behavior which *will* take place in the grip of a visceral factor (e.g. an urge) –when the prediction is made *outside* the presence of that factor- are generally poor in quality.[48] This provides Lowenstein with an account, different from delay-discounting, as to why, "addiction is so commonly associated with inner conflict and attempts to control one's own behavior."[49] In the case of craving it is because, as a visceral factor, it is often in conflict with cognitive deliberation and can, and often does, win out in influencing behavior.

[47] I believe that Becker and Murphy, as well as Ainslie, are committed to addiction as a more crystallized or routine behavior with use occurring at relatively fixed or routine time intervals. This is probably true of addiction to some extent, but I object to the degree to which they emphasize this, which makes the fluidity of Loewenstein's theory a virtue from my point of view.

[48] Loewenstein, 1999, cites several studies to support this claim. In one interesting study Loewenstein, et al, asked (male) college students to watch a video of a classic scenario leading to a 'date' rape. The subjects were then asked to describe how they would have behaved in the same scenario. The experimental group of students watched this video having recently been exposed to sexually stimulatory material while the control group was not. The experimental group predicted more sexually aggressive behavior of themselves than did the controls. This lead Loewenstein (et al) to believe that the control group was under predicting their sexual aggressiveness because they were not, at the time the prediction was made, experiencing the same visceral factors they would in the actual scenario.

[49] Loewenstein, 1999, p.252

Another advantage which Loewenstein points out is that the visceral factor theory is able to explain certain unique patterns in the use of an addictive substance or engagement in a behavior. Addiction to a drug, as an example, does not always necessarily involve the continuous use of a drug. Certain models of addictive drug use suggest that there are cases where periods of excessive use are followed by spontaneous periods of abstinence.[50] The previously discussed approaches would have trouble explaining spontaneous 'gaps' in use.[51] But, "craving, which is assumed to be the major force driving addiction, is as transient as any other type of visceral factor."[52]

Loewenstein also accounts for relapse. According to him, "Relapse results from misinformed decisions taken with an under appreciation of the impact of future craving."[53] Loewenstein cites evidence to suggest that reinstatement of craving can take place either with small amounts of exposure to an addictive substance or behavior, or environmental cues associated with the addictive substance or behavior. In both cases it is the overestimation on the part of the addict that she can handle re-exposure to an

[50] E.M. Jellinek discusses this (i.e. binges separated by periods of abstinence) as being a constitutive behavior of the Epsilon variety of alcoholism.

[51] This is because utility maximization models suppose that whatever someone's preference with respect to future discounting that discount preference remains stable throughout the course of an addiction (and even beyond) which makes spontaneous cessation unlikely (i.e. it would seem to require someone undergo a paradigm shift in their view of how they ought to discount the future). But there is some indication that spontaneous cessation (and in some cases the resumption of use) occurs with addicts on a not infrequent basis. See Kincaid and Sullivan, 2010, p. 368 and Fingarette,1988.

[52] Loewenstein, p.252, 1999

[53] Ibid. p.254

addictive substance/behavior or an environment with which it is associated, and the resultant craving, which leads to relapse.

Loewenstein's visceral factor theory is a highly refined and advanced cousin of the opponent process theory. The opponent process theory of acquired motivation, described by Richard Solomon, posits that the high or euphoria of addiction has a corresponding 'opponent' process which results in dysphoria or anhedonia -the inability to feel pleasure normally. The neurobiological underpinnings for the theory are strong. The following is an explanation of the idea: Due to the excessive production or stimulation of certain hormones or neurotransmitters, (a hormone can function as a neurotransmitter) by drugs or behaviors of addiction, the body, attempting to maintain homeostatic equilibrium, increases production of hormones or neurotransmitters with opposing effects. When the addictive behavior ceases the body is not prepared to independently maintain the high levels of the neurotransmitters which were produced by the drug. However, it is prepared to continue to produce high levels of the 'opponent' neurotransmitters.

A similar phenomenon is commonly observed in people who use steroids or hormone replacement therapy and, upon suddenly stopping, develop traits most commonly associated with the opposite gender. In addicts this 'opponent' process is thought to be responsible, in large measure, for symptoms of withdrawal. The opponent process theory postulates that continued use of addictive substances or engagement in

addictive behaviors is a response to the neurobiological imbalance created by the dysphoria or anhedonia that results from addiction. [54]

One major refinement that the visceral factor theory makes to the opponent process theory is that it accounts for continued use and relapse in terms of cue-conditioning. This is an improvement because it is in keeping with the observation that in many cases addicts use subsequent to the stopping of withdrawal.[55] So, as Loewenstein points out, his theory more accurately represents empirical findings about addiction.

The visceral factor theory contributes largely to one of the two elements that comprise my hybrid theory, and there is much to like about it. But Loewenstein's theory is too much of a shift from the head to the gut. Must visceral factors eliminate or so significantly diminish the role of beliefs, desires, and choices that these take a backseat in constituting addiction or explaining the behavior associated with it? It does not seem in establishing visceral factors as a major contributor to addictive behavior that Loewenstein has eliminated the need to rely on cognitive elements that might contribute to addiction. For instance an addict may believe that only by gambling, taking morphine, or drinking is she able to feel deeply satisfied. As a corollary to this it is unclear whether the divided-self of the addict is primarily a product of visceral urges and cognitive deliberations. Deliberations about the worth of an addictive good could well account for a significant

[54] Solomon, 1980, there is a growing amount of evidence that substance addictions (at least) do result in the 'up regulation' or increase of neurotransmitters that have effects opposite of those produced by the substance of addiction and cause drug seeking behavior. The article by De Witte, 2004, gives an example with respect to alcohol dependence.

[55] Loewenstein, 1999, p.248

portion of the inner conflict. Likewise, the nature of the distinction between addictive and non-addictive visceral factors needs to be made, and Loewenstein does not do this.

In what remains of the chapter I begin the process of finding a central point of gravity for the theories I have examined. The illuminating elements of these theories will eventually be pushed together into a coherent whole. The elements of the theories which are incongruous with the whole should naturally be cast off. Two philosophical theories of addiction provide the center of gravity towards which I begin moving these respective theories.

§ 4. Disordered Appetite and Existential Dependence

There are two conceptual elements central to the hybrid theory. The first claims that much of the phenomena associated with addiction, and existing theories meant to explain addiction, can be thought of as a disordered appetite. But to stop there would be to leave explanatory gaps for which I have criticized other authors in this chapter. So I also propose that much of the remaining phenomena can be explained as manifestations of a disordered passion, which is the second major constitutive element of the addiction concept. The challenge is in combining these two parts into a comprehensible whole. I propose that, *conceptually*, addiction is constituted by these two separate disorders and I leave open the possibility that they may cause or interact with one another.[56] So a disordered appetite may lead to a disordered passion or vice versa, but ultimately disordered passions and disordered appetites can be independent features of addiction in

[56] I also leave open the possibility of other conceptual elements constituting addiction.

an individual addict.[57] For instance, I argue that addiction in its rudimentary forms is capable of being described by a disordered appetite alone, but a comprehensive understanding of the complex symptoms and phenomena with which the addiction of a *moral agent* is associated requires both of these elements.

Gary Watson and Francis Seeburger offer philosophical analyses of addiction that form the basis for the two parts of the hybrid theory. Watson's description of addiction is the foundation of the disordered appetite element of the theory.[58] Seeburger posits that addiction is a form of existential dependence, and this forms the foundation for disordered passion.[59]

I believe Watson's position is essentially correct but vague and in need of fleshing out. I am critical of some of Seeburger's arguments but I believe he touches on something which is often overlooked in descriptions of addiction that confine themselves to basic biological forces or dispositions in decision making.

Watson wrote, "To acquire an appetite is to acquire a felt need, a source of pleasure and pain, that has a motivational force that is independent of one's capacity for critical judgment."[60] He went on," Addictions dispose us to be led on and distracted by pleasure, as though it were our master."[61] Watson admits that he cannot tie down the

[57] The purpose for these distinct elements will be made clear later. One important reason is the need to account for the fact that addiction can arise in complex, moral agents (such as persons) as well as in animal agents.

[58] Watson, 1999a

[59] Seeburger, 1999

[60] Watson, 1999a, p.13

[61] Ibid. p.19

concept of an appetite and that it needs fuller treatment than he is able to give.[62] Prima facie, his view might not seem importantly different from Loewenstein's visceral factor theory, but it is. Loewenstein emphasizes one visceral factor in particular, craving, as the driving force of addiction. But I prefer the term appetite to visceral factors. Presumably visceral factors are a manifestation of a disposition to have cravings, but Loewenstein does not discuss visceral factors in terms of a standing disposition to have things like craving, only that these factors are often associated with things necessary for our survival. An appetite is, conceptually, a disposition to repeatedly have certain visceral factors manifest themselves (which implies that appetite is a bit more specific than visceral factors since, by definition, an appetite is a standing disposition, as opposed to visceral factors, which might include emotions like anger or empathy, which may be important to survival, but for which one may not have a standing disposition –i.e. a disposition that does not require an environmental influence- to engage). I believe the term appetite more adequately captures the fundamental forces driving addiction.

There is further reason to think that this distinction is important to a theory of addiction. There are instances of withdrawal which bear similarities in the appetitive influence of thirst or hunger to someone who is dying of dehydration or starvation. There are cases of heroin and even alcohol withdrawal which have been lethal, and seizure, hallucination, and severe illness are not uncommon in withdrawal from more severe chemical dependencies.[63] Although these cases would be captured by visceral factors as

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[62] Ibid. p.15

[63] Trevisan, et al., 1998, p.61. According to this source alcohol withdrawal can result in spasmodic

Loewenstein conceives of them the specificity of appetite seems preferable to capturing what drives addicts in such cases.

Francis Seeburger's contribution to the analysis of addiction focuses not on the felt needs or wants of the addict, but on the general existential dependence that can characterize addiction. His view is complicated, but according to him an addict is essentially someone 'seeking more' from their lives. Addiction offers the addict that 'something more' that she seeks. The addict is, in disposition and behavior, like a religious devotee. She is trying to find meaning in her life through strong, perhaps total, commitment to an addiction (as a religious devotee would commit themselves to God). This is, according to Seeburger, the 'existential dependence' of addiction that characterizes the addict. The addict sees a deeper meaning in that to which they are addicted. [64]

Seeburger's notion of addiction is interesting. His fundamental claim is too strong to have applicability to addiction in general (nicotine addiction would seem to be an exception), but there are certain arguments he makes which are quite helpful. In particular his exploration of *how* addiction gets imbued with meaning and *what kind* of meaning addiction has for the addict is illuminating. It also provides a unique context within which to discuss certain biological explanations of addiction.[65] Ultimately it is

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muscular seizures; delirium tremens (hyperactivity of the autonomic nervous system and hallucinations); depression; anxiety; and serious sleep disturbance. In extreme cases death has resulted.

[64] I am summarizing Seeburger's view from *Responsibility and Addiction*, 1996.

[65] I also think that Seeburger's claim that addiction is a search for meaning might explain why certain addicts engage in addictive behaviors as a solution to preexisting psychological problems.

relevant to my own theory because it forms the basis for the idea of disordered passions as explaining some of the phenomena associated with addiction.

To this point I have introduced theories about addiction and indicated, generally, what I think can be done with them in the way of creating a unified theory. This was necessary so that the reader will know what sources I am drawing from when I discuss disordered appetites, disordered passions, and the hybrid theory. As it becomes necessary I will return to the ideas examined in this chapter and provide additional details. Chapter 2 begins with a discussion of appetite and provides a neurobiological justification for thinking that addiction is a disordered appetite. I discuss in detail what is meant by the word 'disordered' in this context and explore how addiction understood as a disordered appetite can answer some of the questions I laid out in the introduction.

Chapter 2

Addiction as a Disordered Appetite

The neurobiological underpinnings of addiction lend support to the idea that the concept of addiction is can be understood, in part, as a disordered appetite. In this chapter I will do four things. First, I give an overview of the function of specific areas of the brain implicated in reward and the role of the neurotransmitter dopamine in this process. In connection with this I examine some of the neurobiological mechanisms of food appetite. Second, I review some of the common neurobiological mechanisms of addiction. Third, I consider an existing theory that attempts to explain addiction, in part, as a problem with the way the brain provides incentives for appetite-like behavior. Finally, I will explain what it means for an appetite to be disordered.

§1. A (Brief) Overview of the Role of the Midbrain and Dopamine in Reward, Wanting, and Motivation

Before proceeding I need to briefly discuss three concepts: reward, reward mechanism, wanting, and functional motivation. A reward is an object or behavior to which an organism is generally attracted and which an organism is inclined to pursue. Rewards, as I discuss them in this chapter, should *not* be understood in a phenomenological sense as something which produces conscious pleasure.[66] In the context of the neurobiological discussion of addiction (and behavior in general) "reward

[66] In fact there are some indications that not all rewards are pleasant and that mice can be made to work for aversive foot-shocks. See Wise, 2006, p. 1152

mechanisms" is a designation for those parts of the brain which serve a functional role in generating positive stimulation from environmental cues associated with a reward (i.e. this is known as positive feedback, there is also negative feedback generated from something that an organism might want to avoid). The feedback provided by the reward mechanisms positively reinforces both conditioned and operant behavioral responses to a reward/reward cues.[67] In essence, reward mechanisms promote approach behavior. These mechanisms are aroused by certain perceived stimulus, motivate behavior through wanting, and record the degree of positive feedback from engagement with the reward itself (as a sort of reference for the future).

Wanting, in the neurobiological context, is also a functional term. A want is the actual directing of an organism's attention to a reward. In the functional sense it is an extension of the activation of the reward mechanisms. However, a want is distinct from the activation of reward mechanisms, although each is part of the chain that results in the pursuit or the motivation to pursue a reward. Reward mechanisms can be stimulated independently of consciousness (e.g. when an animal is anesthetized), although in that event they do not serve any part of a functional role. However, wanting is a necessarily functional mechanism which directs the stimulated reward mechanism with potential to direct conscious behavior. It is unintelligible to think of wants occurring, or attention being directed, when an animal is in a state of unconsciousness.

[67] Conditioned response is similar to the type of response observed in Pavlov's dogs. Conditioned response is simply anticipation of an imminent reward associated with an environmental cue. It may only involve the autonomic nervous system and it does not involve any particular conscious or learned *behavior* (only a learned cue). As was the case with the dogs when they began salivating upon hearing a buzzer associated with feeding. Operant condition requires a particular (learned) behavior prior to the receiving of a reward.

There are two senses of wanting which I will use, and reward mechanisms play a role in causing or establishing each. There is wanting as an *occurrence, or state*. Wanting, in this sense, is the directing of an organism's attention or the focusing of desire, which is brought about by the stimulation of the reward mechanisms by some reward. The philosopher T.M. Scanlon comes close to capturing what I mean by this when he discusses desire in the directed-attention sense. Scanlon writes, "A person has a desire in the directed-attention sense that P…if the person's attention is directed insistently toward considerations that present themselves as counting in favor of P."[68] Wanting has the function of making an organism aware both of *the types of rewards* that are available, and it also dictates the degree of attention that each reward receives (I call this second mechanism reward salience and will discuss it in a moment). Since wanting as an occurrence can direct attention to the variety of potential rewards conflicting wants are a common occurrence.

Wanting can also be understood as a *disposition*. On an extended time line, given repeated positive experiences arising from the reward mechanisms in response to a particular reward (and consistent cues associated with the reward), an organism can develop a disposition to want a particular reward under particular conditions.[69] For example, Pavlov's dogs were disposed to want food upon hearing a certain buzzer go off,

[68] Scanlon, 1998, p.39

[69] There might be "negative" wants in some sense (e.g. A lab animal 'wants' to avoid punishment or electric shock that results from certain sorts of behavior….I 'want' to avoid rattlesnakes). But I do not investigate these since they are largely irrelevant to my examination of addiction. In so far as such 'wants' relate to the reward mechanisms under discussion it seems that it is a lack of stimulation that accounts for avoidance or negative wants. Please see Giorgi, et al, 2007 for a discussion of how avoidance (negative wants) can result from a lack of dopamine receptors in the midbrain.

whether there was actually food present or not. In this way the reward mechanism helps to establish a disposition toward an occurrence of wanting by wiring itself to respond to particular cues. So even if an organism is not in a state of wanting, it can have a disposition to want a particular reward. This means that cues associated with some reward generally lead to an occurrence of wanting. The dispositional sense of wanting – being disposed to having the reward mechanisms cause an occurrence of wanting in the presence of reward cues- is especially relevant to addiction.

However, all occurrences of wants are not the same. They can differ in the strength or degree of attention they draw from the organism. One occurrence of a want can be distinguished from another by the degree of *salience* of the respective rewards that are wanted. Salience is the desirability or strength of attention that is given to (or perhaps caused by, since this is to be thought of functionally) a potential reward. Wants can have similar salience, in which case it can be hard to decide which is preferable.[70] However, all else being equal, an occurring want with the greatest salience is likely to be the want that motivates behavior. It is worth noting that while wanting is conceptually distinct from reward without recognition of potential rewards (via mere arousal of the reward mechanisms), wanting is inert or incoherent (i.e. to want 'something' without 'knowing', even instinctively, what, is to not want anything at all, in the functional sense). A want is the directing of attention and the measure of a want is taken by its salience, or the strength of that attention. An exceptionally salient want can produce functional motivation, the third concept I wish to discuss.

[70] I believe this is the root of the supposed problem with Buridan's ass, each pile of hay is equally salient.

A Functional motivation can arise directly from the morass of conflicting salient wants. It can also be the product of inhibition, reflection, and learning applied to conflicting salient wants. A functional motivation essentially *is* the last step in the motivational chain prior to action (that is not to say that functional motivation results in immediate action since one might still need to plan). Functional motivation can be the product of deliberate and considered reflection applied to a want, as when someone thoughtfully chooses to join the army in a time of war. It can also arise in rash and unreflective ways, as when someone punches another for a minor insult. The distinct thing about functional motivation, as opposed to other motivations arising from wants, is that it has passed by our (or any organism's) inhibitory, reflective, or rational checks that are applied to wants. It is a mystery of agency why there is *inconsistency* in how we 'decide' which salient wants get left behind and which become functionally motivating. Sometimes it seems that higher-order organisms (especially persons) are capable of carefully and rationally choosing between motivating wants, and sometimes it does not. However, the decision making apparatus of higher-order organisms (especially persons) is complex and it is important to distinguish this step in the chain of action. Functional motivation reflects the motivation that 'gets through' higher order checks on salient wants. In lower-order animals functional motivation can be thought of as just the most salient want.

There is explanatory value to this picture of reward mechanisms, wants, and functional motivation. The loss of 'willpower' in the face of an exceptionally salient (in absolute terms) want, one which overrides or overpowers behavioral or rational

inhibitions of motivation, can be explained. In such a case the translation of a motivation associated with a salient want into functional motivation was not or could not be impeded by rationality. This picture can also account for conflicts in wants (due to varying levels of salience). *Perhaps most importantly*, it can explain the failure of a functional motivation to correspond to the most salient want (under certain conditions), since inhibitory or rational controls can play a role in the motivational process. That is, the view can account for what is traditionally thought of as *willpower* -the trumping of salient wants prior to the functional motivational level by rational reflection.[71] This functional picture can also explain aberrant wants. As an example, the loosening of what counts as food, under starvation conditions, can be explained by the increased salience of hunger. Under conditions of exceptionally strong salience the range of what is wanted is understandably expanded beyond what inhibitions can prevent from becoming motivationally effective (i.e., a strong want is more sensitive to arousal because it is more demanding of satiation). At the same time, this view nicely explains typical cases of functional motivation: for instance, hunger brought on by seeing or smelling a cake and the resulting functional motivation to eat the cake (especially when one has forgotten about one's diet!).

The understanding of these mechanisms is important for understanding not only addiction, but appetitive behavior in general. Fundamental to the survival of any species, even those without complex brain-based behavioral mechanisms, is the need for

[71] There is no restriction, in my scheme, on rational sources that can trump the simple motivational effect of a strong want from producing a functional motivation. This could include moral reflection. There is no reason to think (in many cases) salient occurring wants cannot be trumped prior to some motivated behavior by rational intervention.

individuals within that species to be adapted with general biological feedback (behavioral reinforcement) mechanisms which indicate whether an environment or stimulus is conducive or hostile to life. Plants whose flowers face the morning sun, ants that follow the chemical signals of scouts to a picnic, and bears that later sniff-out the refuse from the picnic the ants ruin all exhibit this sort of environmental feedback. Of course, unlike in plants and ants, the location of the feedback mechanism in higher-order animals is the brain. In humans –as in rats and mice, and all other higher-order mammals- a central component of the biological feedback mechanism is the reward circuitry of the brain, specifically its midbrain (mesotelencephalic) dopamine circuitry.[72] This system is complex and it spans several areas of the brain as well as different parts within those areas, including:

> …a variety of brain-stem, midbrain, and forebrain loci, including most importantly the ventral tegmental area, substantia nigra, hypothalamus, medial forebrain bundle, septum, amygdale, neostriatum, nucleus accumbens, olfactory tubercle, and portions of the cingulated and frontal cortices.[73]

It is these neurobiological mechanisms to which I now turn. A complete understanding of the midbrain's interconnections as well as its connections with other parts of the brain is a vast undertaking. I will focus on select structures that are commonly associated with addiction and appetite. Restricting my examination to these areas of the brain will be sufficient to make the case that there is good reason to believe

[72] See Copper and Zhou 2006, and Gardner and David, 1999; Gardner and David note, "The existence of reward or pleasure circuits in the mammalian brain had been postulated either explicitly or implicitly as part of every behavior theory from Darwin's time onward (ranging from the older Darwinian, Freudian, neo-Freudian, Pavlovian, and Skinnerian theories to the integrated brain-behavior theories that dominate modern psychology)." It is largely taken for granted in neuroscience that the brain's reward center plays a central role in our behavior.

[73] Gardner and David, 1999, p.96

addiction is a disordered appetite. This narrowing of focus will also make for a clearer discussion. However, my selection of which structures to consider is not arbitrary, although it will leave some details unexplained. As Gardner and David note about the aforementioned areas of the brain," It was soon realized…that all these areas are interconnected by the ascending and descending tracts of the medial forebrain bundle- the nuclei, tracts, and the projections of which connect all major brain sites positive for electrical brain-stimulation reward."[74]

These parts of the brain share the common feature of having high concentrations of dopaminergic neurons (i.e. neurons which use the neurotransmitter dopamine to send inter-neuronal signals). The first area that will concern us is the medial forebrain bundle (MFB). The MFB is a collection of "fibers on their way from the brainstem to widespread cerebral areas."[75] The MFB's role in reward and appetite is as a relay center for neural connections between various parts of the brain. Two other important areas are the ventral tegmental area (VTA) and the nearby substantia nigra (SN) which are rich in dopaminergic neurons and frequently implicated in studies of addiction. The VTA is a "region of the midbrain medial to the compact part of the substantia nigra, containing domapinergic neurons that project to various limbic and neocortical areas."[76] The VTA sends afferent neural projections to the MFB, the nucleus accumbens (NAc), and the

[74] Ibid, p.96

[75] Nolte, 2009, p.674

[76] Ibid, p.689

cerebral cortex.[77] The NAc, in turn, has afferent neural projections which go into the frontal cortex -a part of the brain associated with emotion and decision making. [78] The NAc also has inhibitory neural outputs which through an intermediary connection "modulate the output of [the] frontal cortex."[79] For the purposes of my examination it will suffice to confine discussion of reward mechanisms to these parts of the brain.[80]

The VTA, MFB, and NAc each play a critical role in the reinforcement of motivation and behavioral mechanisms. As Jerzy Vetulani notes, "Behavior of mammals (and presumably lower animals) is a resultant action of three large functional systems of the brain: arousal [sensory], reward, and cognition system. These systems are closely interconnected and are necessary for proper functioning of the organism."[81] Addiction involves repeated and especially strong motivation to engage in a particular behavior or use a certain drug. It should come as no surprise that studies of addiction routinely associate certain kinds of activity in the reward mechanisms of the brain with abnormality in reward, wanting, and motivation.[82]

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[77] Cooper and Zhou, 2006, p.137

[78] The prefrontal cortex is especially associated with emotion, see Vetulani, 2001, p.304.

[79] Nolte, 2009, p.661, of the basil ganglia Nolte writes "damage to certain parts of the basil ganglia can cause disturbances of cognition and motivation."

[80] For a good discussion of the various connections arising from and within the midbrain dopaminergic pathways see Cooper and Zhou, "Development of the Midbrain Dopaminergic Pathway", in Madras, et al., 2006, pp.137-156. When I use the term midbrain it can be taken to refer collectively to the areas of the brain discussed in this paragraph, with the exception of the frontal cortex.

[81] Vetulani, 2001, p.304

[82] I will discuss studies throughout the dissertation, see Chiu, et al, 2008; Dawe, et al, 2004; Hollerman and Schultz, 1998; Vetulani, 2001; and Berridge and Robinson, 2011.

The presence of the neurotransmitter dopamine, in these areas of the brain, plays a central role in reward, wanting, motivation, and even learning.[83] Studies demonstrate the connections between dopamine and the general learned feeding behavior of mice. According to R.A. Wise dopamine antagonists, drugs which impair the function of dopamine, administered to mice," impair learning…by extinguishing…instrumental responding for food. Several lines of study confirm that they do so by blunting reward function itself rather than by simply impairing performance capacity."[84] Dopamine seems to play an even more fundamental role in feeding behavior. Some studies indicate that a lack of dopamine is inimical to the drive for food intake necessary to sustain life. Wise discusses a study in which mice born without the gene necessary to produce tyrosine hydroxylase –a precursor of L-DOPA, which itself is a precursor of dopamine, "eat and gain weight for 10-15 days…Unless treated with L-DOPA, they then lose weight, usually dying by four weeks of age."[85]

It should be noted that dopamine's role in food intake is complex. There are, for instance, 5 different types of dopamine receptors (D1-D5). Two of these, D1 and D2, are especially important in the feeding behavior of young mice. In another study researchers found that mice with these receptors knocked-out (i.e., mice genetically engineered without D1 and D2 receptors) in the midbrain (and elsewhere) are usually dead within the

[83] For discussion of the numerous behaviors associated with dopamine see Wise, 2006, pp.1149-1158

[84] Wise, 2006, p. 1149

[85] Ibid.. p. 1151, the life expectancy of a normal lab mouse is about 2 years.

second or third week of birth due to altered feeding behavior.[86] The researchers noted that, "the reduction of body weight and *food intake* found in DKO (double knock-out) mice…appears a direct outcome of concomitant ablation of DA [dopamine] signaling [in the midbrain] via D1 and D2."[87] As with the study Wise cites, this study also indicates that the action of dopamine (on two types of dopamine receptors) is a necessary condition of motivation to seek survival levels of *food intake*.

Midbrain dopamine seems important to producing motivation for food intake through environmental feedback produced in the reward mechanisms of the brain. This feedback is not only important in recognizing that a behavior *is* rewarding, it is also important in *reward prediction*. Reward prediction is the stimulation of the rewards mechanisms that results from recognition of some potential reward cue or stimulus. Reward prediction is intimately associated with the habitual or repeated process of wanting (or incentive salience) that is associated with a food item, especially in animals. One study found that dopamine neurons in the VTA and SN of monkeys were, "activated when [food] rewards occurred at unpredicted times and were depressed when rewards were omitted at the predicted times. This implies that dopamine neurons code errors in the prediction of both the occurrence and time of rewards."[88]

Wise discusses a study that supports this idea. Mice treated with a dopamine blocker, haloperidol, were fed a food pellet (desirable in control mice) to which they were

[86] Kobayashi, et al, 2004, p.11465. A lack of gastrointestinal motility was also a contributing factor in the deaths of the double knock-out mice. Even force fed mice died. However, the lack of D1 and D2 receptors seemed to generally inhibit feeding behavior.

[87] Ibid, p. 11469, the emphasis is mine.

[88] Hollerman and Schultz, 1998, p. 304. The rewards in this study were juice rewards.

first exposed on the day of treatment (with haloperidol). On the subsequent day, without additional administration of haloperidol, they refused the pellet. Wise believes this suggests that, "the importance of dopamine for the wanting of food on a given day's exposure results from the role dopamine played in the prior liking of food on earlier exposures."[89] Wise's interpretation of these results suggests that dopamine may not play a role in the initial ingestion of the food pellet. However, he suggests that it does play a role in encoding the predictive value of stimuli for the purpose of determining the incentive salience of future potential rewards. Taking these studies collectively, there appears to be good evidence that dopamine is critically important in reward and wanting, especially in establishing an *appetite* for food.

Before continuing, I want to flag the term appetite. The sense in which I use this term here, as something that forms the foundation of the motivation for food reward is the sense in which I use it with respect to addiction. *Appetite* is a habitually reoccurring (occurring repeatedly even after satiation) state of wanting that arises from a dispositional want and motivates an animal or person to seek out a particular reward. This habitually reoccurring state of wanting has the potential to have an *exceptionally strong* salience component, if left un-satiated. To say that a reward has exceptionally strong salience means, in part, that the strength of feedback from cues associated with that reward can show exceptional power in leading to functional motivation. Appetitive wants are disposed to repeated and strong arousal, especially in the presence of cues associated with their satiation. When left un-satiated for long periods of time, (given their strength)

[89] Wise, 2006, p. 1153

appetitive wants are *especially able to overcome motivational inhibition*. Hunger, thirst, and the drive for sex are paradigms for appetites.

By directly comparing the locations (e.g., NAc) and the chemistry (e.g., dopamine) mechanisms of food appetite with that of addictive drugs, we can begin to see the similarity in the mechanisms of reward. By extension these similarities are indicative of a similarity between addiction and food appetite with respect to wanting. Touching on both of these things Wise writes:

> …food reward and food associated stimuli do elevate dopamine levels in the nucleus accumbens. Indeed, just as μ and δ opiate agonists are rewarding in proportion to their ability to elevate dopamine levels, so are different sucrose concentrations rewarding in proportion to their ability to elevate dopamine levels in the nucleus accumbens.[90]

The idea is that the greater the similarity in the functional mechanisms and degree of strength between appetites (for food especially) and substances of addiction the more likely they are to be the same type of thing.

It seems fair to say that the appetitive drive for food reward (i.e., satiation) is dependent on the dopamine circuitry of the midbrain. Experimental findings show that food wanting and drive can be diminished or wiped-out when dopamine is not present in the midbrain circuitry, as with the L-DOPA deficient and double-knockout mice. The strength of wanting associated with cues meant to advertise potential appetite satiation likewise depends on dopamine's presence, as in the case of the haloperidol mice and the variable rewards given to monkeys. It is necessary to draw out further the similarities between the mechanisms (as well as behaviors) of drugs of addiction and those of food

[90] Ibid., p. 1151

appetite and reward. To do this I must examine the impact of the chronic use of certain drugs and engagement in certain behavior on the dopamine rich parts of the midbrain.

§2. A (Brief) Overview of the Basic Neurobiology of Addiction

Various neurotransmitters and parts of the brain play a role in both the acute and long term effects of drug use. The specific set of neurotransmitters that plays a role varies depending on the drug. However, the dopaminergic neural circuitry in the NAc, MFB, SN, and VTA are invariably involved in cases of drug addiction. One study shows a connection between drug addiction and dopamine in the midbrain, and implies a similarity between drug and food reward. Osvaldo Giorgi, et al. studied Roman high avoidance (RHA) and low avoidance rats (RLA), which are selectively bred rat lines. Giorgi et al. note that "compared with their RLA counterparts, RHA rats display a robust sensation/novelty seeking profile, a marked preference and intake of natural or drug rewards, and more pronounced…responses to…acute administration of morphine and psychostimulants."[91] The authors also note that, "in sensitized RHA rats, acute morphine and cocaine cause a larger increment in dopamine output in the core, and an attenuated dopaminergic response in the shell of the nucleus accumbens, as compared with RHA rats repeatedly treated with saline".[92] RLA rats, with a lower concentration of DA D1 receptors, do not show the same sort of sensitization to acute morphine and cocaine exposure. Interestingly, "binding studies indicate that the density of DA D1 receptors in

[91] Giorgi, et al., 2007, p. 148

[92] Ibid., p. 148

the NAc is higher in RHA rats than in their RLA counterparts."[93] Recall that the lack of D1 (and D2) receptors was associated with the inhibition of food intake in the study of double knockout mice. This study is further indication that the neurobiological mechanisms of addiction depend upon the effectiveness of dopamine in the midbrain, and that a lack of abundant dopamine signaling in the midbrain is sufficient to inhibit addiction (as it was with the RLA rats).

To understand the appetitive nature of addiction it is important to examine the role of dopamine in the pharmacology of particular drugs. I will first look at the pharmacology of alcohol. There are a wide range of neurotransmitters and intercellular processes affected by alcohol. But, as noted in *Principles of Addiction Medicine*, the key to understanding reinforcement of the behavior comes from dopamine transmission in the mesotelencephalic area (mesolimbic pathway) of the brain:

> Animal and human studies have suggested that the biochemical substrates for the reinforcing properties of alcohol and other drugs of abuse involve discreet neural pathways in the brain, including the dopaminergic projections to the mesolimbic areas of the forebrain. These neurons originate in the ventral tegmental area, and project to discreet areas of the forebrain, including the nucleus accumbens, olfactory tubercle, frontal cortex…The reinforcing properties…enhance the synaptic concentrations of dopamine in key mesolimbic cortical regions.[94]

The behavioral reinforcement associated with elevated levels of dopamine, and its association with alcohol, have been demonstrated experimentally. In one such experiment an alcohol preferring strain of rats, with operant training to self-administer alcohol,

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[93] Ibid., p. 150

[94] Graham and Schulz, 1998, p. 113. "Mesolimbic" here refers to the midbrain and its role in the limbic system. The mesolimbic pathway of the brain includes the MFB, NAc and VTA and should not be thought of as wholly distinct from the midbrain, although it does include several parts of the brain which I do not discuss.

showed elevated levels of dopamine when compared to a non-alcohol preferring strain of rats even at the same level of total alcohol intake.[95] Another interesting finding was that in the alcohol preferring strain of rats there was a significant elevation of dopamine prior to alcohol self-administration that was not found in the non-alcohol preferring strain. This suggests, "genetic differences in this pathway may contribute to the motivational factors that drive alcohol-seeking behavior in certain individuals."[96] Other studies have shown that the use of dopamine agonists (chemicals which mimic or increase the strength of the effects of dopamine) in rats, "shifted the animal's preference from ethanol to water, especially in those strains of rats that show alcohol preference."[97] This seems to indicate that the motivation of the rats to take in alcohol in the first place was based on alcohol's ability to stimulate dopamine production, and not on some further or additional downstream effect of alcohol.

The specific pharmacology of opiates (e.g. morphine and heroin) is different from alcohol in some respects. Opiates bind directly to certain opiate receptor sites on neurons. Alcohol, while it does increase the level of endogenous opiates, does not bind directly to opiate receptors. However, in the case of opiates there is also a strong "dopamine connection". These drugs produce higher than normal levels of dopamine in both the VTA and the NAc. For instance, "opiates acutely activate VTA dopamine neurons, an

[95] See Weiss, et al., 1992. The study is discussed in Graham and Schultz, p. 113. The rat brain is morphologically the same as a human's brain, consequently rats are used in the vast majority of the studies of addiction. I accept, because scientists seem to, that the rat's brain is an excellent indication of how the human brain would function under similar circumstances.

[96] Graham and Schulz, 1998, p. 113

[97] Ibid. p.111

effect mediated, at least in brain slices, via inhibition of the inhibitory GABAergic inter-neurons."[98] In the NAc, " Morphine at a dose of 1mg/kg, which is rewarding in rats, increased synaptic dopamine concentrations preferentially in the accumbens and also increased dopamine concentrations when tested over a wide range of doses."[99] Studies not only indicate that there are increases in dopamine in the VTA and NAc after the acute administration of opiates, but that long term exposure to opiates alters the morphology of the brain's reward circuitry, particularly the VTA. In Sklair-Tavron et al., the authors' observed a marked reduction in the duration of activity upon stimulation, as well as the size of dopaminergic neurons in the VTA after chronic morphine exposure.[100] The authors note:

> …the chronic morphine-induced decrease in neuronal size was selective for the dopaminergic neurons in the VTA…One possible model of opiate regulation of the VTA is that opiates acutely activate dopamine neurons with an intensity not seen under normal conditions. After chronic exposure, compensatory adaptations occur in the VTA to oppose this activation.[101]

The dopaminergic neurons of the midbrain are also affected by stimulants such as nicotine and cocaine. Nicotine acts on a complex network of neurons and neurotransmitters; however, it too stimulates dopamine release in the VTA and NAc in

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[98] Sklair-Tavron et al., 1996, pp.11205-11206, GABA is gamma-Aminobutyric acid, a major inhibitory neurotransmitter, responsible for dopamine inhibition.

[99] Di Chiara and Imperato, 1988, p.5275. The dose of 1mg/kg, which was administered subcutaneously, is a large dose and would be an overdose for most persons without a tolerance to opiates (it is equivalent to giving a 200 lbs. person a 90 mg does of morphine sulfate -the high end for a subcutaneous doses in a hospital seems to be 30mg over a 4 hour period, although that might vary given body weight, tolerance and other factors). However, doses half that size also increased synaptic dopamine concentrations by 40% up to an hour after administration, so this effect did not only depend on high doses, but was also seen in doses that approximate therapeutic use in humans (see Fig. 4A, p.5276).

[100] See Sklair-Tavron et al., 1996.

[101] Ibid. pp. 11204- 11206

laboratory rats. According to Di Chiara and Imperato," Nicotine, a rewarding drug, at .6 mg/kg increased synaptic dopamine concentrations by ≈100% in the accumbens…and elicited behavioral stimulation characterized by marked rearing, locomotion, and grooming."[102] Like opiates, nicotine inhibits GABAergic neural transmission, which inhibits dopaminergic activity, in the VTA and NAc.[103] Like opiates and alcohol, longer term nicotine use produces more fundamental changes in the neurobiology of the midbrain. Athina Markou notes that," Persistent nicotine use leads to tolerance that is mediated by neuroadaptations occurring in response to chronic nicotine exposure."[104] These adaptations, observes Markou, occur in, "limbic brain sites, such as the VTA, nucleus accumbens, amygdale and frontal cortex which are likely to be critically involved in dependence and the expression of affective signs of nicotine withdrawal upon cessation of drug administration."[105] Nicotine and other drugs of addiction increase dopamine levels during use and can change the neurobiology and morphology of the midbrain.

Cocaine, like nicotine, is a stimulant drug commonly known to produce strong craving, also has a direct connection to increased dopamine levels. According to

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[102] Di Chiara and Imperato, 1988, p.5276. This is a relatively small dose of nicotine. The median lethal does of nicotine for rats, administered orally, is 50 mg per kg.

[103] This is supported by the observation in several studies that the use of GABA agonists seems to block cue-conditioned nicotine use. The excitatory neurotransmitter glutamate also plays an important role in nicotine dependence. Glutamate receptors are found in both the VTA and the NAc and these are the locations of the glutamate receptors involved in nicotine dependence. For a detailed discussion see Markou, 2008.

[104] Markou, 2008, p.3160. One example of such an adaptation is the down regulation (i.e. a decrease in the number) of receptor sites for specific neurotransmitters.

[105] Ibid, p.3166

Principles of Addiction Medicine, "One of cocaine's primary effects is to block the synaptic reuptake of neurotransmitters including dopamine, serotonin, and nor-epinephrine within the medial forebrain bundle."[106] The long-term effects of chronic cocaine use are also,"…associated with a withdrawal syndrome that includes prominent psychiatric features which appear related to dopamine deficiency and possibly also serotonin depletion."[107] Cocaine -like alcohol, opiates, and nicotine- increases dopamine levels in the midbrain, and appears linked to neurobiological changes in that same area.

It is commonly thought that, in addition to drugs, it is possible to become addicted to behaviors such as gambling, sex, and eating. Can behavioral addictions also be conceptualized as disordered appetites? Tentatively the answer is yes. However, behaviors or processes of addiction are less well studied than chemicals of addiction. Even the best studied, and probably most notable, exemplar of behavioral addiction, problem gambling, has not yet been studied sufficiently. Marc Potenza observes that, "few studies have investigated directly a role for dopamine in problem gambling."[108] However, there are experimental indications, in animal behavior and human genetic studies, that problem gambling is associated with higher levels of dopamine in the midbrain. One such study delivered a food reward –a drop of syrup- to monkeys at variable rates. The rate depended on which of three conditioned stimuli (CS) –an image on a computer screen- was presented them. The probabilities of the delivery of the reward were: P = 0, P = .5, and P = 1.0. The timing and order of the presentation of the CS was

[106] Graham and Schultz, 1998, p.141

[107] Ibid., p.142

[108] Potenza, 2008, p.3182

varied. The researchers found, once the meaning of the different CS had been established, that, "the [midbrain] population of dopamine neurons responds to CS onset with a brief increase in activity when P = 1.0….When P = 0, CS onset produced little response…When P = .5 (and uncertainty about reward occurrence is maximal), a slow, steady increase in firing is seen prior to the time of potential reward delivery."[109] While this study does not investigate problem gambling, it is suggestive about the relationship between uncertainty and the stimulation of dopaminergic neurons in the midbrain.

Genetic studies seem to point to a more direct connection between gambling and elevated dopamine levels. One study indicated that problem gamblers showed a greater likelihood to have a variant of a gene associated with the prevalence of dopamine receptors than the general population. Substance abusers were also more likely to have this variant of the gene.[110] Likewise, differences in the midbrain activity of problem gamblers and non-problem gamblers have been observed in fMRI studies. In one study researchers found that problem gamblers showed less activation of the NAc than non-problem gamblers when involved in a gambling simulation in which no money was involved. Perhaps this indicates that real risk, rather than mere procedure, was necessary to stimulate the problem gambler.

Dopamine's role in problem gambling is less certain than for drugs of abuse. However, there are no clear indications, of which I am aware, that the midbrain

[109] Shizgal and Arvanitogiannis, 2003, p. 1857. In this study delivery rates of .25 and .75 were also associated with a unique CS and investigated. However, the authors note that,"…the population firing rate rose steadily throughout…the conditioned stimulus when the probability of reward was .5, attaining a higher rate than when the reward probability was .25 or .75."

[110] The study is discussed by Potenza, 2008.

dopaminergic neurons are not involved in problem gambling; and some intriguing findings indicate that they are involved. Given this -along with the similarity in the behavioral patterns of problem gambling and drug addiction (e.g. continued gambling despite adverse consequences, diminished self-control, urges or craving states prior to gambling)- it seems reasonable, on a weight of the evidence analysis, to *tentatively* identify gambling with drugs of dependence as a disordered appetite.[111] That being said, if it should turn out that the neurobiological mechanisms of problem gambling are different from those of drug addiction, then it might still be possible for my theory to account for cases of problem gambling as addictions. In that case problem gambling could still be included as an addiction under the *disordered passion* arm of the hybrid theory (which I will discuss in chapter 3). It might also turn out that, given enough differences between problem gambling and drugs of dependence that problem gambling turns out to be a related but distinct disorder. However, such findings would be a significant departure from the behaviors and mechanisms of problem gambling as it is currently understood.

I concede that there are limitations to what I have covered and what I have been able to show. In this section I touched on only a small number of the neurobiological mechanisms involved in addiction. Further, I covered only a few of the myriad substances and behaviors involved in addiction. However, the drugs I chose were intended to be representative and diverse enough in type and effect to show the strength of the claim that dopamine and the midbrain are central features of addiction to drugs. I

[111] Ibid., p. 3183 indicates that these behaviors are core components of drug addictions as well as problem gambling.

have attempted to provide enough data so that the reader understands some of the biochemical or morphological effects that the chronic use of drugs has on the midbrain and its dopaminergic connections.

In addition to this I have tried to show the important similarity between the acute action of drugs of abuse and the effect of food on the reward system of the brain. It appears to me that there are significant similarities between the neurobiological mechanisms and locations of appetite for food and the want for drugs of abuse. The behavioral effects of the two are also similar (i.e. both produce strong motivation and habitual behavior). I recognize that I have not established definitively that the drugs of abuse, when used chronically, are a species of appetite. It is possible that there is an unrecognized and deep structure to an appetite, like that for food, which is not present in addiction, or vice versa. However, there seem to be a number of provocative indications that they are the same type of thing.

At this point in the argument addiction remains partially behind a veil. Appetite itself is sitting in plain sight. If dopamine was the only similarity between appetite and addiction that would be weak grounds to conclude that one is a species of the other. When one adds similarities in the brain systems and associated behaviors the case becomes stronger. With the present state of knowledge about addiction its biochemical basis is not fully determined. However, there is some good information about its principle material composition, basic configuration, and function. That information may not be sufficient to make a definitive determination about how to categorize addiction, but it goes a long way in grounding claims about its proper category location.

§3. The Incentive Salience Theory of Addiction

The incentive salience theory holds that addiction can be understood as a problem of the oversensitivity of an organism to a particular type of reward. It explores the specific role of salience (i.e. strength of wanting and associated motivation) in addiction and posits that this salience is disordered. It also makes an important distinction between wanting and liking, and offers an insightful connection between incentive salience and relapse. The incentive salience theory maps nicely onto the picture I have painted about the similarity of appetite and addiction. That being said the theory does not give a satisfactory explanation of exactly what is disordered (or pathological) with the incentive salience involved in addiction. Nor does it attempt to explain any motivation for addictive behaviors beyond neurobiological salience, such as higher-order cognitive or value functions. However, it complements my own theory and reinforces what I think is the correct biological view of addiction.

The incentive salience theory was formulated by Terry E. Robinson and Kent C. Berridge, both bio-psychologists at the University of Michigan. According to them:

> The central thesis of the incentive salience theory of addiction is that repeated exposure to potentially addictive drugs can….persistently change brain cells and circuits that normally regulate the attribution of incentive salience to stimuli, a psychological process involved in motivated behavior. The nature of these 'neuroadaptations' is to render the brain circuits hypersensitive in a way that results in pathological levels of incentive salience…to drugs and drug associated cues.[112]

The incentive salience theory emphasizes the important role of dopamine in drug addiction. Berridge and Robinson write, "Mesotelencephalic dopamine systems show

[112] Robinson and Berridge, 2008, p. 3137

robust sensitization after repeated exposure to drugs such as amphetamine, cocaine, or heroin."[113] In addition the authors recognize a close connection between dopamine sensitization and appetitive behavior. They note that the type of incentive salience associated with drug addiction, "can also sometimes spill over in animals or humans to other targets, such as food, sex, gambling, etc."[114]

There are virtues to this account worth noting. First, incentive salience constitutes the pressure on addicts to behave in a way that is both contrary to and independent of their considered judgment or expressed desire. This element of the theory is especially interesting because it provides a more comprehensive answer to the 'conflicted-self' problem discussed by Ainslie. While hyperbolic delay-discounting could explain preference reversal over time, it did not explain how an addict might be simultaneously divided against herself. In hyperbolic discounting preferences at specific times were uncontested, it was only cross-temporal conflict that account for the conflict in an addict. In my discussion in chapter 1 I suggested that this picture was too restrictive as it ignored simultaneous conflicting desires within the addict. The incentive salience theory explains this by positing two different mechanisms at work in driving and resisting drug use: wanting and higher cognitive mechanisms. Since they can operate independently with respect to the same reward they can be in conflict.

A second virtue of the theory is that it can (partly) account for the hazy boundaries of addiction and explain the phenomenon of relapse. Like George

[113] Berridge and Robinson, 1995, p.72

[114] Robinson and Berridge, 2008, p. 3138

Loewenstein, Berridge and Robinson believe that cue-conditioned craving plays an important a role in relapse. However, they provide a more robust model for the source of cue-conditioned craving. Traditional psychological theories emphasize stimulus-response or aberrant learning mechanisms as being at the root of cue-conditioned relapse. Berridge and Robinson believe that to *merely reduce* the source of cue-conditioning to these mechanisms (stimulus-response or aberrant learning) would be to fail to significantly distinguish addiction from an established habit, like tying one's shoes or brushing one's teeth in the morning. They point out, correctly I believe, that no matter how many times habits like these are repeated they cannot be "stamped into" the brain the way addiction can. Habits like tying one's shoes and brushing one's teeth are routinely done automatically and without reflection, but upon reflection are easy to control (assuming that, some morning, there is a reason not to brush one's teeth). Addiction is not only a generally stronger motivational force than these habits it is also a far more complex process involving consciousness of behavior and an awareness of ends. This is not to deny that aberrant learning or stimulus-response play a role in addiction or relapse, but it is to deny that they play a primary role in addiction or relapse (at least in humans).

This is the source of the distinction between incentive salience and stimulus-response or aberrant learning models of cue-conditioned *relapse*. Berridge and Robinson write, "repeated exposure to potentially addictive drugs can, in a way that is not reducible to learning, persistently change brain cells and circuits."[115] They continue," The nature of these adaptations is to render these brain circuits hypersensitive in a way that…makes

[115] Berridge and Robinson, 2011, p. 31

pathological incentive motivation for drugs last for years, even after the discontinuation of drug use."[116] This is an important observation because it helps show how a disordered appetite can explain relapse separate from learning or stimulus-response.[117] Thus it provides disordered appetite with more of an explanatory purchase on one of the phenomenon associated with addiction.

The incentive salience model makes another contribution to the discussion. It distinguishes liking from wanting. This distinction cannot be thought of in purely phenomenological terms. "Liking", according to Berrridge and Robinson, is a conscious process, but "wanting", in the sense they use it, does not necessarily involve conscious awareness. This distinction is important to them for two reasons. First, they use the distinction to explain findings in which addicts report not consciously wanting a drug at all, while continuing to pursue it. Second, they use it to account for why an addict continues to use despite expressing dislike for a drug. I will discuss this distinction and internal division further in chapter 3.

One shortcoming of Berridge and Robinson's model is that they fail to clearly define the pathological nature of incentive salience as it relates to addiction. They are not alone in this failure. It is difficult to find, in the literature on addiction, a clear explanation of why a process associated with addiction is pathological. The meaning of the modifier "pathological" is generally left vague. Yet calling cancer a pathological

[116] Ibid., pp. 31-32

[117] I read them as claiming that there is something significant apart from just learning that is at work in addiction. I do not think they mean to deny the role of learning completely. To do that would be absurd since cues are external and clearly must be learned. What they mean is that the strength of the salience of an addictive drug is not constituted by learning or stimulus-response alone.

tissue growth hardly gives an illuminating explanation of the pathological nature of cancer. It is the fact that cancer is uncontrolled growth of one's own cells which fail to continue their proper physiological function –and interfere with the function of other tissues- that constitutes the pathological nature of cancer. For reasons that parallel this example I am generally unsatisfied with the conceptual failure to account for the "pathological" nature of addiction and associated brain processes.

Disordered appetite provides a partial model of addiction. The similarity of the reward mechanisms at work with drugs of addiction and those associated with food intake suggest that addiction is an appetite. Appetites can be conceptualized as a reoccurring state of strong want, arising from a disposition to want, for a particular type of reward. I believe that the disordered appetite element of my theory takes the same basic approach to explaining the neurobiology of addiction as Berridge and Robinson.

§4. What Makes an Appetite Disordered?

One of the least explored areas in addiction literature is an explanation of what precisely constitutes the disordering involved in addiction. There is a range of complexity in biological systems, human behavior, and specifically in addictive behavior that makes this difficult. However, in what follows I hope to make significant headway into what makes an addictive appetite disordered.

There are two principal characteristics of a generic disordered appetite. First, a disordered appetite does not exist within strength and satiety parameters that are normal and appropriate to an organism's function. Second, a disordered appetitive can cause

dysfunction of the organism's other appetites. When discussing a disordered appetite of the *addictive* variety (henceforth addictive disordered appetite) both of these characteristics are relevant. Before going further it will be helpful to consider an experiment discussed by Gardner and David:

> A laboratory rat has been surgically implanted with a very small (1mm diameter) electrode device in the middle of the pleasure/reward circuitry in its brain. The rat is placed daily one hour in a test chamber containing a wall mounted lever…the output of which is fed…to the pleasure/reward circuitry. The rat rapidly learns that pushing the wall-mounted-lever delivers…electrical stimulation (just strong enough to activate the brain cells immediately surrounding the electrode tip)…During these test sessions the animal is…lever-pressing at maximum speed and completely ignoring other attractions within the test chamber (food, water, playthings, sexually receptive rats of the opposite sex)…After several weeks the rat faces a new and unexpected behavioral contingency. An electrified metal floor grid has been placed in the test chamber…After some minutes it crosses the floor grid, receiving intensely painful footshock…to reach the lever and once again self-administer the pleasurable brain stimulation.[118]

Gardner and David also discuss an experiment in which cocaine addicted rats starve themselves to death, even when offered food, in favor of continuing to lever press for injections of cocaine.[119] Results that show such extremities of behavior in laboratory mice and rats are common in addiction research, and it is worth keeping these examples in mind. They illustrate that addiction is hostile to the normal functioning of organisms and their appetites.[120]

The first step in understanding an addictive disordered appetite is distinguishing it from generic disordered appetites (i.e. disordered appetites in general) and from cases of mere physical dependence on a drug. This can be done by using two criteria. First,

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[118] Gardner and David, 1999, p. 94

[119] Ibid. p. 96

[120] I have presented reasons to think that addiction is an appetite (and probably a disordered one) in sections 2 and 3. These examples are meant to emphasize the *disordered* nature of addiction with a secondary emphasis on it being an appetite.

addictive appetites are *acquired* through either learned behavior or conditioning reinforced by reward. Second, they are *targeted*. The first criteria may imply the presence of the second, but they are conceptually distinct. If either of the criteria does not apply to a disordered appetite, then it is not an *addictive* disordered appetite.

The acquisition criterion is important because it implies that some conscious behavior or a process of which one was aware, are necessary for an addictive disordered appetite. The acquisition criterion distinguishes addiction from certain cases of mania or obsession by requiring that addiction result from a history of reward mechanism activation associated with a particular drug or behavior. This association must predate the disordering of the appetite. This could also be thought of as a criterion for the etiology of addiction. It is meant to distinguish addiction from cases where traumatic brain injury, hormonal disorders, or latent congenital disorders causes pathological behavior that might resemble addiction (e.g. a hormonal disorder –which is congenital or largely genetic- that causes a disordered appetite for food). Addiction should not be thought of as a disorder which arises from a single event or purely from genetic factors. It must come from a *series* of behaviors or events which reinforce themselves. This means that someone who awakes one morning with an overwhelming and persisting desire to drink (with little history of drinking) should not be thought of as an addict. Some different and more puzzling disorder is occurring in such a case.

Addiction certainly has genetic roots that dispose an individual to find a particular drug or behavior especially rewarding. However, in the case of addiction the drive to take a particular drug must come from a pattern of reinforcement through activation of reward

mechanisms. The acquisition criterion makes this distinction: Addiction is the product of a partnership between genetics and conscious activity whereas some manic or compulsive behavior may be more a product of genetics or some activity of which one is unaware.

The second criterion, targeting, is important because it eliminates unusual cases of physical dependence on drugs from being examples of addiction. Suppose an infant exposed to an addictive drug develops a physical dependence for the drug to which she has been exposed. Perhaps an elderly patient in a hospital on a morphine drip, without knowing what they are on, could develop a physical dependence on morphine. Let us assume, in both cases, the reward mechanisms of the brain function indistinguishably from those of a typical addict. Once drug administration is discontinued, withdrawal in each case can be assumed to be physically similar to that experienced by an addict. But, in neither of these cases, is *craving* for *the drug* experienced. In neither instance does the person have a target for their appetite. This is an important criterion because it highlights the important role targeting plays in the concept of *craving* and its relationship to addiction. If someone does not crave morphine, alcohol, or cocaine, she would not be addicted to them. Desiring release from withdrawal symptoms brought on by unwitting (and unrealized) exposure to drugs is different from craving the drug because of repeated administration. The former does not engender behavior that would perpetuate physical dependence, whereas the latter does. This –correctly in my judgment- eliminates babies born with chemical dependence from being thought of as addicted. They are not in fact addicted and it is perhaps better to think of them as suffering from something more akin to exposure sickness (cf. radiation).

These two criteria are necessary conditions of an *addictive* disordered appetite. But they do not explain anything about the *disordered* nature of an addictive appetite. An appetite may become disordered for several reasons. I identify three different explanations of disordered appetites. Different addictive drugs may disorder the appetite in different ways, and I believe that any one of these explanations could be sufficient (by itself) to constitute an addictive disordered appetite, however, often one will find these disorders co-instantiated in the same addict.

4.1. Long-Term, Marked Interference with Core Appetites

Ordered appetites interfere with one another in the normal course of events. Hydration requirements might trump or overshadow the need for sustenance. Acquired directed appetites that extinguish or excessively interfere with the motivational efficacy of certain core appetites, such as those that are evolutionarily primitive (e.g. the drive to procreate in those with procreative viability), or those that are important to the well-being of an individual organism's health (such as adequate food or water intake), are disordered.

The earlier example from Gardner and David illustrates this idea, even if it does not give it a precise explanation. Recall that the mice lost the motivation to pursue food or sex when given an opportunity to lever press. About the electrical stimulation from the lever pressing Gardener and David write, "Hungry animals ignore food to get it; thirsty animals ignore water to get it. Male animals ignore sexually receptive females to get it."[121] The example shows the extent to which one appetite (in this case lever pressing for

[121] Gardner and David, 1999, p. 95

reward) can significantly diminish (or even extinguish) the motivational efficacy of core appetites. Perhaps the reward value (in terms of raw strength) of feeding or sex itself was diminished; whatever the case may be, it is clear that the reward was no longer sufficient to functionally motivate under conditions which one would expect it to functionally motivate. The appetite for lever pressing was disordered because it interfered –in an excessive and harmful way- with the functional motivational abilities of these core appetites.

In persons, who have rational inhibitory mechanisms, this interference is not so straight forward and easy. Earlier I discussed the potential conflict between rationality, willpower, and the salient wants of addiction. A person, unlike a rat, is perfectly capable of recognizing that they are not eating properly and are malnourished. Yet, things like vitamin deficiencies (especially niacin) are common in alcoholics and other addicts. At the primitive level, even in persons, the addictive want is often more salient than the appetitive non-addictive want. Why rational mechanisms are insufficient to both recognize and prevent acting upon this disordered preference is difficult to say. Sometimes rational mechanisms and willpower are enough. But it is terribly (and empirically) obvious that rational mechanisms are often not enough.

So, what is it precisely that makes an appetite disordered in its interference with the functional motivation of core appetites? A disordered appetite need not extinguish a core appetite. For an appetite to be disordered it need only suppress or interfere with the normal functional motivation of some core appetite in a significant and sustained way. It seems reasonable to maintain that a core appetite, like that for food, has strength of

reward associated with it such that (especially when it has gone un-satiated) given sufficient strength of want with respect to that appetite, the perceived reward will routinely be strong enough to produce functional motivation. In general, this sort of relationship between a core appetite and motivation is necessary to health and survival (either individual or species). As I noted earlier, there may be times when a rewarding food item is passed over, despite hunger, because of dehydration. Despite some 'local' variations, over the long term core appetites should be rewarding enough to motivate behavior that produces pursuit of the core appetite in healthy (certainly within individual survival) ranges. If the reward strength of that appetite, or the prevalence of the functional motivation associated with the reward mechanisms, diminishes for a long period such that these behaviors no longer occur within a healthy range, then they are themselves disordered. If this diminishment (in the core appetite) has occurred because an acquired, targeted appetite has caused it, then the acquired, targeted appetite is an addiction.

There are at least two possible mechanisms for this disordering of the appetite. First, the addictive disordered appetite might be causing the reward feedback from the core appetite to diminish in strength. It may be that a slice of cheesecake no longer offers the same promise of reward when one becomes conditioned to the rewarding strength of heroin, so that the reward potential of cheesecake is just diminished in general. It might also be the case that the need to pursue a particular reward to which one is addicted interferes with the ability of a core appetite to functionally motivate behavior –translate the activity of the reward mechanisms into motivation strong enough to produce

behavior. On this analysis cheesecake might seem as rewarding as ever, but given the choice between it and heroin the cheesecake is just going to lose. I favor the former explanation. Whichever is correct, or if there is another mechanism, an addictive disordered appetite can be thought of as one which undercuts, for a sustained period, the ability of the rewards associated with core appetites to produce functional motivation within healthy ranges.

The possibility of this is suggested by some of the scientific data discussed in sections 1 and 2. Remember that diminished activity of dopamine (or the suppression of certain dopamine receptors) inhibits feeding behavior in mice. Recall also that drugs increase dopamine levels (and dopamine receptor numbers). If drugs of addiction create an environment in which the midbrain (or other parts of the brain implicated in appetite) becomes accustomed to greater amounts of dopamine (and greater numbers of dopamine receptors), then it seems reasonable to think that the *normal* dopamine levels produced as feedback to normal core appetitive behavior (e.g. feeding) produce *relatively* diminished feedback in the reward mechanisms of the brain (think of this as the functional equivalent of a normal person having some dopamine receptors blocked, or as producing less than normal levels of dopamine). On this analysis the amount of dopamine (or the number of dopamine receptors) required to produce functional motivation is ratcheted up within the brain by the drug. When this change takes place, especially when it is artificial, levels of dopamine that once made core appetitive rewards appealing no longer do so.

4.2. Reduction in the Desirability and Efficacy of Non-Appetitive Rewards

Ordered appetites, at times, interfere with our conscious likes and non-appetitive rewards. Listening to music, playing games, or watching movies are examples of non-appetitive reward (one might develop an appetite for these things, but within normal ranges they do not fall within the category of appetite). These rewards are non-appetitive because the wants they produce lack the habitually reoccurring nature of appetites or they lack the exceptionally strong salience component of appetitive rewards that have gone un-satiated. As an example, I may only go to the movies once in a while without any habitual reoccurrence of wanting to go to the movies (e.g., I only go in the event that something looks appealing, without any discernible pattern to my attendance.). Perhaps I do have a habitually reoccurring want (say every weekend) to go to the movies. But in such a case, if I can forego going to the movies on a particular weekend and without feeling an exceptionally salient want to go to the movies, then going to the movies is not an appetitive reward for me (i.e., in this case it is a habit, but I can take it or leave it as I choose with relative indifference). Appetites, like that for food, can easily direct our attention away from these non-appetitive rewards. Of course this is not indicative of a disordered appetite. In fact it is a well ordered appetite for food that gets us to stop watching a movie and redirects our attention to something more important.

However, as with the interference of one appetite with core appetites, there are cases where the interference of an appetite with non-appetitive rewards can be disordered. An appetite that is the cause of a sustained reduction in the perceived strength (i.e. sensitivity to) and motivational efficacy of non-appetitive rewards is disordered. I

theorize that a disordered appetite will make the non-appetitive reward less rewarding by causing a sustained reduction in the strength or worth of the potential reward. That is, the sensitization of the brain and its reward circuitry to an addictive appetite might be matched by desensitization to a significant number of non-appetitive rewards. Perhaps it is because the disordered appetite has conditioned the brain to strength of reward that dwarfs the non-appetitive reward so that the latter becomes inefficacious. The point is that there should be 'elbow room' for non-appetitive rewards to flourish, or at least be maintained. But in the case of disordered appetites there may be no or restricted 'elbow room' for non-appetitive rewards.

There is an interesting study that supports this idea. Pearl Chiu and her colleagues at the Baylor School of Medicine performed an experiment involving an investment task with smokers (under satiated and non-satiated conditions) and non-smokers. Each subject in the two groups was given $100 to invest in a series of investment tasks that involved choosing allocation amounts for a number of stock market segments. After each investment sequence the subject was shown the return (or loss) on their investments, the percentage increase (or decrease) in their investments, and investment history.[122]

The purpose of the experiment was to see how smokers as opposed to non-smokers adjusted their bets in response to predictive or fictive errors. As an example: a predictive or fictive error would occur when a subject (using historical market data as their initial guide) invests 30% of available funds in a particular security and 10% in another when the first shows a subsequent 5% loss, while the second returns a 25%

[122] Chiu, et al, 2008, p. 517. Historical data from the stock market was used and investors were given a range of individual securities in which to invest.

profit. In this case there is a fictive error because the subject risked a greater percentage of their available funds in the investment that returned a loss, while they under-invested in an investment that returned a gain. In the experiment, subjects were expected to recognize their fictive errors and adjust their investments according to the degree of the error in subsequent rounds of investing.[123] This is what Chiu, et al, found that the non-smokers did, while smokers, despite registering the fictive error, did not recalibrate their investments to the extent that nonsmokers did. Neither satiation, nor a lack of satiation made a difference. Chiu, et al, write:

> We found that chronic smokers, relative to non-smokers, showed a reduced influence of abstractly framed learning signals on behavior without any accompanying loss of the associated neural signal. That is, although fictive error was indeed computed in this group (as indicated by the robust neural response in bilateral caudate), 'what might have been' did not emerge as a control signal that guides behavioral choice…error signals derived from 'what might happen' remain intact in addicts, but, as propounded by clinical criteria of addiction, their influence on decision making is absent. [124]

By failing to adjust behavior (to the same degree as nonsmokers) chronic smokers showed motivational insensitivity to the maximization of non-appetitive rewards (and the avoidance of loss). The data from this study is minimally consistent with, and I think suggestive of, the idea that addictive disordered appetites dampen or eliminate the motivational efficacy of non-appetitive rewards by making them less effectual or ineffectual qua a reward. It is unclear how general a conclusion can be drawn from the work of Chui, et al, with respect to smokers and motivation to act on non-appetitive reward recognition, but the study would suggest that the problem might be a broad one.

[123] Ibid, see p. 518 for a complete description of the task.

[124] Ibid, p. 517

It is common knowledge (so common that it is part of the diagnostic criteria for addiction) that addicts behave in a way that implies they are unmindful of alternative (to their addiction) rewards (they also seem to be unmindful of punishments). The delay-discounting accounts from chapter 1 make accounting for this one of their primary purposes. Chiu's study is consistent with these theories, that although smokers *recognize* what they are making predictive errors, they are less sensitive to that loss as evinced by behavior. However, Chui's study was not designed to test whether temporal disparity between two different types of rewards caused switches in preference. Rather, the conclusion that the authors of the study come to is that, "higher-order control signals modulating the influence of fictive error outcomes on behavioral choice may be impaired…and uninfluenced by fictive learning signals."[125] So while consistent with delay discounting, the study seems to pick out a more general disposition on the part of addicts. Namely, that the motivational force necessary to translate cognitive recognition of some state of affairs into recognition of that state of affairs as a lost or potential reward (worth caring about) is inhibited, without explaining exactly why.

My analysis is also consistent with the Chui study and has the general advantage of not placing the entire explanatory force of the results on a preference for temporally nearer goods. On my analysis it is *not only* that the drink is temporally near and desirable, but that the strength and level of reward the brain has become conditioned to makes a wide range of alternatives less appealing –simpliciter- without any appeal to th complication of delay-discounting. This might provide an answer to the puzzle about an

[125] Ibid, p. 518

addict's anguish over quitting. Why do they repeatedly express a desire to quit, but yet do not? It could be because by quitting the addict gives up the only alternative available that holds out the promise of reward. There is consistency between this and anhedonia –or the inability to find experiences pleasurable- that addicts experience even after physical withdrawal.[126]

My point is not that a disordered appetite globally reduces all non-appetitive rewards to matters of indifference. Nor is this sort of desensitization a necessary condition of addiction. But I believe that in many, probably most, instances of addiction one will find the addict less motivated by and interested in non-appetitive rewards, because the strength of addictive reward has desensitized their reward mechanisms to respond to non-appetitive rewards qua rewards.

4.3. Disorder in the Quantity-Duration Relationship of Appetite

One of the trickiest things in explaining the phenomenon associated with addiction is addressing the notion of the 'functional addict'. A functional addict is one who can use large quantities of an addictive substance (for example), acquire a robust appetite for it (i.e., become physically dependent on it), while still continuing to live an apparently successful personal and professional life.[127] Winston Churchill is a famous example of a functional alcoholic (among historians it is controversial whether Churchill

[126] I think this account of disordered appetite should be especially appealing to proponents of Herrnstein and Prelec's melioration theory. Their idea is that withdrawal makes all other alternatives return a low rate of utility. My suggestion is more robust because it does not require withdrawal for this to be true, only a general reduction in the appeal of non-appetitve rewards which can occur even after withdrawal ceases via alteration of the brain's reward mechanisms.

[127] It is said that someone with a physical dependence on heroin can function quite capably for years taking only maintenance (non-intoxicating) doses of heroin. However, I do not take this to mean that they are no longer considered addicts. For some discussion of this see Watson, 1999b and Seeburger, 1996.

was an alcoholic, although it is widely recognized that he was a daily drinker who did imbibe heavily on occasions). Among Churchill's accomplishments: he served as a soldier on three continents, was Chancellor of the Exchequer, Lord of the Admiralty, twice prime minister of Great Britain, and a Nobel laureate in literature.[128] Let us suppose that Churchill was physically dependent on alcohol and consumed large quantities frequently (perhaps good genes prevented extreme intoxication and hangovers). It seems to me that such a person would be an addict. How do you capture this sort of addiction within the framework of disordered appetite? Assuming that a highly functional addict retains an ordered relationship between his appetite for an addictive reward and other rewards (both appetitive and non-appetitive) it can be difficult to specify in what the disorder consists. However, I believe that a disordered appetite need not be based on the relation of one appetite to another (or to a non-appetitive reward). A disordered appetite can also consist in a disorder in the structure of the appetite itself.

This structural disordering (or malformation) of the appetite reflects the tolerance, habituation, and strength of appetite seen in addiction –even in those who are highly functional. The idea explored in this section can also serve as a general account of a disordered appetite since it also explains instances of appetitive aversion (e.g. anorexia). The model of structural disorder I propose is centered on the quantity-duration relationship that exists for appetites.[129]

[128] His health was also apparently unaffected, as he lived to be older than 90, dying in 1965.

[129] I am coining the phrase "quantity-duration relationship", but the idea is fairly basic and I would not be surprised to find that it has been introduced elsewhere. I am simply unaware of any other discussion.

For an ordered appetite there is a general or typical quantity of the rewarding object (e.g. food) of the appetite that an individual will find satiating. In addition, there is a general or typical period of time over which satiation will be maintained (given an initially satiating level of consumption). By placing the amount consumed over the period of satiation we get what I call the *satiation ratio*. This ratio, when it reflects an ordered appetite, is not a fixed number but rather a range. As an example, the amount of food one requires to feel satiated, and the length of satiation it provides, might fluctuate slightly given one's level of activity during the day. This ratio is not necessarily stable over the long term and it might increase, or decrease, over long periods of time. For instance teenagers begin eating larger quantities, and more frequently, in order to fuel pubescent growth, whereas the elderly, as they become less active, might tend to eat less and remain satiated for as long as they did when they were younger.

In isolation the satiation ratio cannot be used to determine an appetitive disorder. But compared to normal satiation ratios ranges for the same reward (when compared to demographically similar members of the same culture) this ratio *might* be indicative of a structural abnormality in the appetite. Over a long period of time large quantities consumed with decreased or tenuous stability in the duration of satiation provided indicates a structural abnormality. I am *not* claiming that this can be used to diagnose addiction. Rather, I claim that one can *model* the structural disordering of the appetite using the concept of a satiation ration (e.g. increased levels of consumption associated with the shortened or unstable satiation periods of the addict).

How does this work? Take an easy case for which we probably have a wealth of information about consumption levels: appetite for food. Here it would be easy to gather population information as a basis for comparison -like a range of average quantities of food consumed for various groups which are separated based upon factors which contribute to food consumption (e.g. age, gender, activity level, height, etc.). From this one can generate theoretical ordered consumption (TC) and theoretical satiation time (TST) ranges for ordered food appetite in an individual. *Theoretical order consumption* is a population based construct. It is meant to reflect the range of consumption that one would see in a population of consumers (perhaps restricted by certain criteria such as age, gender, or race) and which does not, in the population as a whole, lead to inordinate levels of disease, societal, or environmental problems as a result of consumption. This theoretical construct does not apply exclusively to appetites, although it can. For instance theoretical order consumption of food would be amounts that do not lead to high rate of diabetes or heart disease in given population. As a non-appetitive example, theoretical order consumption of fossil fuels would put a minimal strain on the environment. However, when paired with satiation length the resulting satiation ratio applies exclusively to appetite, since it does not make sense to talk about the satiation a tank of gas provides.

Theoretical satiation ratio data can be compared against the consumption ranges (CR) and satiation time ranges (ST) of an individual. The comparison works like this: ((CR/TC) / (ST/TST)). A normal ratio is 1/1. An exceptionally high or low ratio is indicative of a disordered appetite. For instance, an individual who consumes twice as

much food as other members of 'his group' to experience the same length of satiation would have a 2/1 ratio (2), and might be addicted to food (depending on acquisition and targeting criteria). A very low ratio is also indicative of a disordered appetite, but of a non-addictive type. Someone who consumes a quarter as much food as the comparison group to experience the same period of satiation would have a .25/1 ratio (1/4).[130]

The assumption is that, when one is compared to a peer group with similar biological attributes, an ordered appetite will be roughly similar to those other individuals. Certain appetites (e.g. food) are like hands, hearts, or lungs. Their proper functioning occurs within ranges and they are not terribly idiosyncratic in their function unless pathological or disordered. The heart of a 24 year-old, with a given set of characteristics, should function within a range expected of an average 24 year old with those same characteristics. So too should the amounts consumed and the length of satiation should be similar among similar individuals.

Disorders or pathologies impacting the appetite, like anorexia or tapeworm, would show disordering of the appetite by showing consumption and satiation ratios that are exceptionally low or high.[131] However, *there is no 'natural' appetite for nicotine, alcohol, heroin, or gambling* (and so no comparative basis for the disorder). There is also no sense that a low ratio of consumption to satiation indicates that one has an appetitive deficiency with respect to gambling (an absurdity).

[130] On a personal note this was precisely what happened to my grandmother during chemotherapy. She found food unappealing, her lack of appetite and subsequent weight loss were a concern to both her family and her doctors.

[131] Someone with a tapeworm might appear to have a food addiction. However, there is an etiological component to my analysis of addiction that would exclude conditions like this.

However, acquired appetites cannot have a disorder of deficiency. *I am making an assumption that should be pointed out:* acquired appetites for nicotine, alcohol, even heroin are not necessarily addictive or disordered. I believe that one could have an appetite for any of these things that is not disordered. The structural disordering of the appetite in the case of addiction is the high satiation ratio of the addictive disordered appetite compared to the *theoretical range* of a well ordered appetite of the non-addict for the same thing (e.g. alcohol). The exact criteria of a theoretical well ordered appetite of this sort of thing (i.e. an acquired appetite for a potentially addictive substance) are hard to set, but it seems plausible that if the appetite can become disordered it can have an ordered precursor. Perhaps the theoretical consumption and satiation time ranges for a population with an ordered appetite for alcohol or heroin could be whatever amounts do not cause significant health, personal, or professional problems in the population. Criteria for disorders are when such consumption levels do reach ranges where they cause disorder. Of course, my whole point is that one can have a disordered appetite in the individual without these problems, but to determine disordered ranges it might be necessary to see them present in the population to which the individual is being compared.

Whatever the standard for the theoretical comparison range it is the comparatively greater consumption on the part of an individual, along with reduction or stagnation in satiation length, which makes one appetite structurally disordered.[132] The

[132] I think it is worth emphasizing that I am referring to an *appetite*. If someone consumes a large quantity of a particular thing without having an appetite for it then this structural disordering does not apply to them. I mean for this to be a comparison of *appetites*.

structural problem consists in consistently high consumption levels within an extended timeframe that are significantly greater than levels of consumption within the same timeframe that would be considered ordered.

This *modified satiation ratio* (individual appetite compared to an ordered paradigm) applies to appetite generally. Of course an appetite could grow and become disordered without being acquired. A congenital or glandular problem might cause a disordered appetite for food, but in such cases the disordering of the appetite would be the result of innate pathological forces that *do not* require acquisition as the basis for the disordered appetite. Such disordered appetites would not be cases of addictive disordered appetite, since addiction requires acquisition.

I believe disordered appetite can be explained in at least three ways. An appetite is disordered because of its inhibitory effects on core appetites, non-appetitive rewards, or because structurally it no longer bears similarity to what is taken to be an ordered appetite for the same reward. Having a disordered appetite, which meets the criteria of acquiring and targeting I set out, *is sufficient* for active addiction.[133] It is less clear whether having a disordered appetite is a necessary condition of addiction.

At any rate, disordered appetite is *only part of the story*. Addiction, in persons at least, can be something other than just the disordering of the reward mechanisms of appetite. Addiction can also result from a disorder of value, perceived importance, and world view associated with a particular object of desire. This can be manifested in the phenomenology of the addict in a way that is distinct from appetite. Addiction as a

[133] By active addiction I mean an addiction which is being expressed or reinforced through ongoing behavior.

concept is also constituted by disordered passion. It is to element of my theory that I turn in the next chapter.

Chapter 3

Addiction as a Disordered Passion

The questions surrounding addiction cannot be exhaustively answered by neurobiological accounts. An explanation that comes closer to being complete must at some point consider the phenomenology of addiction in a substantial way. Of course my intention of providing the groundwork for a comprehensive theory of addiction will demand integration of these two concepts. But for the moment put aside the question of how disordered appetite (discussed in chapter 2) will fit in with disordered passion. We must take one thing at a time. My purpose in chapter 4 will be to show how these fit together, for now I will only concern myself with the phenomenology of addiction.

Addiction is a sustained behavior associated with a welter of attitudes and emotions. Feelings of loss and desperation, love and hate, interspersed with moments of incredible relief and pleasure are often recursive experiences for a single addict during the duration of an addiction. Any explanation of addiction that ignores this range of experience does so at a great cost to its explanatory power. As important as it is to understand the phenomenology of addiction it is, in a way, far more difficult to address than the neurobiology. The sheer range of idiosyncrasies and experiences alone is enough to make the task daunting, to say nothing of the inherent difficulty in fully appreciating the inner experience of another.[134]

[134] In "What is it Like to be an Addict?" Owen Flanagan suggests that a comprehensive phenomenology of addiction might involve detailing a separate phenomenology for every type of substance of addiction. That is to say the phenomenology of the alcoholic is different from that of the heroin addict, which is different from that of the methamphetamine addict, and so on. I do not disagree with him, but I believe there are also

These difficulties mean that my own exploration of the phenomenology of addiction will be somewhat limited in scope. However, the phenomenological elements of addiction that I do address are sufficient to show that phenomenology plays an important role in explaining addiction. In this chapter I focus on the emotional relationships and attitudes certain addicts have developed with the thing to which they are (or were) addicted. I think that this examination will indicate that (in some cases) the addict's emotional relationship with their substance or process of addiction is a manifestation of a disordered passion.

In what follows I am concerned with accomplishing three tasks. First, I consider first-personal accounts of addiction related by addicts, of both genders, in memoirs and papers.[135] These accounts cover addictions to heroin, cocaine, prescription medication, and alcohol. Next, I explain why it is appropriate to analyze these accounts as exemplifying passion. Finally, I explain the specific mechanisms of an addictive disordered passion.

§1. Three Distinct Aspects of the Phenomenology of Addiction

1.1 Self-Satisfaction, Meaning, and Identity

There are at least three aspects of the phenomenology associated with addiction that are important to understanding why addiction can be understood as a passion (explaining why it is disordered will occur later in the chapter). The first of these is grounded in an addict's experience of dependence on an addiction as critical to happiness

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prevalent commonalities in the phenomenology between addicts as well from which helpful conclusions can be drawn.

[135] For the memoirs and first personal accounts I use see: Flanagan, 2012; Knapp, 1996, Stahl, 1995; and Styron, 1990. I also make use of William S. Burroughs' classic book about heroin addiction: *Junky*. Although *Junky* is strictly a work of fiction it is considered a semi-autobiographical account of Burroughs' own struggle with heroin addiction.

with themselves, happiness in their own skin. The addicts whose memoirs I consulted expressed the feeling that, at one point or another, the non-intoxicated version of themselves – the often anxious, depressed, or irritable version- was not truly them. At least it was not a version of themselves they were willing (or wanted) to accept. This is a kind of general experience associated with the addictive experiences I discuss in this chapter. The phenomenology of addiction, for some addicts, revolves around the addiction providing a general sense of self-happiness, self-satisfaction and to a certain extent their identity.

This first aspect of addictive phenomenology is something akin to the idea, which I discuss in chapter 1, of "existential dependence" which is developed by Francis Seeburger.[136] In that chapter I stepped back from the term "existential dependence", as used by Seeburger, since according to him it implies that addiction can provide an addict with an identity just as a religion does for a zealot. I disagree with him because that might incorrectly imply that the phenomenological experience I am talking about is pure and untainted by doubt and internal conflict. It is certainly *not the case* that the relationship between an addiction and general self-happiness is not riddled with self-doubt and inner conflict. However, I believe that one important aspect of the disordered passion of addiction arises from the close relationship addiction has with the view addicts have of who they are, their self-worth, and what makes life worthwhile (and in this way it is a cousin of "existential dependence").

[136] I will discuss existential dependence and its distinction from disordered passion in the next section of this chapter.

Addicts sometimes express this feeling as if the addictive substance were some agent capable of bringing out a better version of their personality. It can sound much like how someone would talk about a lover or spouse. Several authors write about the state of being addicted to drugs as like someone caught up in a consuming love affair. Caroline Knapp, a newspaper columnist and author -and an alcoholic for more than a decade- is explicit about this. She wrote:

> It's not at all unusual in AA to hear people refer to alcohol as a best friend, and to mean that on the most visceral level: when you're drinking, liquor occupies the role of a lover or constant companion. [137]

Knapp goes on:

> The paradoxical thing about drinking alone…is that it creates an illusion of emotional authenticity which you can see as false only in retrospect…the liquor truly seemed like the one thing that gave me access to my true feelings, a route to real emotion.[138]

Knapp is not alone in the depth of her feeling. Jerry Stahl, a magazine and television writer -as well as a heroin and cocaine addict for nearly two decades- had similar experiences.[139] In his memoirs Stahl makes an interesting observation about his relationship with heroin:

> I could no longer stand where narcotics took me. But I knew I would go back to them. I was like the battered wife who goes back to her husband because she likes the way he treats her when he isn't killing her… .[140]

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[137] Knapp, p.104, 1996.

[138] Ibid, p. 116.

[139] Stahl wrote scripts for several popular shows in the late 1980s and early 90s including, *Alf*, *Moonlighting*, and *Thirty-Something*.

[140] Stahl, p.296, 1995

Like Knapp he also saw drugs as a way of getting in touch with a more authentic self. In recalling his experiences of caring for his infant daughter, while still using heroin, he wrote:

> There's no defense, really, beyond the niggling fact that the dope kept Dad's hands steady. It took away the sickness. Enabled the joy and compassion buried under all those drug encrusted layers of self-loathing to trickle to the surface. If you've been there you get it. If you haven't, what can I say? [141]

Owen Flanagan, a professor of philosophy at Duke University, had similar sentiments about alcohol and a sedative/anxiolytic class of drugs called benzodiazepines, to which he was addicted for 20 years. He writes:

> There is something it is like to be an addict…to be addicted to booze, versus being addicted to benzodiazepines, specifically clonazepam, the name of my generic pharmaceutical lover…..But both alcohol and benzos did produce some sort of safe feeling. It is hard to describe but it is less like feeling objectively safe …It was more of an existential anxiety involving not feeling safe in my own skin – being scared *simpliciter*.[142]

In each of these cases we see a personification of an addictive substance as a lover and comforter. Each author reinforces the notion that an addictive substance can be so deeply integrated into the addict's experience that it seems to take the role (metaphorically) of a sentient being.

William S. Burroughs in *Junky* -his semi-fictional account of his struggle with heroin addiction- also seems interested in finding a purpose or meaning to his life through the use of drugs. Discussing the "kick" he gets from drugs Burroughs writes," Kick is seeing things from a special angle. Kick is momentary freedom from the claims of the

[141] Ibid., p.222

[142] Flanagan, p.275, 2011

aging, cautious, nagging, frightened flesh. Maybe I will find in yage what I was looking for in junk and weed and coke."[143]

In my view these examples show that the phenomenology associated with an addictive drug can take on a richness that strongly mimics (perhaps in some sense actually is) a genuine attempt to find something that will bring the addict self-satisfaction and peace with themselves. Giving them a different sense of themselves just as finding a new love might do for anyone at large. This is what Stahl meant when he wrote of shooting heroin in order to tap into the joy and compassion lying beneath, "drug encrusted layers of self-loathing".[144]

The most common theme in of the phenomenology of addiction, among the authors I have read, is that the addictive substance in some ways makes living, in the general sense, an agreeable idea. I believe many addicts tie themselves to an addictive substance as a means for a better life –simpliciter. For as many problems and as much turmoil as addiction causes, understanding an addict's phenomenology means one must understand that *at certain points* the addict sees the addictive substance *as* a necessary means to a satisfying life. As Jerry Stahl puts it, "I did drugs because there was another

[143] Burroughs, p.128, 1977: The 1977 edition contains a glossary in which "kick" is defined in two ways," It can mean the effect of a drug [in an acute sense]…A kick is also a special way of looking at things so that the man who is "on kicks" sees things from a special angle." Yage is a new drug he wishes to try. Given the reflective and general tone of the reference I think Burroughs clearly is using "kick" in the second sense.

[144] cf. footnote 141 ; One can already see the conflict arising for Stahl in seeing heroin as necessary for authentic emotion and heroin as causing his authentic emotion to be buried and obscured.

world, and I wanted to live in it. Because I preferred it to the one I happened to inhabit."[145]

Francis Seeburger points out that, according to Nietzsche, meaninglessness itself is painful. He claims the addict is in search of something to fill the void left by meaninglessness. According to Seeburger, "What makes addiction so tempting is precisely that it promises to fill the gap [of meaninglessness]. The problem is that addiction's promises are hollow. Unfortunately, however, once we find that out it is already too late." Seeburger seems to be addressing both the underlying motivation that leads to addiction along with the reason for the continued use of an addictive substance. I am only addressing the phenomenology from the point of view of someone who is addicted. However, his observation about meaninglessness is an interesting one. In so far as the experience of addiction involves it seeming to the addict that they are making themselves and their lives better, more worthwhile, or authentic, it must appear to have *some* meaning to them.

Again, my focus is not on any antecedent meaninglessness that might drive one to addiction, but on the force of the phenomenology associated with addiction. The phenomenology surrounding addiction can seem strikingly similar to the sort of phenomenology one develops with respect to a turbulent, but passionate, romantic relationship. It is worth emphasizing that I have no wish to focus on a specific meaning of addiction (e.g. whether it is the answer to a root sense of meaninglessness or it is always a palliative for untoward emotions). My focus is on the *simple fact* that there is a

[145] Stahl, p.241, 1995

prevalent passionate quality to the general nature and strength of the phenomenology of addiction that revolves around a sense that the addict has about the importance of the addictive substance in their lives. The root of the disordering of the addict's passion for an addictive substance lies in this sense of importance. That is, the passion for the addictive substance is well out of proportion to its actual importance from an objective, or even reasonable, standpoint. I will discuss this further in section 2.

The addicts' stories I am considering make it seem quite clear that the addict's phenomenology is broadly ensnared by addiction. However, the phenomenology of addiction can invade particular parts of the addict's life. The disordered passion of addiction is not simply of a general variety (e.g. for self-happiness in general). Disordered passion for an addictive substance can also be manifested as a passion for some particular role it plays in an addict's life.

1.2 Specific Roles of Addictive Substances in an Addict's Phenomenology

The phenomenology of addiction is not only couched in the broad terms of self-satisfaction, meaning, and identity. The addict also experiences an addictive substance as something which plays specific roles in work, relationships, and family life. For instance some addicts doubt whether they can continue to be creative and productive in their professional lives without using. Others find dealing with the difficult emotional elements of interpersonal relationships without their addictive substance a near impossibility. In this way the phenomenology of addiction is like a fluid. It can not only soak the addict's

broad life view but it seeps into the crevices and cracks of different (and specific) parts of the addict's life. The phenomenology of addiction is especially invasive in this way.

Jerry Stahl and William Styron, as writers, both express the feeling that heroin and alcohol, respectively, were critical elements to their creativity. After one of a series of detoxes Stahl expressed his concern bluntly, "My big fear, as ever, was that I couldn't write without getting high. A problem I resolved simply by not writing."[146] Styron, who admitted to abusing alcohol and sedatives, likewise expressed the perceived value of drinking to his creative abilities. He wrote:

> I used alcohol as the magical conduit to fantasy and euphoria, and to the enhancement of the imagination….which had contributed greatly to my writing… Alcohol was an invaluable senior partner of my intellect, besides being a friend whose ministrations I sought daily… [147]

Stahl and Styron are expressing a perceived connection between heroin and alcohol and their success in a profession to which both are devoted (each makes it clear that he believes writing to be his calling). It might be a bit melodramatic to say they connect successful writing with substances of abuse as closely as they connect it with language itself, but only a bit. They both seem to express a view, or feeling, that their creative facility is locked away in a place that the addictive substance (and perhaps only the addictive substance) can access. The emotional concern that Stahl and Styron express here is instrumental in nature. However, it is no less an emotional concern than a man who craves luxury or fame has for the currency that will bring him those things (i.e. someone who loves money, not an uncommon person at all). For Stahl and Styron their

[146] Stahl, p.336, 1995

[147] Styron, p.40, 1990; Interestingly Styron claims to never have written a word while under the influence of alcohol, although clearly the creative (not technical) process of writing he freely admits was heavily influenced by his drinking.

experiences seem to be a mixture of a fear of loss (of their creativity) and a love of liberation that the addictive substance provides.

But the phenomenology of addiction is even more insidious. It can manifest itself by making one come to believe that personal and even intimate behaviors, i.e. social and romantic relationships, are themselves dependent on an addictive substance. The addictive substance seems a kind of necessary 'silent partner' in the health of these relationships. In the most general case it can be seen as vital (perhaps necessary) condition for making even basic social connections. Caroline Knapp describes this particularly well:

> Drinking was the best way I knew, the fastest and simplest, to let my feelings out and to connect, just sit there and connect, with another human being. The comfort was enormous…. one of liquor's most profound and universal appeals to the alcoholic: the way it generates a sense of connection to others.[148]

Knapp is not simply saying that alcohol reduces inhibitions and allows one to more easily strike-up a conversation with a stranger, or deeply connect with a friend. Many non-addicts find that a few drinks allow for easier conversations with friends or strangers. Knapp seems to be maintaining that drinking, for the addict, becomes *the* preferred (and perhaps only viable) option for approaching such social interactions. For the addict it becomes the foundation for connecting with others whereas for the nonalcoholic it is merely beneficial, or a luxury, in social situations. Knapp's point is this: For the alcoholic alcohol is like the engine of the social interaction. For the non-alcoholic it is more like the comfortable cushion, nice but not strictly necessary.[149]

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[148] Knapp, p.68, 1996

[149] I think it is worth noting that I once heard a member of A.A. express that his main fear, upon quitting

Knapp's observations about herself and the alcoholics and addicts she met during her recovery include an even more disturbing role for alcohol -in her estimation especially for women- in romantic life. Knapp wrote, "The deeper connections between alcohol and self-worth and sexuality, the way women (at least women like me) use alcohol to deaden a wide range of conflicted feelings….weren't addressed with much texture or depth."[150] Knapp continued, "If you both long for intimacy and fear it, if you feel unworthy of it and ill equipped to receive it and ashamed of yourself for wanting it, alcohol becomes a most useful tool, a way of literally drowning the conflict."[151]

It should be no surprise that addictive substances also are a method of coping with familial challenges. Jerry Stahl began using drugs, in part, as a way out of the stress and anxiety that arose from his father's suicide (when Stahl was a teenager) and his overbearing mother. Perhaps the most powerful example of his use of narcotics in this regard (i.e. towards the end of psychological relief from familial anxiety) is when he recounts finding out about the pregnancy of his wife. Stahl recalls:

> "I'm pregnant," she said, her voice almost a whisper.
> My heart sank…You're pregnant, fine, I have to go get fucking high, okay?
> …To kill all the feelings I couldn't even feel, numb emotions I couldn't name.
> To vanquish the rapidly mounting panic that already had my heart slamming
> at my ribs, like an animal who realizes it should have fled the cage when it
> was still open.[152]

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drinking, was that he would never again be able to socialize normally at parties or other gatherings.

[150] Knapp, p.80, 1996

[151] Ibid., p.81, Knapp tells an interesting story about a female friend and fellow alcoholic who felt that drinking, "released this current, let it stream up and out. There was a fuck-you element to it: a feeling of fuck you, I am going to get what I want even if I don't believe I deserve it."

[152] Stahl, p.191, 1995

Knapp has a different experience in relating her addiction to family. For her alcohol was a means to connect with family (especially her father), whom she often found aloof and distant. She recalls a particular instance from her late teen years:

> I also remember a feeling of emptiness, a wariness, something I often felt in my father's presence…But then the wine came, one glass and then a second glass…The wine gave me a melting feeling…and I felt like safety itself had arrived in that glass, poured out from the bottle and allowed to spill between us. I don't remember what we talked about, but I do know that the discomfort was diminished, replaced by something that felt like a kind of love.[153]

I believe what is beginning to emerge among the addicts' stories I am considering is a pattern in the phenomenology that is far distinct from what one would expect if addiction could be explained solely in terms of biological appetite. There is widespread personification of the addictive substance, coupled with the feeling that life and many aspects of it, can be swaddled and protected by alcohol, or cocaine, or benzodiazepines, or heroin (if only fleetingly). These addicts seem to have a sense that their addictions keep life in general, relationships, work, and family life functional, or at least tolerable. But there is an additional element of addictive phenomenology that must be considered. The addicts being discussed were not so self-deluded that they failed to see the dark side of their addictions (I suspect this is true with most addicts). A deep part of the phenomenology of addiction involves the experience of opposition and hatred for the continued use of the addictive substance. It is to that which I now turn.

1.3 Addiction and Internal Conflict

Addicts recovering from addiction often realize, to their horror, that the addictive substance they had come to believe was their salvation from anxiety, depression,

[153] Knapp, pp.39-40, 1996

existential fear, self-loathing, or uncertainty of purpose was a mere palliative. As William S. Burroughs observes about heroin use, "Like a man who has been away a long time, you see things different when you return from junk [i.e. heroin addiction]."[154] It is clear, though, that most addicts have some idea, or get a glimpse of this different view during active addiction. This is what (in part) causes the anguish or internal conflict felt by the addict about his or her addiction. The different view Burroughs mentions is one, I think, that inclines the addict to realize that the investment of passion and self into an addictive substance was a waste of emotional energy on something that not only failed to live up to its promise, but hurt the addict in the long run.

In many instances the activity surrounding addiction is probably done without much reflective thought (and so no real internal conflict). That is, seeking out a heroin dealer, or going to the local bar for drinks after work becomes routine (a habitual behavior) with no conflict. But within the addicts' stories that are under consideration in this chapter there are myriad moments where a conflict arises between the desire they have to pursue their addiction and their desire to be rid of it. How can this be explained? Some have suggested that what is occurring with addiction is a kind of self-deception in which the addict willingly puts on blinders –simply through the habitual practice of not reflecting on consequences- in order to prevent them from seeing the truth. The conflict arises when those blinders occasionally slip.[155]

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[154] Burroughs, p.127, 1977

[155] For an interesting discussion of self-deception see Herbert Fingarette's book *Self-Deception*. Originally published in 1969, the 2000 edition includes an additional chapter.

Whatever the source, the experience of internal anguish associated with self-conflict is common in addiction. Jerry Stahl has two interesting accounts of this conflict. In the first instance he had his infant daughter in the car after he had bought heroin, and he was pulled over for a traffic violation. He told the police officer that he was on the road at 3am because driving was a way to calm the baby. Stahl writes:

> Only once –*I used my own child as a front…to save my ass… I risked everything. I did that*…Only once, as the plunger was driving home, did I let the thought sneak out.[156]

Stahl recounts another instance which occurred shortly before one of his numerous attempts to quit heroin and cocaine:

> And then, in a single astonishing insight, it hit me: *This is what I am.* I am one of those people normal people see and think "*sick*…" Think "*fucked-up*"…And I didn't care. I didn't care about anything except making it back to my Alvarado to get high enough to blow all of them right out of my fucking brain.
>
> That moment scared me. I'd seen something I didn't want to see.[157]

Knapp shares her experiences along these lines. She recalls:

> It amazes me now that a part of me recognized the problem so long ago. But I guess that's the way alcoholism works, you know and you don't know. Or, more accurately, you know and the part of you that wants no part of this knowledge immediately slips into gear, sliding the fear into a new category…it's reclassified: *a little problem with drinking*, something you'll take care of when you're less depressed.[158]

The conflicted feelings of the addict do not remain stagnant. In the final analysis Knapp believes that it is the gradual growth of the conflict between the addiction and the desire to be free of it that leads to recovery (the point at which the strength of the conflict causes someone to quit, which is known as "hitting bottom"):

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[156] Stahl, p.228, 1995, the italics are in the original text.

[157] Ibid, p.254, the italics are in the original text.

[158] Knapp, p.108, 1996 the italics are in the original text.

> Truly landing, landing with such finality you realize you have to get off the elevator or you'll die, requires an elusive combination of despair and grace, something known in AA as "the gift of desperation."[159]

The tendency to personify and build a relationship with an addictive substance crosses paths with the disturbing realization that the 'lover', 'friend', or 'partner' is merely an indifferent and inanimate entity capable of producing great harm. I believe that over the lifespan of an addiction this realization comes and goes, and, as problems associated with addiction begin to grow the realization becomes stronger for most addicts. I do not deny that there may be willing addicts who do not experience such conflict. However, it is an important part of understanding the disorder associated with the phenomenology of addiction that some (I suspect most) addicts do experience internal conflict about addiction.

This naturally leads one to wonder: How does the addictive side of the emotional and cognitive conflict get privileged (in terms of producing action) over the other? There are several possibilities as to why the addictive passion wins out over the alternatives. In chapter 4 I claim that addictive disordered *appetites* can exist simultaneous to addictive disordered *passions*. Perhaps the appetite lends its weight to the passion in some way and is a deciding factor. It might be the case that addictive passions are less emotionally painful to pursue than their alternatives, and so are simply privileged because they are the best of a bad lot in a series of near term choices. I suspect another prevalent reason for the privileging is that addictive passions are more highly desirable because of their direct

[159] Ibid, p.215

experiential effects and it is much easier to follow a passion that provides immediate pleasure.

§2. The Phenomenology of Addiction Understood as a Disordered Passion

It is now necessary to explain why I believe the experiences I have been cataloging deserve to be categorized as passions, and second what it is for a passion to be disordered. In regard to the former I think it is important to set out some sketch of what it is to be a passion. I will ground my notion of passion in David Hume's use of the term in *A Treatise of Human Nature*.[160] I chose to use Hume's notion of passion because it is a classic and deeply rooted philosophical notion of passion. I also believe it to be basically correct.

According to Hume passions are secondary, or reflective, impressions derived from our sense experiences. For Hume this includes what we traditionally call the emotions. Passions are divisible into two types: calm and violent. According to Hume, "Of the second are the passions of love and hatred, grief and joy, pride and humility."[161] Hume goes on to say that there is a further division of passion into direct and indirect categories. Direct passions arise immediately (e.g. anger at being insulted), whereas indirect passions, "proceed from the same principles, but by the conjunction of other qualities."[162] These other qualities are ideas and reflection and the associations they have

[160] Shelby-Bigge (ed.), 1978

[161] Ibid, p.276

[162] Ibid, p.276

with impressions. Many of the violent passions (i.e. the strong passions) fall into this category. Hume believes that hatred and love are, "always related to a thinking being".[163]

I do not necessarily disagree with Hume that the standard notion of love seems to require that the object of love be a thinking being. When one person loves someone else they are interested in the well-being of the other for the other's own sake. I believe this to be a core conceptual element of love, and it is conceptually problematic to be interested in the well-being of thing, for the thing's own sake.[164] However, I believe that the persistent personification of addictive substances we have seen from the addicts discussed shows that a passion connected with love can exist for a thing.[165] I will specify what exactly I take this to mean in a moment.

With that as a rough outline of passion, I will start to lay out the case that what the addicts in the previous section were describing was a passion for the object of their addiction. One thing that seems undeniable is that the addicts I have discussed had strong emotions for the object(s) of their addiction. I think this is the best way to explain the *consistent personification* of the addictive substance in terms of a lover, a friend, or a

[163] Ibid, p.331

[164] For a detailed discussion of love and its conceptual elements please see "The Reasons of Love" by Harry Frankfurt, 2006.

[165] The metaphoric notion of love is a rather common one. We often think of people as having a 'love of power' or a 'love of money'. In those instances I do not think love is used in a literal way. My interpretation is that these are metaphorical uses of love, and a literal interpretation means that the person deeply cherishes those things. Usually we cherish things because they are often connected to something we do literally love. In the case of greed usually it is one's self. To be literal one should say of the greedy, "He cherishes money, because he loves himself and his lifestyle."

partner. As Knapp observes, "When we're deeply in love with drink, we have no idea what kind of fire we're playing with."[166]

For Knapp, as well as Owen Flanagan these feelings had their origin in the simple impressions each got from the reduction of anxiety and angst that drinking (and taking benzodiazepines in Flanagan's case) brought. That association was, of course, reinforced over long periods of practice in these methods of anxiety reduction. Styron, Stahl, and Burroughs also built upon their early impressions of drugs as keys to creativity through repeated exposure. At some point in the string of using drugs there arose for each of these addicts the reflective impression that the particular sensation (what it did for them) was an integral part of a way of life which they preferred (e.g. an anxiety free, or creative, or socially connected life).

Given Hume's view of passion I believe that this qualifies the addicts I have discussed as having a passion for their drug of choice. I earlier noted that conceptually it is hard to think of caring about an object for an object's own sake, especially when one's intention is to consume that object (as is the case with addicts). So it seems problematic t maintain that the addicts actually loved their drugs. However, to argue that the addicts were *just concerned for the instrumental value* of the addictive substance (e.g., alcohol) is to miss or ignore something important. To argue that only a simple relationship of instrumental purpose exists between addicts and their substance or behavior of addiction (as something similar to the relationship between me and my shoes) does not capture the passionate description and entanglement that one sees time and again in their words.

[166] Knapp, p.124, 1996

The closest I can come to a precise interpretation of the passion the addicts felt toward their addiction is to say that they *cherished* their drugs, in a way that goes beyond viewing them as mere tools. That is to say *they showed care for and emotionally valued* their substances of addiction. The addicts cherished drinking or shooting heroin not dissimilar to the way a Christian might cherish the sacrament of communion or, a wife might cherish a letter from her departed husband, or a grandson might cherish a watch given to him as an heirloom by his grandfather.[167] I noted earlier that I while I do not disagree with Hume (although he may be wrong, my job is not to investigate the notion of love thoroughly), there is something in the addicts' words that indicate a *passionate* attachment to these addictive substances that seems connected to love. I believe this is why the notion of cherishing -which is closely connected to the passion of love- is probably the best description of what is being manifested in the accounts given by the addicts in this chapter.[168]

I claim that one can cherish an unthinking object.[169] I believe this is because cherishing, as a passion, is closely connected to love. What is it that our addicts were actually in love with? I suspect it was a style or manifestation of their lives, when using

[167] I do not think this brings me into agreement with Seeburger, since he thinks addicts behave as though they were religious devotees and this is at the core of addiction. I am not making a broad claim like that, rather, I am merely thinking of another context in which the notion of cherishing is clearly at work.

[168] Hume himself recognizes the connection, when discussing animals, in Book II, Part I, section xii of *A Treatise of Human Nature*. Hume wrote, "Accordingly we find, that by benefits or injuries we produce their love or hatred; and that by feeding and cherishing any animal, we quickly acquire his affections; as by beating and abusing him we never fail to draw on us his enmity and ill-will." Hume may be using cherishing to describe acts, but they are acts that arise from care and value, or the feeling of cherishing.

[169] Think about an heirloom from a dear departed loved one. It makes clear sense to cherish such things, and they are cherished precisely because they are closely connected with a person one loved (or loves).

drugs, with which they were in love (I think Hume and renowned philosopher Harry Frankfurt, who holds the aforementioned view of love, could accept this as a subject of love). In short, they loved themselves, or the person that the drugs allowed them to become, and thus they cherished the drugs. It seems clear that each one could *cherish* his or her addictive substance, even if, given the conceptual framework of love, they could not love it for its own sake.

The reflections of the addicts discussed shows that a strong passionate connection can be a part of the phenomenology of addiction. Especially telling in this respect is what the addict feels when faced with giving up their drug of choice. Caroline Knapp expressed her *despair* at the prospect of never drinking again in the days leading up to her entering rehab:

> Between the day I knew I had to stop drinking and the day I finally did, I cried almost every night…..I felt like I was giving up the one link I had to peace and solace, my truest friend, my lover. I felt like I was trading in one form of misery for another, like I was about to leap into a void, like my life was ending.[170]

Owen Flanagan also related his prospective thoughts about quitting. In discussing the final days of his addiction, Flanagan wrote, "I found the idea of complete abstention from alcohol inconceivable, terrifying."[171] Jerry Stahl, looking back after being clean for a couple years, reflected on what giving up heroin meant for him, "What is heroin, really, but every junkie's teddy bear? What makes a soul feel all snuggly and cutesy-poo…"[172] These feelings of prospective fear about the loss of the warmth and comfort that result

[170] Knapp, p.242, 1996

[171] Flanagan, p.290, 2011

[172] Stahl, p.309, 1995

from giving up an addictive substance is clearly not like losing a pair of shoes, or a cell phone, or even a car. It might be a pain to lose these things but insofar as they are purely instrumental the pain involved is not (usually) of passionate loss, but one of inconvenience. Giving up an addictive substance, however, seems much closer to the experience of losing a deeply cherished heirloom or giving up a passionate relationship. I think the words of the addicts I have presented bear this out. Their passions ran deep. I now offer two reasons to think these passions might have been disordered.

2.1 Addictive Disordered Passions Exclude Countervailing Considerations in Decision Making

One way for a passion to be disordered is for it to grow and continue to grow in a way that is beyond control of the agent, much like cancer cells grow beyond the control of regulatory (inhibitory) mechanisms of the immune system. I believe the problem of disordered passion occurs with the weakening, over an extended period of time, of one's ability to properly recognize countervailing considerations (including but not limited to reasons, perhaps other passions) that ought to be sufficient to move the agent to suppress (or at least not act upon) the disordered passion. It behaves just as cancer does when it causes the weakening of the function of healthy cells.

A healthy passion is one that *can be* routinely blunted in its motivational efficacy by sufficiently strong countervailing reasons. Specifically, a healthy passion is one for which parameters or conditions exist (be they normative or otherwise) under which a reasonable person would stop acting upon the passion. Suppose a new father gives up ice climbing as a hobby because it is too dangerous, and he believes that with a family to

look after his risky passion is not worth it. This appears to be an instance in which we could say the person is acting reasonably with respect to their countervailing reasons against some passion.

An example of a disordered version of passion might involve someone refusing to hand over twenty dollars to an armed robber out of a sense of pride or machismo. It seems to me that passions not amenable to sufficiently strong countervailing reasons are usually both self-defeating (e.g. the robber shoots the resistor and takes the money anyway), or reflect a serious misjudgment in the strength of countervailing reasons (e.g. it is no real insult to pride to hand over an insignificant amount of money under duress or threat).

So one way for a passion to be disordered is when despite clearly sufficient reasons or conflicting passions which ought to trump its motivational efficacy, the passion continues to motivate action. While I use Hume's definition of passion, *I reject* his classic notion that reason is always the slave to passion. I believe sufficiently strong reasons are able to overcome passions, when the passion is within a healthy range (and sometimes when the passion is disordered). However, I also believe that passions can thwart countervailing reasons.

How does this work? In one of two ways, the force of countervailing reasons can be recognized but ignored, or the strength of the countervailing reason might go unrecognized for various reasons. Either way, there is more of a contest afoot between reason and passion than Hume appreciated.

Let me illustrate an instance of *ignoring* a countervailing reason in the context of an experience of one of the addicts I have been discussing. As I already related, Jerry Stahl once took his infant daughter to a 'drug-house' to procure heroin (and was almost arrested driving home afterward, his daughter in her car seat). Having done such a shameful and dangerous thing one would think that that would lead Stahl -with great force- to come to a decision to give up his heroin addiction. Most parents would give up a passion, were it a healthy passion, if it caused them to endanger their child. He simply ignored the gravity of having taken his daughter out with him to score heroin at a 'drug-house', by avoiding thinking about what he did by getting high.

Of course, I would not expect the fact that he endangered his daughter to automatically blunt his appetite for heroin, he may remain an addict in that sense (in chapter 4 I will claim that disordered passions and appetites can be contemporaries). What I, and most reasonable people, would expect is that Stahl would divorce himself from any deep feelings for heroin. That in fact he would *focus* on his dastardly act and the strength of reason it provides for him to hate heroin. In other words, even if his physical appetite was not reasons responsive to what he had done his passions should have been. This means he should have divorced himself from his passion, or at the very least ceased to allow his passion for heroin to drive his behavior. If he continued to use heroin one would expect his relationship to be similar to one a patient has with hospital food, or a recruit does with army chow. In short as something necessary to suppress appetite and avoid withdrawal, but nothing to cherish.

However, according to the quotation I cited earlier (see footnote 156) Stahl let the thought 'sneak out' only once that he had done such a terrible thing. His passion for heroin did not swerve or abate because he did not dare focus on his daughter's endangerment. While he clearly had knowledge of the terrible thing he had done he did not yield to its weight in guiding his passions, because he never bothered to put it on the scale. Years later he reflected on the fondness for heroin he continued to feel even after that incident, "I could exist in imaginary circumstances with greater ease than real ones. I did drugs because I felt the exact same way about my life my little girl already seemed to feel about hers."[173]

His experience has parallels with the stories of Knapp, Flanagan, and Burroughs. Each has embarrassing or terrifying or painful experiences because of their addictions. But as long as their addiction continues these experiences tend to get discounted or forgotten or excluded as counting in favor of quitting. Colloquially this is thought of as the product of what is called rationalization, or selective memory.

However, there is *another reason* that addictive disordered passions persist, despite good reasons that one ought to give up or not act upon the passions. In some rare cases of addiction it seems the passion can be sufficiently strong to *overpower* countervailing rational considerations that *ought to be a sufficient condition* for altering behavior. This is not a case of ignoring countervailing reasons it is a handicap in recognizing their force (the scales in the addicts mind are rigged). This is a less frequent (perhaps uncommon) manifestation of the way addictive disordered appetites are not

[173] Ibid, p.241

responsive to reasons. They usually occur when an addict suffers severe cognitive impairment or perhaps a co-morbid psychological disorder. All the same this is a second and distinct model for an addict's failure to respond to reasons.

One could rightly point out that everyone gets swept up in a passionate decision that causes them to ignore or overlook countervailing reasons once in a while. What makes the addict distinct in this regard? The first difference is that the addict acts diachronically on the passion that reason says they ought to give up. For them getting swept up in their passion becomes a way of life. Second, it is the establishment of either of the two aforementioned sorts of *standing* relationship between addictive passion and countervailing reasons that explains the disorder of passion. *Either disordered passions cause one to ignore strong countervailing reasons, or they simply do not allow one to appreciate the strength of those countervailing reasons.*[174]

The fear associated with losing (i.e., giving up) an addictive substance seems to involve much more than fear of withdrawal or dope sickness (which would be more closely associated with appetite). The fear is at the prospect of going on in life without something one *cherishes*, in an existential sense, as we saw with Knapp, Stahl, and Flanagan. Giving up an addictive substance can feel as though it were the gut-wrenching loss. It is no wonder that passions, of this strength, can either drown-out or (in rare cases) overpower other elements relevant in the deliberative process.

[174] I am using a distinction made by John Martin Fischer. I am supposing that there are two different mechanisms which might cause the addict to be unresponsive to countervailing reasons. The first is to (as Stahl seemed to do) simply ignore or suppress the reason, the second is that passion may overcome the addict's ability to appreciate the force of a reason.

I should point out that this in no way relies on the concept of delay discounting (although delay discounting is consistent with what I am claiming). I am making no claims about the relative temporal distance of addictive and non-addictive substances. Rather I am claiming that the weight of considerations which run against the addictive passion are ignored (or not recognized due to inability) because of the disproportionate absolute value (not necessarily delay discounted value) given to a disordered passion. In that way these countervailing reasons are excluded from having a proper place in the decision making processes of the agent. Temporal delay is not critical to this picture.

It is important to recognize, and contrast, strong passions that are *not disordered* with those that are. It is also the case that there are disordered passions that are *not necessarily of an addictive nature*, and so a distinction should be drawn here as well. In regard to the former, passions that are deeply held but not disordered and usually justifiably resistant to a large range of countervailing reasons are those determined by reasonable moral commitments and values. The love a parent feels for a child, a spouse for a partner, or patriots for their country seem to be examples of passions grounded in a deep moral framework. Passions of this sort will work to direct the thoughts and considerations that determine a person's actions, often in the face of strong countervailing reasons (e.g. sacrificing one's life to save one's child). But such passions are deeply justified by the rational framework of morality, so implicitly they have good reasons that justify them. So that even if it is the healthy, deep passion that motivates the person it is grounded in good moral reason. In the case of disordered passions such grounding of the passion is lacking.

Let us take a closer look at one of these cases. When the passion of love is felt for a child the subject of the passion is a person, not a behavior or substance (giving the passion held for a child greater moral standing by almost anyone's lights). If the passion of love causes one to decide to give up one's own life for one's child's life, the passion is outweighing countervailing reasons, but not in a disordered way because the passion is deeply grounded in moral justification (i.e., there is good moral justification for one to love one's child). Even with justifiably deep passions there is still a reasons threshold that ought to be sufficient to override the passion. In the case of the love of one's child that threshold would be extraordinarily high (e.g., space pirates would destroy most of humanity if you did not sacrifice your child, even one's passion for one's child can be disordered, I will discuss this in the next paragraph). Passions for addictive substances seem to have a much weaker moral grounding (e.g., they might be grounded in the right of self-determination), and the disordering of addictive passions occurs much more easily because the countervailing reasons which justify not acting on the passion are much lower (of course addicts behave as if the countervailing reasons had a very high threshold, therein lies the problem).

But even with parents the passion for their children can become disordered. When a parent begins to believe their child can do no wrong, or refuses to let the child go outside (at all) or have any friends for fear that he or she may get sick, the passion has become disordered. This sort of parent has lost the ability to appreciate countervailing rational considerations that have crossed a threshold which should override the effectiveness of the passion. In the case of a parent with a disordered fear for the safety of

their child he or she fails to take a proper perspective on the epistemic (e.g. a child playing outside in the area has never been seriously hurt), and rational (e.g. to properly develop children need friends) considerations that ought to trump their own deep fear (i.e. the extremely overprotective parent fails to recognize *the importance* of allowing a child to have friends and play outside, because the *deep fear* they have for the child's safety vitiates the reasons that countervail against the overprotectiveness (or just makes them ignore the reasons)).

The same basic structure of disorder occurs with the addict. Evidence for this can be seen simply in the fact that most addicts recognize, after they are no longer actively addicted, the incredible force of the reasons they had to quit all along. Jerry Stahl records this in his memoirs:

> *Can you understand this? Shooting dope is about getting warm and fuzzy. Dependably so. But the Daddy-rush…forget about it! I've never felt anything so terrifying! It's so real, even the pleasure can break your heart. Which, in the grand scheme of things, is what separates shooting smack from loving your little girl.*[175]

I think Stahl is making my point. In retrospect heroin was a passion, but it drown out an understanding of an important reason to quit using heroin: the power of a loving relationship with his little girl. So even if there were some moral grounds for Stahl's passion for heroin they were decisively eclipsed by the reasons acting against them (i.e., being a father to his little girl). It seems in the end Stahl is admitting that the weight of this reason, being a father, should have outweighed his passion for heroin all along (had he not been ignoring that reason).

[175] Ibid, p.309

Knapp makes an observation about how a recovering addict's perspective on life decisions changes (presumably because the reasons for and against these things are now viewed differently, or considered at all). Knapp wrote, "I'd never really given myself a chance, a sober chance, to reach any conclusions…Do I want a child? Do I want to be married? I don't know. I can only sense the shadowy outlines of my own hopes, as though the haze from all that alcohol still needs time to burn away."[176] Knapp, like Stahl, is appreciating the weight and depth of certain concerns only after her passion for alcohol has abated. In part it was the committed romantic-like relationship to drugs that drowned out the depth of value in parenthood for Stahl and obscured the importance of making deeper value considerations for Knapp. The problem was that the passion for drugs, in both cases, lost each addict the ability to make accurate value assessments about countervailing reasons to quit using drugs (or just caused them to ignore such considerations).

Is there a manifestation of disordered passion that is specific to addiction? Yes, if one has a disordered passion (of the type already discussed) which involves cherishing a *substance or behavior* primarily because of the experiential effects it has on the psyche, then that is an *addictive* disordered passions.[177] I suspect that what might make this variant of disordered passion specific to addiction is that it is self-centered. That is, the passion that exists in the case of addiction is *essentially* unidirectional coming from a

[176] Knapp, p.277, 1995

[177] I am taking anything that can be loved (like a person) off the table. The key element to understanding addiction is that it involves cherishing something that is connected to love. Love itself may present its own set of disorders but it is not something to which one can be directly addicted. I *am not* claiming that the addicts in this chapter had disordered passions because they were addicted to loving themselves.

person and being directed at a thing, because of that thing's *cherished* instrumental value. I suspect that this in part distinguishes the non-reasons responsive disordered passion of the addict from the same type of disordered passion in a non-addict (e.g., the paranoid parent).[178] I also suspect there is more to the story that I do not yet currently see.

It is important to emphasize a parameter and add an observation about the structure of a disordered passion of this type (i.e. one that drowns out or prevents countervailing reasons from effecting action or decisions). The parameter is this: For a passion of this type to be disordered it must act repeatedly, over the course of a long period of time, to thwart reasons or considerations that might be contrary to its fulfillment. Just as a single skipped heart beat does not mean one has a cardiac pathology, or a poor night's sleep does not mean one has pathological insomnia, having only a few instances of a passion trumping reasons that should out-weigh the passion (in determining how to act) does not mean one has a disordered passion. It is the consistency of a particular passion directing decisions, over a long duration, that makes it disordered.

The observation that needs to be included is this: To determine whether a passion is thwarting reasons or considerations that should act to prevent that passion from motivating action might require a 'law-like' reasonable person standard. I think a more plausible construal is to rely on specific ethical commitments (e.g., deontic or utilitarian ethics) to determine what counts as sufficient countervailing reasons. For a law-like reasonable person standard one may need to appeal to cultural normativity. This might

[178] This section touches on the idea of reasons responsiveness as it is discussed by Fischer and Ravizza, 1998. See chapters 2 and 3 in particular.

result in some cultural variations of what counts as addiction under this disordered passion standard. A moral standard would presumably be universal.

I think the principle underlying the idea is sound. The experience of the addicts examined in this chapter shows that their passion for their drug of choice often overrode considerations that their relationships were unhealthy because of drink (e.g. Knapp), or that they were endangering their family due to heroin use (e.g. Stahl), or that their career was going down the drain because of pills and alcohol (e.g. Flanagan). What those of us from the outside looking in (and the addicts themselves in retrospect) see is that they had powerful reasons to quit that were not properly motivating, at least in part, because of the deep passion they felt for their drugs.

2.2 Addictive Disordered Passions as a Loss of Self and a False Path to Existential Meaningfulness

A passion can also be disordered because it can lead one to the existential equivalent of fool's gold. Most of the addicts discussed in this chapter seemed to feel (at one time or another, to one extent or another) that through their addiction they were engaging in a process that made their lives more existentially meaningful. *I cannot exhaustively define existential meaningfulness*. However, I take it to be comprised of the sort of projects, interactions, or relationships that an agent seeks out in order to feel as if their lives are not only narratively coherent but offer a measure of self-respect, self-worth, and value to others.

Wanting to make one's life more meaningful and expressing the self are wonderful goals. However, most of us recognize (including most recovering addicts) that

doing it by repeatedly injecting heroin or swilling vodka by the liter is not the best way to accomplish this. What I claim in this section is that on one analysis an *addictive disordered passion* leads to a corruption of meaning in the life of the addict, because it requires that they lose themselves in their passion in such a way that any meaning they find is not reflective of *their (the agent's)* interest in meaningfulness. This variant of disordered passion might be a corollary of the approach to explaining disordered passion I took in subsection 2.1. However, it is a distinct aspect of disordered passion and could follow for reasons other than the misjudgments described in subsection 2.1.[179]

Someone with this sort of disordered passion *mistakenly* believes that the object of their passion is a legitimate path to worthwhile goals. Such an addict may feel, as Knapp recounts that she did, that pursuing their addiction leads to a more authentic self, someone with whom they can be more content or more readily identify (see also Stahl, 1995). But I claim that disordered addictive passions *only appear* to lead to meaningfulness and in fact lead to meaning that is inconsistent with the authentic agent's desires or intentions. Three things account for why an *addictive* passion is disordered for this reason: *first,* the object of the passion only transiently meets the addict's needs for

[179] That an addict might properly judge all the countervailing considerations but still cling to their addiction seems plausible. Consider a case: Suppose Jim has been faced with severe anxiety his entire life and has developed a heroin addiction. He judges correctly that his addiction to heroin has cost him money, his job, and the affection of some loved ones. He weighs the value of those things properly, but loves the feeling heroin provides and the anxiety relief it produces. He feels that heroin gives him the best life he could expect. Let us suppose Jim makes no misjudgment in considering the value of heroin use and anxiety reduction vs. countervailing considerations (e.g. losing his job, or familial relationships (perhaps he never had much of a family life any)). A disordered passion for heroin might still exist *if* Jim believes that heroin has made his life meaningful *when it has not*. This is *not* to say that drugs could never help to give someone meaning, but it would be disordered to feel something which does nothing more than alleviate severe anxiety gives one meaning and purpose. That may make life better than some set of alternatives, but it does not necessarily providing meaningfulness.

existential meaningfulness; *second,* over the long term the object of the passion tends to harm the kind of meaningfulness the addict was seeking; and *third,* without the substance or process the addict finds themselves in a worse position than they were before vis-à-vis existential meaningfulness.[180]

Francis Seeburger discusses the root of the false appearance of meaningfulness that is caused by this sort of disordered addictive passion (although he implies a global necessity to the role of this type of disorder I think is lacking). His observation about addiction is that the object of passion (e.g. the drug) will not actually provide *you* with meaningfulness. Seeburger wrote:

> Addiction robs one of oneself. It deprives one of the ownership of one's own life. That life ceases to be "one's own" and becomes nothing more than an expression of the underlying addiction. Genuine creativity is gone…All of the addict's behavior comes to manifest the addiction, rather than any uniqueness of the addicted person or that person's rich individual inheritance.[181]

I do not believe that losing oneself in a passion is by itself disordered. The disorder occurs when the agent loses themselves in a passion that is intended to provide or be consistent with what they take to make life meaningful, when in fact it is inconsistent with meaningfulness for the agent. For a passion to be a *healthy passion* it must have the following attributes: A passion is healthy when one recognizes that the pursuits or outcomes of the passions are coherent, meaning that the motivation the passion engenders is consistent with fulfillment of the passion. Healthy passions cannot

[180] The second and third conditions are distinct. Taking steroids does not harm my ability to be a good athlete while I take them, although once I cease taking them I will lose muscle mass and gain fat putting me further away from my goal of being a good athlete.

[181] Seeburger, p.15, 1996

be grossly immoral (i.e., it cannot be a motivator for an immoral action the agent recognizes as grossly immoral). Finally, they must be consistent with deep genuine agential self-expression (i.e. The passion cannot be one that harms the genuine agent's attempt to find meaningfulness when it is sought, while appearing to the agent to be helping in that quest, nor can the passion be the result of a deep manipulation about the category of thing the agent finds meaningful in the first place).[182]

Typical examples of healthy passions are those one would expect to see in a good marriage, from someone with meaningful work, or from a parent toward a child. As an example, the properly passionate husband deeply loves his wife, not because he has been manipulated, but because he appreciates her for who she is and knows that she will return his love. In comparison to the disordered passion of the addict the object of his passion does not provide only transient meaning and does not harm his long term goals of finding a meaningful loving relationship (I would say this is true even in the case of divorce, so long as there was authentic love in the relationship). The properly passionate intellectual is one who, out of her own interests, pursues understanding and insights in a field she believes will benefit her and others. The properly passionate parent has genuine love for their child which they temper with moral judgments about what is in the best interest of

[182] This does not mean that a healthy passion cannot be a *superficially* manipulated passion. Suppose a wife gets her husband to like opera by taking him to an opera she knows he will like, because she knows he is already disposed to find music and dance of a particular sort enriching (say he likes musicals). Further suppose that this opera is unlike most other kinds of opera, which she knows he probably wouldn't like. Suppose the husband comes to believe based on that one performance that he will appreciate opera as much as he does musicals and immediately buys opera season tickets. Such manipulated passion can be a healthy passion, because there is no manipulation of the agent's dispositions and authenticity. In this case the wife does not manipulate her husband's agency or dispositions, just his perception of what opera is like. An important difference is that the husband is still free to change his love of opera to one of dispassion when he sees that the initial opera he viewed is not representative of what opera is like in general.

the child for the child's own sake. These examples are meant to be clear cases of healthy passions which lead to genuine meaningfulness.[183]

However, I claim that addiction is *not consistent* with genuine agential self-expression, in part because it is incoherent in certain way. It is for this reason primarily that I think addiction is a path to the equivalent of existential fool's gold. It does not necessarily seem that way to the addict. Knapp, Styron, Stahl, and Burroughs, expressed the belief, at one point or another, that the object of addiction made *their* life more meaningful either through enhancing creativity, relationships, or promoting personality change.[184]

Knapp, for instance, liked the kind of person she became when she drank. She felt more outgoing and less anxious. Her increased sociality with friends and family made her feel as if she had a deeper connection with people. These things can be significant in making life meaningful. This was certainly what Knapp thought.

Yet her passion was disordered. Why? Knapp seemed to be able to find the meaning she was looking for without hurting anyone, so her passion was not grossly immoral. She *did* become a more gregarious, more open person and she did it by drinking of her own volition (e.g., she was not manipulated to drink). This seems to be the groundwork of a normal healthy passion. However, it is not sufficient because Knapp

[183] If you disagree I think we are at a point of intuitional loggerheads. I think of existential meaningfulness as like the visible universe and the *definition* of meaningfulness as somewhat like dark matter. Physicists know that there must be huge quantities of unobservable dark matter out there because that is the only way to explain how the universe holds itself together (gravitationally). But no one doubts that the universe is (at least currently) holding itself together. I feel the same reason to accept existential meaningfulness applies here. It is clearly apparent and genuine in some cases. It is just as apparent as the fact that the universe is holding itself together (even though we cannot tell exactly what is holding the universe together).

[184] I emphasize "their" because I take it that at some point each seemed to think their addiction was genuine self-expression.

found, as did Stahl, Burroughs, Styron, and Flanagan that the meaningfulness they sought was transient in appearance. Ultimately their goals of stable and healthy meaningfulness were harmed and they found that they were worse off with respect to attaining these goals.

There is an interesting anecdote from Stahl's memoirs that speak to the transient and ultimately harmful nature (with respect to meaningful goals) of an addictive disordered passion. Recall that Stahl though heroin was vital to his writing, so vital that he feared getting clean because he did not know if he could still write for a living (see footnote 146). Towards the end of his addiction he was given one week to write a script for David Lynch and the television series *Twin Peaks*. But he lost this opportunity, not because he was clean, but because he was using heroin (and cocaine). Stahl recalls, "So enfeebled were my perceptions that I thought only a day or two had passed when the *Twin Peaks* messenger showed up at my…hideaway and asked for a draft. To say that I was unprepared is like saying the A-bomb broke a lot of windows. The sad fact is I spent the previous six days injecting speedballs."[185] Stahl's passionate feeling for the power of drugs to make him a better writer betrayed him and took him further from the meaningful goal he sought for himself.

Styron and Burroughs also expressed a feeling that their drugs of choice had abandoned them in their grander pursuits. Knapp found that drinking was a transient form of meeting her goals of being more gregarious and outgoing. In the end she felt that drinking had left her in a worse place than she had started with respect to the meaning she

[185] Stahl, p.271, 1996

sought. This is reflected in her own words, "Early sobriety has the quality of vigorous exercise, as though each repetition of a painful moment…serves to build up emotional muscle."[186] Knapp's reliance on the transient meaning she found in alcohol only served to weaken her ability to find meaning, at the end.

Addicts with this second kind of disordered passion will show all three signs I have discussed. They will find the meaning they seek fleeting, in the long run they will see it harmed, and when they cease using substances they thought would provide meaning they will find themselves further away from that which they were seeking. Of all the addicts that I discuss in this chapter Knapp and Stahl appear to come closest to having a disordered passion of this second type (there is insufficient evidence about the others to draw any conclusions).

It is for these reasons I think that addictive disordered passions of the second type involve a loss of self. The goals of the person before, during, and after addiction are harmed by the addictive behavior, which is perpetuated by the disordered pursuit of these goals. Yet we have seen time and again addicts express deep feeling for their addictive good. The best way I know to sum up the kind of loss of self I am writing about is this: if anything is a loss of self, then it would be the case of a passion which acts to support behavior that is ultimately destructive to what one genuinely wants.

Although it is a somewhat amorphous notion I think what the addictive passion displaces is the *authentic-self*. The person one truly is without being wedded to some behavior or substance that acts transiently and ultimately harmfully. In looking back,

[186] Ibid, p.257

after years of sobriety, Knapp wrote, "When you're actively alcoholic, you don't bother to solve problems, even petty ones, in part because you have no faith in your ability to make changes…You begin to feel you're trapped in quicksand…You get so used to being a passive participant in your own life…"[187]. What this reflects is *Knapp's recognition* that the meaningfulness she thought she had found in drink was a counterfeit or inauthentic version of the meaningfulness she (the authentic Caroline Knapp) had intended on finding.

In saying this Knapp is contradicting the view she held, transiently, when she was drinking. At times she believed that she was drinking to bring out her authentic-self. But the discontinuity of this feeling, the fact that at other times she seemed to know (or suspect) that the passion for drinking was not consistent with the meaning sought by her authentic-self is *evidence for* the disordered nature of the passion that I am arguing can explain some cases of addiction. Even early in Knapp's drinking life the ingredients for doubts about whether drinking was *suppressing* the authentic Caroline Knapp were there. Knapp wrote:

> I think I understood in that instant I had created two versions of myself… In between, for five or ten minutes at a stretch, the real version would emerge: the fearful version, tense and dishonest and uncertain. I rarely allowed her to emerge for long. Work…kept her distracted and submerged during the day. And drink –anesthetizing and constant- kept her too numb to feel at night.[188]

I am taking up a line of thought that is related to Harry Frankfurt's position about wholeheartedness and volition. In an address to the American Philosophical Association

[187] Knapp, p.260, 1995

[188] Ibid, p.17

Frankfurt said, "Wholeheartedness does not require that a person be altogether untroubled by inner opposition to his will. It just requires that, with respect to any such conflict, he himself be fully resolved."[189] What Frankfurt is saying in his address is that one loses authority over the volitional self when one cannot hold fast to a certain position, in the face of conflict, regarding what one wants to be their will.

This is a relevant parallel to what is occurring with the disordered passion of the addict. The addict is deeply conflicted when they experience this type of disordered passion. They lose authority over themselves and what they stand behind (the passion or sobriety). This makes the addict like someone who is not wholehearted. The addict is unsure which side he or she ultimately comes down on, and so they lose themselves in their passion for their addiction.

It is worth noting that the losing of one's authentic self, due to disordered addictive passion for a drug, *is* quite different from those who depend on a drug (e.g., an antidepressant or and antipsychotic) as medication to alleviate a pathology. In cases where a psychopathology is being treated the effects of the drugs may be transient (and meet some of the criteria of an addictive disordered passion) but in these cases the drugs in fact aid the person in finding authentic meaningfulness in their lives by silencing the disease which presumably made meaningfulness difficult. This is a critical difference between the two cases.[190]

[189] Frankfurt, p.9, 1992

[190] I am grateful to Agnieszka Jaworska for bringing this concern to my attention.

In closing, I believe Seeburger described this type of disordering of addictive passion well. I do not think losing oneself in a passion necessarily makes a passion disordered, as long as what the authentic agent wants results from pursuing the passion. However, time and again the agent acts on the addictive passion and what comes out of the pursuit harms what the agent is seeking, e*specially* if what they were originally seeking was to themselves be the author and owner of their own creativity, success, or inner-peace.[191]

§3. Conclusion

A passion is disordered when it exhibits long term control over an agent in a way that prevents the agent from being properly reasons responsive or when the agent's authentic-self or goals are subverted by the passion. This is what one finds in the case of addictive disordered passions. The passion for a substance or behavior can grow to such strength as to simply push aside (or cause one to ignore) countervailing considerations that ought to trump it. Also, an addictive disordered passion can cause the agent to feel that the passion is a path to meaningfulness, when in fact it subverts the goals of the agent who was originally seeking meaning.

I think the nature of the true experience of the addict is often lost in routine studies with standardized questions. In this chapter I have turned to sources in which addicts tell their own stories in their own ways. Admittedly there are a limit to the

[191] I certainly leave open the possibility that an addiction could be an extension of someone's non-addicted self and thus not truly impair or unduly influence their authentic agency. Some addictions need not even negatively influence agential self-determination in any significant way (e.g. smoking comes to mind as one such sort of addiction). But some do, and that has been the point.

number of papers and memoirs written by addicts, and so there is some limit to the conclusions one can draw about how widespread disordered passion is within addiction. However, I think that it is undeniable that the pictures of addiction painted by the addicts' whose stories I discuss show a deeper side to addiction than just appetitive drive. It is with that in mind that I now turn to my final chapter in which I will partially reconcile the biological and phenomenological aspects of addiction and provide answers to the questions I raised in the introduction.

Chapter 4

The Hybrid Theory: Explanations, Answers, and Implications

In this chapter I will do three things. First, I explain the hybrid theory in some detail, showing how it pulls together the concepts of disordered appetite and passion. Next, I will answer the questions I laid out in the introduction which I intended to be a test for the strength of my theory. Finally, I look at diagnostic, legal, and moral implications of my theory.

§1. The Hybrid Theory

Addiction, understood in broad conceptual terms, is a cluster of concepts that are of at least two different *kinds and so it is a hybrid concept.* The hybrid concept is the first of two elements comprising *the hybrid theory*: the hybrid concept *requires* that in order to discuss conceptual constituents of addiction, in the abstract, one must recognize that it is a mixture of different concepts of (at least) two kinds (passion and appetite). Each is *necessary* to understanding what constitutes addiction, although the subject is so complex that I am reluctant to say that taken together they are sufficient to understand the concept of addiction.[192]

[192] Consider an example. Take competitive sports as a concept. Multiple participants and ways to record performance are necessary to understanding the concept of competitive sports. But they are not sufficient to explain the concept entirely. There must also be a way of comparing performance measures so that a victor can be determined. I feel as if something similar may be going on with the hybrid concept so I leave open the possibility of additions to the necessary constituents.

What is important to note first is that the concept of addiction is necessarily comprised of two different categories of things: appetites and passions. Think of this as somewhat similar to the concept or explanation of what it is to be an amphibian. The concept of an amphibian is the concept of a creature whose life cycle is comprised of two distinct stages. An amphibian starts life as an aquatic dweller, with gills -as a tadpole (for example). If it survives to maturity it will become a land dwelling creature, with lungs -a frog (for example). The *hybrid concept* can be understood loosely in the same way. Understanding what it means to be an amphibian is to recognize that two distinct and different types of existence (one aquatic, one terrestrial) are contained in the concept. To address the concept of addiction requires recognizing that there are (at least) two distinct types of disorder (and the sub-types of each) that are contained in the concept.

That being said there is a second layer to the hybrid theory, which has to do with the way addiction presents itself in the individual addicts. First off, I believe that any single exemplar of addictive disordered appetite or passion is *sufficient* to constitute addiction in an individual. I have made it plain throughout the dissertation that one may encounter an addict with only one sort of disordered appetite. Just as a tadpole is an amphibian (even though it has not yet, and may never, become a land dweller), so too an individual can be an addict by only exhibiting one of the cluster of concepts that comprise the hybrid concept of addiction. Unlike with the broader concept of addiction, instantiations are much narrower. The reverse of what is true for the broad concept of

addiction is true for individuals. Disordered appetite or passion alone is sufficient for an individual to be an addict, but neither is by itself necessary.

But how can it be that disordered passions and appetites are not sufficient to constitute the general concept of addiction, but are sufficient to account for addiction in the individual? Suppose one wanted to explain what constituted the concept of the nuclear family in the United States in 1955.[193] Necessary to that concept would be the constitutive elements of a mother/wife and her offspring. However, that would not be sufficient for completely understanding the concept of the nuclear family (since the nuclear family in quaint old 1955 would also involve a father/husband). So being a child would be sufficient for concluding that an individual is a member of a nuclear family, but would not be sufficient, only necessary, to explain the concept of a nuclear family. I believe something similar is going on with respect to how the ideas of disordered appetite and passion relate to the concept of addiction as necessary conditions, yet are sufficient conditions for including individuals within the category of addiction.

This is how, for instance I account for mouse and rat addiction. It is impossible by my lights for a mouse to have a disordered passion of the sort discussed in chapter 3. A mouse is not "reasons responsive" in the way I mean it in that chapter, nor can a mouse set long-term meaningful goals for its life. Yet a mouse can be addicted to something like heroin or nicotine. All the mouse needs to do is participate in or

[193] I am falsely but usefully imagining that the average family of that falsely quaint time period was comprised of a father, mother, and a couple of children (say a boy and a girl). Although the definition of a nuclear family just is the parents and their offspring. Today that can cover any number of combinations of parents and offspring. I think those are nuclear families as well, I just choose to simplify my example for discussion sake.

instantiate one member of the cluster of concepts that comprise addiction (in this case a disordered appetite). A tadpole is an instantiation of an amphibian even though it may never live out all the stages that conceptual account for what it is to be an amphibian (e.g., it might be eaten by a fish).

I believe that addictive disordered passion is also sufficient to include an individual in the category of addict. Suppose a man has an extremely well regulated use of nicotine. He may have an ordered appetite for it, and use it only on rare occasions (say the odd weekend drinking with friends at a bar, or the occasional cigarette as a way to unwind after work). But suppose that in this person's family there is an extensive history of heart disease and lung cancer. Someone could rightly point out that for him even modest amounts of smoking (non-disordered appetitive levels or even non-appetitive levels) greatly increase their risk of cancer, heart disease, and dying young. If the smoker feels passion (it need not necessarily be cherishing, just a passion) for the pleasure that his occasional use of cigarettes bring and for this reason alone ignores warnings about health effects, then he may be an addict for reason of disordered passion alone. This might also be true for someone who believes the occasional use of peyote, or something similar, provides existentially meaningful revelations when in fact all it does is serve to depress them in the long run and make them feel that life is in fact meaningless.

With this in mind I will move on to the second layer of the hybrid theory. Properly stated the second layer to the hybrid theory is the concept of *hybrid instantiation*. I believe that one can find individual addicts that manifest both a

disordered appetite and a disordered passion at the same time. This contributes greatly to the complexity and strength of addiction as it is instantiated in persons especially. Addiction in the individual addict may be as complex as a *hybrid* of several of the disorders discussed in the earlier chapters and those disorders may come from the two distinct categories discussed (appetite and passion). In such a case we see a hybrid instantiation of addiction with an individual having both a disordered passion(s) and a disordered appetite(s). Hybrid instantiation is the second, and final, component of the hybrid theory. Thus *the hybrid theory itself* is making two fairly straight forward claims: First, in understanding the concept of addiction in general it is *necessary* that one recognize that addiction is comprised of disordered appetites and passions; Second, that an addiction in an individual may be instantiated as a wide variety of combinations of the cluster of concepts associated with addictive disordered appetites and passions.

One question naturally arises about an earlier example: The aquatic stage of the amphibian gives rise to the terrestrial stage. Is there something similar occurring between appetite and passion? I cannot answer this question as this point in time. The similarities I am drawing are conceptual not causal. Appetite and passion may causally interact, although I leave the question of causal interaction for another time. I must admit that I suspect that disordered appetites can cause disordered passions, although I am not yet prepared to defend that position.

The point here is that according to the hybrid theory there is great complexity to addiction at both the abstract level and at the level of the individual addict. Perhaps one way to capture the interdisciplinary importance of the hybrid theory is to borrow an

idea from George Vaillant, professor of psychiatry at Harvard University, and an expert on alcoholism. Vaillant wrote of alcoholism, "Just as light can consist of both waves and particles…alcoholism…can simultaneously reflect both a conditioned habit and a disease."[194] Valliant is thinking about alcoholism in slightly different terms than I am, but it is the parallel with light that I wish to draw attention to. Light has both particle and wave-like qualities, at the same time. I think Vaillant, with his comparison of alcoholism's structure and light, is tracking something that aids in understanding the importance of both the hybrid theory and the interdisciplinary approach it purports to take.

According to the hybrid theory addiction broadly construed is necessarily a cluster of different concepts of two kinds, and individually it can be manifested in a simple or compound way. Disciplines like biology, or psychology, or philosophy have each picked up on different elements of addiction. In the early years of experimentation with light some believed it to be a particle and some believed it to be a wave because of their particular approaches or experiments. I believe a similar thing is occurring with addiction studies.[195]

In the case of light both sides turned out to be correct, but independently their views were incomplete because they approached the question from only a particular angle (in this case particular types of experiments). Similarly I believe that addiction, *as a whole or in all particular cases*, cannot be entirely understood from only the

[194] Valliant, p.308, 1983

[195] Issac Newton fell on one side of this debate because of his work with prisms. He conceptualized light as particles. Christiaan Huygens was a contemporary who believed light was a wave.

biological, or psychological, or philosophical perspectives. All may turn out to have something to contribute. Individually they probably are picking up on different properties of addiction that are accurate, but they are not integrating their findings with the other disciplines.

Specific disciplinary approaches (e.g. molecular biology) generally track a particular aspect of addiction.[196] However, by just scratching the surface of an interdisciplinary approach, by considering the phenomenological (and philosophical) characteristics of addiction along with some of its biological characteristics, a picture of something that can be two distinct things at once, or at different times, seems to emerge.

There is some dis-analogy between what physics discovered about light and what I am claiming is true of addiction. Whereas light is in all individual cases both a wave and a particle, addiction is not necessarily in all individual cases (all instantiations) both a disordered appetite and a disordered passion. As I pointed out earlier in the case of mouse or rat addiction it probably only makes sense to talk about the addiction as a disordered appetite. There is another dis-analogy between the example of light and addiction. As far as I am aware light's wave properties do not cause its particle properties or vice-versa. On my hybrid *view I leave open the possibility* that disordered appetite can cause, or at least might have explanatory relevance with respect to a disordered passion (the reverse might also be true).

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[196] For a discussion of various technical (and I think crude) definitions of addiction please see the introduction to this dissertation.

What the hybrid theory is meant to emphasize is that addiction can present more than one face at the same time in the individual addict, and that each face bears some scar or disordering of its features. However, the hybrid theory is entirely compatible with a particular instantiation of addiction being accounted for by only one of the cluster of concepts. As an example, the dual (wave and particle) nature of light is unnecessary to explain the photoelectric effect (one can explain the photoelectric effect by invoking only the particle account of light) the dual nature of addiction is not necessary explain mouse addiction.

A 'grand theory' of addiction requires an interdisciplinary approach. My hope in approaching addiction in the way I have is to galvanize movement beyond the state of discussion that physicists were in about light in the 17th century. At that time some pointed to experiments that showed light to be a wave and others could point to experiments that showed it to be a particle. The same incompleteness results from trying to account for what constitutes the general nature of addiction from just one experimental or analytical perspective. To try and do so misses the richness contained in the concept.

This is why I *do not* wish to entirely discount the possibility of additional members of the cluster and perhaps even a different kinds or categories of members belonging to the general concept of addiction (i.e., the hybrid concept, not the hybrid theory). I want to emphasize that as a concept addiction has at least two major explanatory elements that fall into two distinct categories: neurobiological and phenomenological. For the individual addict I have argued that either is sufficient for

addiction, and there is no reason to think the two cannot be contemporaries. Indeed, Jerry Stahl and William Burroughs both talk about metaphorical 'hunger' for heroin.[197] Knapp at times discusses alcohol in a similar light.[198]

Other theories of addiction (e.g. opponent process theory, or delay-discounting theory) are utterly inadequate as *complete* theories, although they *might well pick up on aspects* of addiction which play a role in disordered appetite or disordered passion.[199] I think that other theories, in so far as they have merit, will be included within the hybrid theory in some manner, even if only in a supplementary role. At the same time the hybrid theory is *not guilty of open ended inclusivity*. In so far as a theory is inconsistent with the general concept of addiction being comprised of both disordered passion and disordered appetite that theory must be wrong. The hybrid theory can also put existing theories of addiction in their place, that is, it is a test of their relative merit (e.g. hyperbolic discounting seems less important to explaining addiction than does the matching law proposed by Herrnstein, hyperbolic discounting only reinforces tendencies that lead to disordered appetite, matching explains their acquisition).

I hasten to add that my hybrid theory is *not* adequate as a complete theory of addiction. There might be other core elements to the hybrid concept. For instance, I did

[197] See Stahl, 1995, and Burroughs, 1977.

[198] See Knapp, 1996.

[199] It would not be surprising to find out that fear of withdrawal (i.e. part of the opponent process theory) plays a role in the disordering of the appetite in the sense that such fear would make other appetitive and non-appetitive goods unappealing due to the pain of withdrawal (i.e. perhaps the pain of withdrawal is bad in itself, but that pain partially *consists in* leaving the addict with no alternative goods to pursue). Likewise delay discounting might play a role (although not a necessary role) in disordered passions of the first sort. If someone tends to favor more immediate goods that would certainly act to reinforce their passion for an addictive good vis-à-vis a more distant non-addictive good.

not consider a sociological analysis of addiction. It may turn out that one of the critical features of a complete explanation of the concept of addiction is a feature revolving around how we relate to one another and to social norms. If something like this is the case, then my own theory is only a partial theory (but closer to being complete than others).

Since the hybrid theory has an impact on how we are to understand addiction it also has practical implications for how we diagnose and treat addicts, as well as for why and to what degree we hold them morally responsible. I will turn to these implications momentarily, but first I wish to answer the questions I set out in the introduction as a test for the coherency and usefulness of my theory.

§2. The Test Questions

In the introduction I presented seven questions as a means for testing my theory. I think I provided answers to the first two questions in chapter two and do not see any reason to repeat the answers, especially since it would take several pages to do so. The questions were: 1) What distinguishes addictive (disordered) appetites from non-addictive ones?; and 2) What biological mechanisms perpetuate the wanting and decision making that sustains addiction, often in the face of negative consequences, and how do they account for a disordered appetite? In chapter two I attempted to explain the difference between addictive disordered appetites, non-disordered appetites, and appetites that were perhaps disordered but not of an addictive nature. I also attempted to explain the functional mechanisms (e.g. wanting arising from reward center stimulation) which

put pressure on behavioral inhibitory mechanisms (e.g. the will or other higher order cognitive mechanisms) and caused the strong tendency in the addict to favor repeatedly taking up an addictive substance. [200]

In the introduction I ask a third, more complex question (in two parts), to which I do not think a direct answer can be found in an earlier chapter. The first part of the question was this: How can one account for the diversity of substances and behaviors of addiction and the phenomena they produce? The diversity of substances and behaviors to which one can be addicted is explained by both the seemingly universal importance of dopamine in helping to establish appetites and the ability of a person to acquire a disordered passion.

For those reasons I leave open a wide range of possible substances and behaviors to which an individual may become addicted. I do this because what constitutes addiction, while not itself idiosyncratic, may have idiosyncratic origins. It is not uncommon, for instance, for some people to feel little or no effect from certain types of benzodiazepines (the class of sedatives to which Owen Flanagan was addicted). Yet Flanagan felt more than enough effect to become an addict to this class of drugs, and no one would have any problem accepting that he could be addicted to benzodiazepines.[201]

Applying the same thinking to substances and behaviors at large there seems little

[200] A good friend of mine, while still an undergraduate, once had to make a late night choice. He had only 5 dollars cash and no credit or debit cards. He was both hungry (with literally no food in his apartment, only condiments) and running out of cigarettes. He went to a convenience store thinking he would get something to eat, he left the store after purchasing a pack of cigarettes and no food.

[201] The FDA has most benzodiazepines listed as schedule 4 controlled substances meaning there is mild to moderate risk of physical and psychological dependence. However, there is little dispute that benzodiazepines are addictive and often are a component of complex addictions (i.e. addictions involving more than one substance).

reason to constrain what may or may not be an apt candidate for an addictive substance or behavior. Just because some or even most people will not or cannot become addicted to some substance or process does not mean it cannot be an addictive substance for someone else. Shopping, or using the internet seem unusual or unlikely candidates for being addictive substances but that does not seem any reason to eliminate them as being something to which someone may become addicted. The unusualness or unlikelihood that some object or process may be the source of an addiction is meaningless in determining whether or not it is an object of addiction. What matters is the presence of an addictive disordered appetite or passion. Benzodiazepines would not cause addiction in some subset of the population immune to their pharmacological activity. However, that seems irrelevant to discussing the addiction that Flanagan had to them.

I also asked the question: What unifies the different instantiations of addiction? In other words, what feature(s) makes the nicotine and heroin addicts both addicts? The *most common* unifying feature seems to be that addictions are abnormalities that result in *disorders of interference*. In chapter 2 I argued that appetite was addictively disordered because the appetite *interfered* with other appetites and non-appetitive rewards. In chapter 3 my two principal claims were that addictive disordered passions *interfered* with an agent's ability to respond to reason and an agent's ability to find authentic meaningfulness in their own lives. A heroin addict may experience several of the addictive disorders that fall within this cluster (and manifest a true hybrid addiction), whereas the nicotine addict may only fail to respond to countervailing reasons in the relevant way. What makes each person an addict is that at least one member of the cluster

of concepts applies to them.

Remember that appetites are functional, do not necessarily involve liking, but rather involve a recursive and visceral wanting for a particular thing. Passions are emotional, they are the result of reflections on impressions, and do not have quite the same visceral nature as appetite. Recall my example of the regulated smoker who may have a passion for the stress relief cigarettes provide (e.g. they might cherish the breaks and relaxation smoking provides) and thus not be open to countervailing reasons (e.g., you are much more likely to die young) to give up their smoking. A heroin addict may not be driven by passion but simply by an appetitive disorder (e.g., he cannot eat or function normally without using) which makes it more challenging for him to stop looking for his next fix. At the same time one might find a heroin or nicotine addict driven by disordered appetite and passion.[202]

There seems to be an exception to the idea that the abnormality of addiction always results in some sort of interference. The type 3 appetitive addict or the "Churchill-Addict", who I claim has a disordered appetite but does not exhibit signs of interference with other pleasures or appetites appears to be an exception. But the "Churchill-Addict" has a disorder in the same sense other addicts do (although it may be benign, I will discuss this shortly). However, there is not, in this case, interference with recognizing reasons, self-determination or other goods/appetites. Someone with type 3 appetitive addiction would (=df) find it difficult to quit (after all it is a very 'healthy' *appetite* they

[202] Overall, I think both heroin and nicotine addicts can manifest the same degree of indifference to the countervailing reasons to quit that are associated with the health risks of each addiction. In other words, both seem to manifest roughly the same degree of disordered passion with respect to at least sensitivity to countervailing reasons when those reasons have to do with long term health.

have acquired) and so in some sense having this excessively large appetite does create some discomfort or inconvenience (e.g., in so far as they have an appetite they will still seek to satiate it), but not in the same way as it does with other addicts. This example forces me to concede that direct interference of other functions (of reason or appetite) is not necessarily a constituent of addiction. However, *interference* of normal functioning of other appetites, non-appetitive rewards, reasons responsiveness, or self-determination that I discuss in chapters 2 and 3 are certainly extremely common features of addiction. Yet, there may yet be some outliers that are properly included in the category of addiction (because of abnormality of appetite) without exhibiting some sort of interference.

In the introduction I also asked: How can an agent cross-temporally or simultaneously be divided against herself with respect to her addiction? As I showed in chapter 1 delay-discounting can only answer part of this question (e.g. it cannot explain the simultaneity of the desire to use and not use a drug). To answer this question I point first to my explanation of addiction as a disordered appetite. With this model the explanation of internal division is easy to give. Appetite, as I conceived of it, was functional, and drove wants, which the addict may (or may not, according to Berridge and Robinson) be aware of, however higher order cognitive processes were also built into my picture. Presumably anyone driven by a strong appetite could want to be, at a conscious level, strongly against indulging even though at the appetitive level they are motivated to indulge. As an example it happens all the time with people who are dieting! The same conflict they feel is the same type that is occurring with the addict, only in the addict's case the appetite one is trying to suppress is disordered and so especially strong

(it may be disordered in some cases of dieting). The conflict the addict feels between appetitive motivation and cognitive attempts to suppress that motivation can occur simultaneously, or what one favors may alternate. The causes of this fluctuation need not be some standing disposition to view the future in a particular way, but can be explained more simply in terms of changes in body chemistry and satiation levels.

The phenomenology discussed in chapter 3 can also be used to explain the addict's internal division. The addicts whose stories I considered in chapter 3 felt the importance of their addictive substance, and yet tended to realize the dark place addiction had taken them and that the thing they cared deeply for was also leading them to ruin. Each went through a series of 'pushes' and 'pulls'. The evidence from that chapter suggests that the complexity and clash of passions and thoughts is enough to explain how someone might both feel desire and revulsion toward the same thing at once, or cross-temporally. My account of the addict's internal division is a simpler and more naturalistic explanation (unlike delay-discounting, which relies on a specific cognitive process) and it relies only on the complexity of the human mind and the ability of that mind to recognize desirable and undesirable aspects of an additive good. Such recognition seems almost a brute fact about human capability.

The next question I presented in the introduction is, in my view, one of the hardest to answer. That question is this: What accounts for relapse and how does the concept of relapse inform our understanding of remission (or the boundaries) of addiction? Here is where the hybrid theory shows a great deal of promise. Unlike theories of addiction that rely on singular mechanisms as the primary causes of relapse there are

several mechanisms of relapse for which the hybrid theory can account.

Before going on I want to introduce the concept of the addict in remission, or the non-active addict. In the technical literature, such as the DSM-IV, there are different classifications of remission based on the amount of time without use of the substances to which one was addicted, or the use of any addictive substance.[203] For my purposes I define an addict in remission as one who ceases to exhibit any addictive passions or appetites within a disordered range, but who once did.

Before turning to my own hybrid theory consider the limitations, with respect to this question, of the melioration theory proposed by Herrnstein and Prelec and the visceral factors theory put forward by Loewenstein. In the case of the former, it seems difficult to comprehend why an addict who has overcome the physical element of addiction, and is no longer matching average utility of choice around a low level of utility, would return to using. In fact, on their theory the addict should almost never return to using a substance of addiction once his non-addictive choices have returned to normal levels of utility (since the addict knows where addictive substances can lead). But that does not address the pull and desirability of the addictive substance in its own right, nor can it explain why after years of sobriety an addict can relapse.

In the case of Loewenstein's theory I believe there is difficulty in accounting for why novel life stressors, or other non-cue conditioned stimulus, might cause an addict to relapse. Loewenstein surely does not maintain that cue-conditioning is the only mechanism of relapse (I agree that it is one of the mechanisms). Yet it is the only one for

[203] Please see Morrison, 1995.

which his theory clearly provides (although it is open-ended and could *accommodate* others). As an example, Loewenstein is silent about the possibility that relapse can occur in contexts devoid of any cues associated with an addictive drug or behavior.[204] My theory can accommodate Loewenstein as well as different mechanisms of relapse, as I will show momentarily.

I lack the (biological) background to explain how appetites are permanently extinguished, but it would make sense (given my definition of appetite in chapter 2) to think of appetites as robust things. By that I mean the functional mechanisms that drove the appetite are at least subtly retained in the brain's wiring for long periods of time, even after indulgence in the appetite has ceased. Let us suppose that after a long rehabilitation an addict's appetite for their addictive substance of choice *ceases to occur within a disordered range* (i.e. they go into remission). If I am correct and appetites are robust (meaning that the disposition to reacquire the appetite remains reflected in the brain's wiring) it seems reasonable to think that the *disposition* to want (which, in part, constitutes the appetite) a particular drug or to engage in a particular behavior remains, and that so long as that disposition remains there may well be *occurrences* of such wants.

Further, it is not hard to imagine that the strong salience (by which I mean the strength of desirability of a reward) once associated with the occurring want associated with an addictive disordered appetite could reoccur under certain conditions -cue-conditioned stimulus being among those conditions. If an addict in remission begins to

[204] See Tate, et al, 2004.I discuss the different contexts of relapse later in this chapter.

engage in addictive behavior again, then it seems that it would be easy for the brain to readapt to the old addictive wiring (the wiring is already 'in place', so to speak, and need only be reattached). My hybrid theory supports this idea, so I am sympathetic to Loewenstein's cue-conditioned account of relapse. However, the hybrid theory is an improvement over Loewenstein's account because it provides more detail (especially the distinction between dispositional wants and occurrence of wants) as to the internal mechanisms of relapse than does his explanation.

But the hybrid theory in general is superior to Loewenstein's account, or any other such account, which relies primarily on the retention of a physical disposition to want an addictive substance as the source of relapse. The hybrid theory shows that addiction involves passion and some form of concern or care for the addictive substance. Suppose an addict no longer experiences his or her passion within a disordered range (i.e. they are in remission). However, assume that passions, like appetites, can be robust and not entirely extinguished even if they are significantly diminished. The idea of rekindling passion, especially after long absence, is an old one. Francis Seeburger seems to be picking up on this in making the observation that," The non-addictive mind is the detached mind….To be freed from an addiction is to be brought into detachment toward the object of addiction, to be released, in Heidegger's sense, toward it."[205]

[205] Seeburger, p.172-73, 1996. Seeburger uses detachment in the sense that Martin Heidegger uses the term "releasement". Essentially it is a genuine freedom that results from neither trying to control something, nor being dominated by it.

Many addicts can overcome physical withdrawal repeatedly, as both Jerry Stahl and William Burroughs did, but cannot become detached from the passion they have cultivated for their addictive substance. In such cases relapse can come quickly, or it can come after months or years of sobriety. As Stahl put it he felt like a battered wife going back to an abusive husband because when he (heroin) treated her (Stahl) well everything was wonderful. Recall also that –during a period when he was clean- he expressed fear that without heroin he could no longer be an effective writer. His pull toward the drug certainly seems to involve more than stimulation of his appetite, but a genuine concern about his ability to live life without heroin. He appears not to have detached himself emotionally from heroin. And once he indulges in the passion again it seems a short road to it becoming a disordered passion.

William Burroughs describes an interesting instance in which the protagonist in *Junky* relapses. The protagonist expresses sentiments akin to rekindling a relationship with a lover from the past. From *Junky*:

> "I was in the can," he said. "Anyway, I didn't want to come around because I knew you was off [heroin and morphine]. You off completely?"
> "Yeah, I'm off."
> "You wouldn't want a shot, then?" Old Ike was smiling.
> "Well…" I felt a touch of the old excitement like meeting someone you use to go to bed with and suddenly the excitement is there and you both know that you are going to bed again.[206]

It is important that the hybrid theory accounts for relapse of this sort. If my supposition is correct and appetites and passions are about equally robust, then each is equally difficult to extinguish. If this is true, then relapse triggers can originate from the

[206] Burroughs, p.118, 1977; Here Burroughs might be manifesting both an appetite and a passion but it is worth noting the simile he uses to describe the temptation to relapse is one that closely tracks passion.

deeper emotional structure the addict developed in his or her relationship with an addictive substance, not just cue-conditioning. A rekindled feeling of love for an addictive substance in the guise of a lover can cause relapse.

This analysis is actually borne out by studies that show co-morbid mental disorders (such as depression and PTSD) can lead to contexts of relapse which differ from those without co-morbid psychological disorders.[207] This would seem to suggest that the role of an addictive substance as a soother of existential angst (i.e. the thing that makes its appeal passionate, given the evidence from chapter 3) is just as much a trigger of relapse as more externally based cue-conditioning. From a 2004 study on the context of relapse the authors' concluded:

> …SUD-PSY [addict's with comorbid psychological disorders] adults reported negative affective states prior to use at higher rates than SUD [addicted without a comorbid disorder] adults. The majority of SUD individuals initially resumed use in social contexts with others present, whereas SUD-PSY participants typically first resumed substance use when alone and most often in intrapersonal/ environmental contexts…the contextual antecedencts of initial posttreatment substance use episodes differed for substance-dependent adults with concomitant PTSD or mood disorders compared to those…wthout such Axis I dsorders.[208]

If my hybrid theory is correct such a disparity results from the fact that those with depression, or anxiety, or other mental illnesses relapse because they have not learned how to detach themselves *emotionally* from their addictive substance and the things it was able to do in alleviating their psychological discomfort (cf. Knapp and Flanagan).

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[207] See Tate, et al, 2004. The study was carried out by University of California, San Diego in conjunction with the Veteran's Administration Healthcare System of San Diego. For the purposes of this study," Relapse (heavy use) was dichotomized using the following criteria: three of more consecutive days of drinking at least 6 drinks a day for men or four drinks or more for women, or using drugs, without quantity parameters." (p. 1713)

[208] Ibid, pp.1719, 1721

Something still lingers in them for the comfort of the alcohol, heroin, gambling, smoking, or cocaine, and that thing seems to be distinct from appetite. It seems more a longing, a desire to rekindle passion -perhaps from desperation- for the addictive substance that once soothed them.

One unfortunate aspect of the hybrid theory is that it makes the problem of relapse more complex. There are two, in principle, distinct causes of relapse with each having a variety of ways they might manifest themselves. Appetite driven relapse, *or the reoccurrence of wants stimulated by the brains reward mechanism*, may occur because of specific social cues or interpersonal relationships. Passion driven relapse, *or the reoccurrence of the phenomenology and emotion associated with an addictive substance*, may occur because of environmental stressors or negative affective states. I suspect that in many instances of relapse both appetite and passion play roles. What this does is make relapse extremely difficult to address from a treatment standpoint. However, this complexity and the difficulty of addressing relapse is entirely consistent with the idea that addicts often relapse, and with the fact that 6 month relapse rates for some drugs are as high as 70% (nicotine), and are between 40% and 50% for many others (opiates and alcohol).[209] A comprehensive understanding of addiction minimally recognizes that the addict in remission is liable to relapse not only because of a range of appetitive or social cues, but due to emotional circumstances as well.

The fact that both of these elements are at work means that the borders of addiction are hazy, and that precisely when (or if) one ceases to be an addict is hard to

[209]Graham and Schultz, p.311, 1998

define. It is fair to draw an *important boundary between* an active addict (one who still manifests at least one of the disorders) and an addict in remission. But this division does not appear to be a definitive dividing line, at least for purposes of the treatment of addiction, between being an addict and not being an addict, given the problem of relapse.[210] Rather, the lack of any active disorder (but having had one in the past) seems to create a special set of circumstances which require their own designating term, which still recognizes that the person in question has a special disposition to relapse, or to again manifest one of the disorders discussed in chapters 2 and 3. From this comes the idea of calling a formerly active addict an addict in remission. This broadens the category of addiction, but it does so in an important and (I think medically) helpful way.

So when does an addict stop being an addict in any sense of the term? The hybrid theory cannot provide a definitive answer to this question. The best effort that I can make is to suggest that when the dispositions to have a want for a substance are absent from the brain entirely (or have returned to pre-addicted levels) or, as Francis Seeburger suggests, one has completely detached oneself from the object of addiction, then one is no longer an addict. In such a case a person would no longer have a greater tendency, than the average person, to fall back into the disorders of appetite and passion that constituted their addiction in the first place. Unfortunately the only real test of this (whether or not the addict no longer is predisposed to return to disordered levels of use/behavior) is to attempt to experimentally use the substance (or engage in the behavior) to which one was

[210] Former addicts tend to redevelop their former quantities and methods of use extremely rapidly, far more so than a first time user could do. Jerry Stahl relates an interesting incident where it took him only a few weeks to return to prior levels of use after several months of sobriety.

addicted, and try to control it. Unfortunately, many addicts try this only to find out that they are merely in remission, and so become actively addicted again within a short period of time. Experimenting with controlled use of an addictive substance is such a risk that it is understandable (if not entirely justified) why programs like AA encourage complete and permanent abstinence.

The penultimate question I asked in the introduction was this: In what sense (if any) is addiction a disease? Before exploring an answer to the question let me provide a definition of disease used by Norman Miller, of Cornell University Medical College. In a paper discussing this topic he defines disease as, "…any deviation from or interruption of the normal structure or function of any part, organ, or system (or combination) of the body that is manifested by a characteristic set of symptoms and signs, and whose etiology, pathology and prognosis may be known or unknown."[211] Miller also proposes Koch's postulates as a more stringent definition of disease. Koch's postulates require that an agent (suspect pathogen) believed to be causing illness be isolated from an original host, transplanted to another (healthy) host, that the healthy host contract the same disease (symptomology) and that the same agent (suspect pathogen) be isolatable from the second host.[212] Dr. Miller believes that on both accounts alcoholism and drug addictions meet the definition of a disease. [213]

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[211] Miller, p.296, 1991: Miller draws this definition from *Dorland's Illustrated Medical Dictionary*, 27th edition, 1988.

[212] Ibid, p.296

[213] About the 'deviation' criteria of disease: it is not apparent from the definition given by Miller that a deviation need cause harm to a subject in order for a deviation to be considered a manifestation of disease. This seems reasonable, although a bit problematic. Let me explain why it seems reasonable. I may contract

I am not entirely sure that I agree with Miller, where Koch's postulates are concerned. Addiction is perhaps metaphorically, but not literally, a communicable illness (the kind of disease that Koch's postulates are designed to identify). But if disease is defined as the deviation or interruption with parts or systems in the body, then it seems clear that my explanation of addiction qualifies it as a disease. Each account I have given is a case of interference with or deviation from the normal functioning of some system. My analysis of addiction, both from the neurological and phenomenological points of view, revolve around addiction interfering with other appetites, non-appetitive goods, deviation of consumption levels to ranges that are well beyond what can be healthfully tolerated by similar demographic sections of the population, as well as interference with responsiveness to reasons, and the ability to find meaningfulness in one's activity. There is not a single explanation of addiction that I provide that would not meet the first criteria of disease discussed by Miller.

I think it is fair to say that addiction in all the forms I have discussed it can be classified as a disease using Miller's first definition. In chapter 2 we saw evidence that morphological changes occur in the dopamine rich cells of the Ventral Tegmental Area in heroin addicted mice.[214] In numerous studies an overabundance of dopamine and dopamine receptor sites are found to be present in the brains of addicted mice as opposed

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a virus or be exposed to some kind of radiation that gives me functional wings, or allows me to shoot spider webs from my hands and scale tall buildings by using the webs. In my judgment such strong deviations from normal function or morphology are dramatic enough to justify saying that I have a disease. It just happens to be a disease that has benign or even helpful symptomology.

[214] See Sklair-Tavron et al., 1996

to non-addicted mice.[215] The behavior of addicted mice also shows marked pathological symptomology.[216] The studies discussed in chapter 2 seem to be clear physiological and behavioral indicators of the types of deviations and interruptions with normal functioning that constitute disease.

But there is yet more reason to think addiction is a disease, beyond clear changes in morphology, function, and behavior. The words of the addicts discussed in chapter 3 point to a psychological (perhaps existential) disorder that is not in principle different from thinking associated with commonly recognized mental diseases such as severe depression and bipolar disorder. The addicts who discussed their addiction were challenged (if not overwhelmed) by a chronic and ever present (or at least ever looming) psychological state or disposition from which they found scant relief. I think the interruption of the expression of their authentic agency and their inability to routinely recognize (or recognize with sufficient force to act upon) reasons counting against their addiction make them little different from someone with chronic depression or bipolar disorder. If those disorders are considered mental diseases (or illnesses, if one prefers that term), then I see no reason why addiction should not be as well.

The disease of addiction is generally self-caused in the sense that one's own behavior leads to it (although there certainly can be exceptions to this, but they would be rare). But this is hardly an objection. Diabetes and heart disease, even many types of

[215] For a general discussion of this see Wise, 2006.

[216] See Gardner and David, 1999.

cancer are caused, or the etiology is significantly contributed to, by an individual's behavior. This does not make any of them any less of a disease.

Interestingly, I think this implies that addicts, in standard cases, are responsible for their disease, since the addiction can be traced back to their own free behavior (just as someone who eats nothing but fried foods is responsible for their congestive heart failure). In so far as an active addict fails to respond to reasons, to which they are intellectually capable of responding, simply because they are ignored (e.g., Stahl simply refusing to think about the danger he put his daughter in) I think the addict can be held responsible. Only in what I take to be relatively rare cases (e.g., instances were drug abuse has caused severe cognitive erosion) do I think the disease is strong enough to largely mitigate responsibility (even though there will be some responsibility traceable to earlier behavior). I will return to this discussion momentarily.

In any event, I suspect a reasonable definition of disease will include most of the disorders of passion and appetite I discuss within the disease matrix. I must concede that the third form of disordered appetite might not be a disease under all definitions because in the individual with that kind of disorder there is no particular harm caused, other than the burden of an extremely large appetite (e.g. some reasonable definition of disease might include a harm requirement, in such cases a benign tumor might not count as a disease. I consider disordered appetite type three to be the benign tumor of addiction). That notwithstanding, the majority (i.e. the standard) cases of addiction that manifest the types of disorder I discuss should be expected to meet the criteria for disease.

The last question asked in the introduction was about how one might hope to overcome addiction. The answer to this is not clear. However, the hybrid theory suggests that the potential complexity of addiction is such that the more individualized an approach to treating addiction the more likely one is to overcome it (i.e. at least become an addict in remission). Some addicts may manifest more of a disordered appetite than a disordered passion. In such cases pharmacological treatment and behavioral conditioning might be the best approach for them. Other addicts may show a marked disordered passion as the major element of their addiction. In such cases psychological help and talk therapy used to look at underlying concerns and deeper emotional issues may be more important in attacking the addiction.

While the complexity of addiction suggests more individualized approaches to treatment will be more successful, it also means that addicts, and those treating them, must be cautious in identifying more individualized sources of relapse. I suspect that addiction has to this point proved (statistically) such a difficult thing to overcome because addicts (and those treating them) fail to guard against the multiple sources of relapse. Cue-conditioning, negative affective states (e.g. depression), boredom (anhedonia with respect to other goods), unmotivated reoccurring wants, forgetting of consequences, feelings of loneliness or stress, inability to accept never again engaging with the addictive substance (passionate longing), and probably numerous other conditions lead to relapse, or the inability to permanently overcome the disordered elements of addiction.

The unfortunate news is that for most addicts, once they have overcome active addiction, they are –for quite some time- in the position of a sentry on guard at a forward operating base in enemy territory. They must be ever alert and vigil. If the enemy in this case is metaphorically the drug or behavior to which they were addicted they must accept that the enemy is liable to approach at any moment. If they are lucky that will not happen, but an addict should expect to get lucky no more than the soldier. I suspect that it is the camaraderie of fellow addicts that make 12 step programs like AA successful (to the extent they are), just as it is the tight bonds between soldiers that makes serving in a forward operating base tolerable. However, both the addict and the soldier must be vigilant and accept that the danger is ever present, and can come from any direction, at any time.

There is one significant difference between a soldier and an addict: while the soldier has trained to be vigilant the addict has not. So the addict must find the strength to stand guard against myriad sources and types of temptation, despite having long been conditioned to give into temptation. It is an odd position in which to be, dumped into a scenario in which all of one's immediately preceding experiences have worked against preparing one. Jerry Stahl captures this beautifully:

> The horrific part about getting clean is that at the weakest point in your life you're required to be the strongest. Your nerves are shot, you can't sleep, your brain's still woozy, your pockets are empty, and some combination of fear and detox and naked unrelenting pressure still has you sweating buckets and puking on street corners, but you have to be more together than you've ever been. You have no choice.[217]

[217] Stahl, p.312, 1995

§3. Further Implications of the Hybrid Theory and a Conclusion

3.1. Improvements in Diagnostic Tools

One clear implication of the hybrid theory is that the diagnostic tools used to determine whether one is an addict require improvement. The standards in the DSM-IV, for instance, are incapable of individualizing the nature of a particular addict's addiction. A good diagnostic tool should be capable of discriminating between addictions that are primarily appetitive, primarily the result of disordered passion, or in what combination the two are present. The ICD-10, which focuses less on cultural norms and more on the physical problems associated with addiction has the same limitation. It does not nuance the diagnosis across the appetitive and phenomenological disorders.

An ideal diagnostic tool for addiction would involve three things. First, it would require a preliminary diagnostic tool, a role which the DSM-IV might fill nicely. Secondary to that a detailed questionnaire (or interview with specific questions) that would evaluate the strength of the appetitive element of the addiction should be undertaken. Third, the addict should discuss with a mental health professional the history of their drug intake, especially their early impressions of why the drug to which they became addict seemed appealing. Taken together such a three step process would give a richer and more detailed picture of the nature of an individual addict's disease.

The suggestion I am making is not that current diagnostic tools are useless, indeed they are useful, but the hybrid theory suggests serious limits to their diagnostic power. Of course, multi-step, detailed procedures are probably not economically feasible for the vast majority of addicts. I have no way of accounting (economically) for how to make a

more complex diagnostic procedure widely available. However, the best diagnostic procedure will be one which takes a 3 dimensional image of the addiction (a sort of MRI of addiction). First it will make a general diagnosis, it should then determine the particular strength of the appetitive and passionate elements of the addiction, and finally it should account for how these appetites and passions relate to one another (if they do).

3.2. Morality, Autonomy, and Addiction

I believe that my account of addiction is, in general, morally neutral (about addiction itself, i.e. addiction is not necessarily normatively bad). The fact that addiction usually involves disorders does not mean that it is in and of itself a morally bad thing. This implication of my theory is worth considering because it aligns with an interesting thought experiment proposed by Gary Watson. Watson believes that, "We cannot dismiss a regulated devotion to tobacco or drink as demeaning or enslaving just on the grounds that it involves dependence."[218] I have at no point claimed that addiction is *necessarily* enslaving, demeaning, or requires the loss of autonomy. Although in most cases it does result in these things, especially when manifested as a disordered passion of the second type, it does not necessarily result in enslavement or demeaning of the person. Type 3 disordered appetites are proof of this.

My analysis of addiction can account for the following intriguing thought experiment proposed by Watson:

> …imagine that a certain severely addictive substance, S…in a certain culture, Otherwise similar to ours…S is not only tolerated but respected as highly spiritual beneficial. This culture regards the dependency on this substance, which is to say,

[218] Watson, p.18, 1999b, notice that Watson uses the word regulated, this might imply a healthy appetite for a substance which I would not even consider an addiction.

> the vulnerability to various kinds of diminished self-control, as a small price to pay for the enrichment of human life provided by S.
> This fantasy makes it clear that the moral significance of an individual's volitional vulnerability depends not only on individual responsibility…but also on the judgments about the meaning and the value of the behavior…In our imagined society, both the use of and dependence on S are regarded as entirely fitting and normal, on a par with appetites for food and drink…The threat of being deprived of one's S is here on par with the prospect of imminent starvation.

On my analysis such a model of addiction, one where it is either morally neutral or morally positive, is completely possible. Yet in Watson's imagined society at least two of the *disorders* of appetite I use to pick out addiction can be present. It seems that in the imaginary society, S would almost certainly detract or interfere with non-appetitive goods, since S is used with enough frequency to engender serious dependence. Given the critical role of S that Watson proposes it seems that the attractiveness of non-appetitive goods which do not involve S, like spending time with friends when not on S, or playing games with family while S free, would seem less appealing or rewarding were it not for the availability of S. Starving makes discourse with a friend unappealing, because its appetitive demands weigh heavily on the one who is starving. I see no reason to think that the same limitations on enjoyment would not hold for members of this society when they are starving for S.

Second, S is clearly an *acquired* competitor with core appetites that are necessary for survival, such for those for food and drink. If S is capable of trumping these core appetites, which Watson seems to concede that it can (since being without S is akin to starving), then the acquired appetite for S is disordered. That does not mean it is morally bad, it just means that the people in the imaginary society must live with an additional appetite which is disordered because it is liable to interfere with the core appetites

necessary to their survival. If they believe the spiritual benefits outweigh the burdens placed on them by the disordering of their appetites, then they are morally and autonomously no worse for the wear.

The members of Watson's society are *willing addicts* who find themselves in a normative situation where their addiction is readily accepted. I can accept this claim without giving up my hybrid theory of addiction, or my account of it as a disease. The acceptability of S does not free them from having disordered appetites of the first and second kind. Perhaps their passion for S is such that they may be unwilling to be swayed by countervailing reasons against using S (e.g. attempts to prove there are no good grounds to believe people have spirits and so spirituality as they understand it is nonsense). However, I am perfectly willing to accept that the disordering of appetite (and perhaps passion) does *not entail* that one is enslaved or demeaned by one's dependence in a morally objectionable way. Addicts in this imaginary society do carry with them the burdens of their disordered appetites, but they might properly judge those burdens to be worth it. I leave the existence of such a society an open possibility.[219]

3.3. Moral and Legal Responsibility

One of the most difficult issues surrounding addiction is the degree to which addicts should be held morally and legally responsible for immoral or illegal actions that

[219] I think we have our own version of S (and a similar scenario) in the form of morphine or the more powerful fentanyl when given in large doses to people in severe chronic pain (who are also usually terminally ill). Given my explanation of addiction it would be fine to call such people addicts but we would probably judge, given the alternative of intense and chronic pain, the interference that the morphine or fentanyl causes with their other appetites or with non-appetitive pleasures is worth it, since the chronic and intense pain would interfere with those things anyway (and possibly to a greater degree). Of course, my definition of interference is with a baseline range of normal functioning, so special conditions like chronic pain do not mean the morphine user is not an addict because the pain they are avoiding would interfere with their core appetites and non-appetitive pleasures to a greater degree. They are still an addict because the morphine or fentanyl does interfere with their ability to enjoy other non-appetitive pleasures.

are motivated by their addiction. I do not intend to work out in any detail what can be inferred from the hybrid theory in this regard. However, I do intend to gesture at both moral and legal considerations that should be made in light of the hybrid theory.

Addicts, acting as a result of their addiction, are not freed from moral responsibility under the hybrid theory. As I suggested earlier I think the hybrid theory must lead one to believe that addicts are responsible for their behavior in so far as it is traceable to their own free past choices or when the addicts failure to respond to reasons is not due to an incapability as much as a willful ignorance or failure to focus on the result of the behavior their addiction engenders. I am inclined to think that for these reasons addicts can be held morally responsible, although their blameworthiness (i.e., the severity of punishment and sanction that is appropriate) might be reduced. Only in severe cases of cognitive impairment due to drug use (e.g. Wernicke-Korsakoff syndrome, commonly called alcoholic 'wet brain') is the addict resolved of blameworthiness, although there still may be some responsibility given the historical conditions I have discussed.[220]

With addictive disorders of both appetite and passion there are clear mechanisms, or evidence, that *most* addicts (in general) can still exert some control over themselves and their behaviors. What is less clear is how much control can be expected. There are rational inhibitory mechanisms to inhibit appetite and we expect in many instances of normal appetite that those mechanisms are operative.

[220] Wernicke-Korsakoff syndrome is actually the combination of two different pathologies that often manifest themselves together in severely chronic alcoholics. The symptoms include confusion, inability to form new memories, loss of existing memories, and hallucinations.

Consider a case: I am hungry, but that does not give me the right to eat the last pieces of pizza in the refrigerator if they belong to my roommate. However, it is more understandable that I should eat the pizza if I had not had anything to eat all day and was beginning to feel dizzy. My responsibility seems grounded in my rationality (I am capable of recognizing that it is wrong to eat my roommate's pizza without permission). My blameworthiness seems grounded in my recent history (I am broke because I spent the last of my money the day before and will not get paid until tomorrow) in addition to the fact that I was dizzy (which might mitigate my roommate's anger toward me). In such a scenario my roommate could expect that my mechanisms of rational control would stop me from eating the pizza and he is justified in his anger toward me although it is perhaps mitigated by his knowing how hungry I was at the time I ate his pizza (e.g. I would have had to wait until the next day to eat after I was paid). Only under extremely dire conditions (say I thought I was going to die if I didn't eat the pizza) should I perhaps be absolved of moral wrongdoing, or at least absolved of blameworthiness.

I think the same is true for the appetite of the addict. Under most scenarios (by that I mean most instantiations of addictive disordered appetite) it seems that we have some right to expect the addict to be able to resist his most salient wants (and their motivational force) with higher order mechanisms of inhibition. However, we can also recognize, as my roommate could, that the power of an appetite is sometimes extremely difficult to suppress and that the action of the person motivated by strong appetite is less blameworthy than that of the person driven by greed, spite, or jealously. The person who acts wrongly because of their conscious cognitive motivations is more blameworthy

because their intention to act is more closely tied to a clear considered, recognition of the ends of their action. The person who is unable to stop their more basic appetites from motivating their behavior did not intentionally select the motive of appetite (although through negligence they may have allowed it to occur), so they seem less blameworthy.

The addict who acts from disordered passion is in a similar position. Their passions are likewise motivating and resist rational inhibitory mechanisms. As with appetite, addicts with disordered passions presumably did not intentionally cultivate the disordered passion and so find themselves in a position where strong forces press on their rational inhibitory mechanisms without having intended that end.

Like disordered appetites we can have some reasonable expectation that addicts motivated by disordered passion will be able to inhibit their morally wrong behavior. After all, many of the addicts discussed in chapter 3 recognized that their passion was leading them to do things they ought not to do, or to go (metaphorically) places they ought not to go. And in some cases they showed a clear ability to resist these motivations (e.g. by going to rehab). But like the addict with a disordered appetite those motivated by strong passions will find them exceptionally hard to control. In the case of morally wrong actions of addicts motivated by disordered passion we should perhaps view them as analogous to the impassioned lover who strikes out upon finding his or her significant other in bed with another. Here again there is responsibility on the part of the addict, but it should be associated with diminished punishment or blameworthiness. Gideon Yaffe discuses a number of these issues in more nuance, from a similar perspective, in a recent

work about addiction entitled, "Lowering the Bar for Addicts". [221] Yaffe claims that the more serious the moral wrongness of the deed the addict might do, the more rightful the expectation we have that she will inhibit her behavior. However, as I noted earlier there may be exceptions to even this standard.

Legally addiction should probably be no more of an excuse than it is morally. However, as was the case with moral blame and punishment the legal system ought to take the severity and nature of an addiction into account. The legal system's expectations for addicts and the ways they can be expected to temper their behavior should be tempered by a recognition of the forces pressing against the mechanisms they have available to inhibit their behavior.

An especially interesting implication of the hybrid theory, for both moral and legal responsibility, is that not all addicts are necessarily alike with respect to what we can rightfully morally demand from them. The more complex and the more severe the addiction (i.e. if the addiction is manifested as a hybrid to a strong degree), the more pressure the addict has to engage in behavior that supports their addiction (whether it is morally wrong or not), and perhaps all the less strong is their cognitive mechanism to resist this pressure. This suggests to me that more advanced diagnostic tools could not only be used for improvements in treatment but also to determine the ability of an addict to resist addictive motivators for purposes of assessing legal responsibility and punishment.

[221] The article can be found in Graham and Poland, 2011.

3.4. Conclusion

The hybrid theory is not a comprehensive theory of addiction, and I indicated as much in the introduction of this dissertation. I recognize the limitations of my evidence. The neuroscience of addictive substances is vastly more complex than what is covered in chapter 2. Numerous downstream effects of drugs in parts of the brain other than the reward center and large numbers of neurotransmitters implicated in addictions to different substances went unexamined. Such a comprehensive examination of the neurobiology of addiction would have been impossible for me alone. The neuroscience data I examined shows that the reward center of the brain plays a necessary role in appetite, but not that it alone is sufficient for creating an appetite. Still, I maintained that when adding in the behavioral aspects of addiction it seems that -given a weight of the evidence view- the best neurobiological analysis of addiction available suggests that it is best understood as an appetite.

My phenomenological discussion was limited to those few addicts who were skilled enough to record their stories of addiction in detail and lucky enough to have survived to do it. However, I recognize that while there was some depth to my analysis of addiction as a disordered passion it is lacking in breadth. I do not see this as a problem for the conclusions I drew in chapter 3. Rather, it seems to be a problem with *the breadth of the applicability of disordered passion* as a manifestation of addiction. Full and focused narratives that center on the experience of addiction are few in number. That being noted, given that my methodology in selecting authors who had written about their experience with addiction *was simply that they had written on the subject* the similarity in

the stories seems to lend weight to my conclusion: that addiction is constituted in part by disordered passion. I think I have sufficient evidence to draw this conclusion, even if I am uncertain how broadly the conclusion applies.

Lastly, there is the hybrid theory itself, which is comprised of these two different analyses of addiction. Minimally, it is my hope that the hybrid theory will show that addiction is a complex idea, which requires an interdisciplinary approach to be fully understood, explained, and eventually treated. Reading this dissertation one should be convinced that theories of addiction which approach the topic from only one point of view will, invariably, be insufficient. Maximally, I hope that this dissertation has laid the groundwork for a comprehensive theory of addiction. That a grand edifice can arise from the foundation I have poured is probably overly optimistic. What I expect is most likely is that my efforts, occurring as they do in the midmorning of the interdisciplinary work to understand addiction, will have enough merit to be a valuable tool in the later construction of a comprehensive theory.

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