Governing desire in the biomolecular era: addiction science and the making of neurochemical subjects

A thesis submitted for the degree of Doctor of Philosophy

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Abstract

This thesis investigates the development and implications of contemporary understandings of addiction that have emerged over the last half century within biomolecular and neurobiological 'styles of thought.' The analysis, based upon historical and archival research, is organized around the shift from conceptualizations of addiction as an organic or molar disease – that is, a disease that was thought to affect individuals in some general, but unspecified way (for example, by affecting 'the will') – to neuroscience conceptualizations of addiction as a disease of the brain.

The thesis examines the interplay of cultural, political, economic, and technological factors that have influenced which particular ways of going about studying, thinking about, and researching addiction have been pursued most actively. In doing so, it brings into question the assumption that changes in styles of thinking about addiction occur as a consequence of the discovery of 'natural' neurochemical truths of the brain, independent of political rationalities, material considerations and realities, and scientific entrepreneurship.

It also investigates how neuroscience models are transforming the ways that clinical, legal, and, personal, and social problems associated with drug use and addiction are dealt with. It particularly focuses on the development and use of 'anti-craving' medications, which are today being prescribed to treat compulsive desires for a range of drug addictions, including 'behavioural addictions' such as pathological gambling and compulsive shopping. It relates these new forms of 'brain-targeting' treatment and intervention to the emergence of new classifications of mental health and illness, and to new ways of thinking about and acting upon individuals as neurochemical subjects.

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CHAPTER 1: INTRODUCTION

INTRODUCTION

> A man should pray to have right desires, before he prays that his desires may be fulfilled.
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> Plato, *Dialogues* (1990)

What sorts of desires should be pursued and gratified? Which should be avoided, or brought under control? How does one know if a desire is becoming too urgent, and what can one do about the matter? Problems of desire – questions about how best to understand, evaluate, and manage our appetites, our wishes, our motivations – were encountered by the ancient Greeks no less than they are in contemporary societies. But do we encounter, today, the same sorts of desires as Plato and his compatriots did? Certainly, there are some similarities in the objects and experiences that appear to have been desired throughout history – certain types of food and sex, for example. But what of desires themselves – their manifestation, their meaning, their nature?

This thesis suggests that the desires we have today are *not* the same sorts of desires experienced by the Greeks. It suggests that desires can only exist insofar as they are known within distinctive and historically specific ways of understanding, perceiving, and acting; thus desires, in their essence, change over time, and can only be described in terms of an historically specific ontology. And so too, the possibilities for thinking about and acting upon ourselves as subjects of desire are limited by the times and circumstances in which we find ourselves. This study contributes to an 'historical ontology' (cf. Foucault 1997a; Hacking 2002a) of desire by examining how, in contemporary societies, we have come to explain our desires in terms of biochemical processes within our brains, and how we are coming to act on ourselves in light of that knowledge. In particular, it examines the historical development of ways of

scientifically knowing and managing what is frequently taken to be the most pathological form of compulsive desire – namely, addiction.

'Addiction' is of course a highly overdetermined term with multiple meanings and connotations that vary according to specific users and settings.[1] Not only is the concept of addiction (and its partial corollary, dependence) used differently by politicians, neurologists, 'addicts,' and a range of other actors, but it may shift in meaning for any of these agents within different contexts. Discursive shifts are especially prominent in lay and political discourses, where different 'registers of meaning' are mobilized according to various aspirations (Fraser and Gordon 1997). However, this thesis only investigates conceptualizations of addiction that relate to the models and treatments of the contemporary brain sciences.

As a result of advances in a range of scientific fields over the last three or four decades, addiction has increasingly come to be viewed as a neurological condition which results from specific, molecular processes within the brain, and which is characterized by intense, pathological desires – usually referred to as cravings. While these new neuroscience understandings have yet to establish themselves definitively – that is, beyond any serious doubt – there is widespread belief among addiction researchers and professionals that they soon will. As a recent editorial in an

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[1] Even the term 'addiction,' while still widely used in scientific contexts, has been rejected by many researchers and organizations in favour of a variety of alternatives: most often 'dependence,' but also 'substance use / misuse / abuse,' 'compulsivity,' 'behavioural pathology,' 'habituation,' 'tolerance,' and so forth. And each of these terms, in turn, is subject to varying definitions and characterizations. While part of my interest is in examining how different expert and professional lexicons demarcate the territory of addiction research in different ways, I myself use the terms addiction and dependence more or less interchangeably (unless otherwise specified) to indicate a general domain of inquiry into issues regarding the contemporary science of pathological desire: persistent forms of wanting or craving that are deemed not only problematic, but are also considered to be manifestations of an underlying neurobiological disorder.

influential addiction studies journal reported: "Breakthroughs in the neurobiology, genetics and treatment of addictive disorders are generating a sense of excitement both within and outside the field" (Babor 2000: 10). This sense of excitement comes, at least in part, from the anticipation of a maturation of a 'preparadigmatic' field of addiction studies (Shaffer 1985) into an increasingly positive and objective science of addiction. While research and theorization of addiction continues in fields such as psychology and sociology, there is an implicit – and, increasingly frequent, explicit – understanding that there is a neurological basis to the phenomena such projects investigate, and that at some point in the future, psychosocial phenomena will be knowable on a more basic, biomolecular level (Heyman and Ainslie 1996; Leshner 2001; Lingford-Hughes and Nutt 2003). Because of this perceived potential for change – and because there are already clear signs that this change has begun – investigation into molecular conceptions of addiction is of paramount importance to sociological understandings of human choice, conduct, and desire.

The focus on the neuroscience of addiction helps to explain why this introduction so far has been framing this thesis in terms of an historical ontology of (pathological) desire, rather than what might appear as a more obvious alternative – namely, an historical ontology of *addiction*. The field of addiction neuroscience focuses on the study and treatment of craving, which it identifies as the essential feature of addiction; in contrast, the general field of addiction studies does not. As has just been noted, 'addiction studies' is an incredibly diverse specialty; and within this vast transdisciplinary field, problems of desire or craving have not been singled out as the most crucial feature of addiction. Rather than being represented as a problem of desire, addiction still appears as something like a 'disease of the will' – a condition

which is attributed to a lack of will-power, a deficiency of character, or a psychological maladaptation that impairs the capacities of self-control possessed by normal persons. Within such understandings the addicted subject is not thought to be afflicted by particularly urgent desires, more powerful than those experienced by 'normal' individuals; instead, the problem is that the subject suffers from deficiencies which make her unable to say no to the desires that normal people are able to control.

Thus, addiction has undergone a fundamental transformation within contemporary neuroscience representations: it is no longer attributed to a pathological personality, a problematic subjectivity, a behavioural disorder, or anything else associated with an individual's psyche or soul. Instead, addiction is explained as a neurological condition in which the brain's endogenous reward (or 'pleasure') system, which motivates all sorts of normal, essential behaviours (such as eating and having sex), becomes dysfunctionally hyperactive. The addicted brain responds too much to certain stimuli (for example, the sights and sounds associated with drug-taking), and this neurochemical overreaction is what gives rise to the cravings which are so urgent that they overwhelm an individual's abilities to think and behave normally. The essence of the problem is not that the individual herself is abnormal, but that her desires are pathologically intense; and these pathological desires are what have come to be identified as the problem in need of regulation and management in addiction treatment (with, for example, brain-targeting 'anti-craving' medications which regulate the reward system).

This study contributes to an understanding of the constitution, or making, of our present selves as neurochemical subjects of desire in a general sense – for in

contemporary societies, neuroscience knowledge about desire is applicable not only to addicts, but to all individuals. However, the study by and large focuses on the particular, historical strands of the neuroscience of addiction and pathological desire. The reason for this is simple: it is within the biosciences of addiction that craving has been scientifically studied, and that individuals have come to be understood as subjects of neurochemical desire. It has been, to a significant extent, the study of pathological desires and desiring subjects which has made possible general neuroscience explanations of both normal and abnormal impulses and motivations. Thus, in studying the neuroscience of addiction, this thesis also sheds light on the construction of normal desires (which have been defined in relation to pathological ones).

Roughly the first half of the empirical and substantive material in this thesis – Chapters 3 through 5 – focuses on providing a genealogy of the neurochemical understandings of desire by examining the theoretical, material, and political origins of today's scientific knowledge about addiction as a disease of the brain. This is a genealogy rather than a history of addiction, because the goal is not to attempt an explanation of why the neuroscience of addiction developed the way it did, but only to trace the present understandings of addiction as a disease of the brain back to particular points of origin. This genealogical investigation, in contrast with prevailing approaches to social studies of addiction and dependence (which tend to investigate the interdisciplinary field of addiction studies as a fragmented or 'preparadigmatic' whole), limits itself to tracing the emergence of a particular style of thought within addiction studies: a way of thinking about, representing, and (re)defining addiction as a fundamentally neuromolecular phenomenon.

The latter half of the substantive material of this thesis (Chapters 6 through 8) focuses more closely on the contemporary constitution and governance of neurochemical subjects of desire; that is, it examines how today's biomolecular psychiatry is providing new ways of representing and intervening on a range of personal, social, and legal problems that are based on conceptualizations of individuals in terms of their neurochemistry. While these chapters largely focus on how scientific understandings of and treatments for addiction are beginning to be deployed within the governance of 'addicted' subjects, they also examine how ideas about the brain and practices of regulating desire as a neurochemical phenomenon are beginning to inform the ways that the subjects of non-pathological desire are known, classified, and managed. These chapters demonstrate that understandings of addiction as a form of pathological desire have not only had implications for how drug-using individuals are described and treated, but also play a role in understanding desires and motivations unrelated to drug use. Indeed, they suggest that, to the extent that addiction is explained in terms of (problems with) the brain's own mechanisms, the once clear-cut distinction between the physiology of drug addictions (including alcohol) and so-called 'behavioural addictions' (such as pathological gambling and compulsive sexual behaviour) has been brought into question. And as anti-craving medications have been found to have a modulating effect on all sorts of desires – not just the most urgent, intense forms of craving that result from drug use – individuals seeking treatment for gambling problems, eating disorders, and other troubles are in fact beginning to be treated as subjects of neurochemical desire.

Ultimately, the material in this thesis provides an examination of how neurobiological conceptualizations of addiction, as they have historically developed, have figured in the constitution of new ways of explaining and managing human subjects in relation to their desires. Although there are a number of ways that this study could conceivably have been undertaken, it was loosely organized around (and inspired by) the work of Michel Foucault, particularly his studies of how sexuality came to be constituted in Western societies as an experience which was accessible to diverse fields of knowledge and linked to a system of regulation (Foucault 1985; Foucault 1990a; Foucault 1992). As will be discussed in more depth in the next chapter, Foucault's work is not primarily drawn upon in this thesis for theoretical formulations or empirical data;[2] instead, it is used mainly to develop general heuristic principles for studying the relationship between expert knowledges, forms of governance, and subjective experience. These principles are specified most clearly in the second volume of *The History of Sexuality* when Foucault describes the three axes which constitute modern experiences of sexuality as:

> (1) the formation of sciences (*savoirs*) that refer to it,
>
> (2) the systems of power that regulate its practice,
>
> (3) the forms within which individuals are able, are obliged, to recognize themselves as subjects of this sexuality (1990b: 4; Foucault 1992).

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[2] Although there are close, sometimes intimate, connections between notions of addiction and madness (e.g., addiction is most often classified as a mental illness, and is sometimes thought to bringing about forms of insanity), criminality (addiction is linked to crime rates, and most forms of drug use are criminalized or disciplined), illness (addiction requires forms of medical and professional intervention), and sexuality (sex is often considered an addiction; drug addiction has also been linked with 'dangerous' forms of sexuality and the transmission of HIV), the parallels between addiction and Foucault's substantive topics of investigation become quite obscure in a discussion of the neuroscience of addiction and dependence. Cf. **Foucault, M.** 1973 *The birth of the clinic : an archaeology of medical perception*, London: Tavistock Publications, — 1979 *Discipline and punish : the birth of the prison*, Harmondsworth: Penguin, — 1985 *The history of sexuality*, Vol. 1, The will to power, London: Penguin Books, — 1989 *Madness and civilization : a history of insanity in the Age of Reason*, London: Routledge..

These three dimensions correlate to the three major foci that guided the current study of addiction science and the constitution of its neurochemical subjects: first, the emergence of neuroscience knowledge about addiction as a disease of the brain and a problem of desire; second, the development of new principles for and techniques of acting on individuals in light of that knowledge; and third, the ways in which the neurosciences of desire, as they inform understandings of human identity, choice, and conduct, are coming to be deployed in the governance of social and personal problems.

While the material in this thesis is by no means strictly divided into these three investigational domains, the chapters do sequentially shift their focus from one domain to the next. Chapters 3 and 4 emphasize the historical development of bioscientific studies of addiction which made it possible to conclude that addiction was a chronic, relapsing disease of the brain. Chapters 5 and 6 primarily examine how, as the study of the brain's endogenous reward system led to an emphasis on pathological forms of craving, new ways of treating and classifying individuals (as subjects of neurochemical desire) emerged. And Chapters 7 and 8 explore the application of neuroscience understandings of and treatments for pathological desires to different sorts of (legal, medical, and civic) subjects. However, as the following outline of the remaining substantive chapters in this thesis indicate, there is also significant overlap of investigational domains within each chapter.

SUMMARY OF CHAPTERS

Chapter 2 – Towards an historical ontology of desire (and a sociology for the biomolecular era)

The following chapter outlines the theories and methods drawn upon in this thesis, and situates the present study in relation to three main fields of social science inquiry.

First, the ***sociology of desire and subjectivity***. The chapter suggests that much of the social and philosophical theorization of desire (from Freud onwards) has overemphasized the extent to which desire is repressed, and has left aside questions about how we come to know what desire actually is. It suggests that this work has tended to take the essence of desire for granted; that is, it has implicitly assumed that desire exists as something stable and unchanging, and as something that is simply dominated or brought under control in the interests of society, or some powerful element thereof. Drawing upon the work of Jean-Francois Lyotard and Michel Foucault, the chapter develops an alternative to such approaches, which is based on the assumption that the nature of desire changes over time, and thus that an important question is how we come to know about our desire, and about ourselves as desiring subjects. Second: the ***sociology of addiction***. The chapter provides a critical overview of the main issues regarding addiction that sociologists and allied disciplines have engaged with. It suggests that while many sociological accounts of addiction have offered important insights on the ways that addiction discourses and treatments are 'socially constructed' and change over time in relation to broader social norms and values, very few have engaged with neuroscience theories; it argues that such an engagement is of increasing importance, as the presumed objectivity of the new brain sciences are posing challenges to traditional sociological accounts of mental health and illness. Third: the ***social study of neuroscience***. In order to develop a framework for examining how social factors are involved in the production of even the most technical forms of neuroscience knowledge about mental health and illness – and, more importantly, for examining the emergence of neurochemical models of addiction – the chapter draws upon the analytical insights developed by Ludwik Fleck, Ian Hacking, Bruno Latour, and others on the ways in which expert

and scientific discourses represent and intervene upon their subject matter. Finally, the chapter describes the strategies used for selecting and analyzing material for this project.

Chapter 3 – Biomedical problematizations: the construction of a chronic, relapsing disease

This chapter examines how the most fundamental assumptions about addiction that are held by brain scientists today can be traced back to concepts and research strategies that were developed by pioneering physiological researchers in the middle of the twentieth century. It shows how, as new ways of thinking about and studying addiction as a somatic problem emerged, the nature of the addiction itself came to be redefined. The chapter looks at research that was undertaken between the 1940s and the 1960s by physiologists and research scientist who developed standards for studying addiction as an objective condition that was amenable to investigation with the laboratory sciences, focusing on the emergence of two of the most fundamental concepts to contemporary scientific understandings of addiction, namely homeostatic adaptation and behavioural conditioning. It investigates how these concepts came to be adopted by addiction scientists, how they were used to structure early scientific research on addiction as a physiological condition, and how they shaped the directions for future research that would move from observing organic subjects – persons or organisms as indivisible wholes – to penetrating the physiological interiors of subjects in order to implicate precise biological processes and structures in the disease.

The origins of these concepts are examined in relation to the scientists with which they are attributed:

- *Homeostasis.* C.K. Himmelsbach, the physiologist who first theorized that addiction was the result of homeostatic adaptations to drugs. Himmelsbach argued that compulsive drug-taking could be explained as a result of the addict trying to avoid withdrawal symptoms. He thought that drugs were only addictive if they produced withdrawal symptoms, and that once a withdrawal syndrome had subsided, an addict could be considered a cured 'postaddict.'
- *Conditioning.* Abe Wikler, who argued that Himmelsbach had over-emphasized the role of negative reinforcement, and argued that Pavlovian conditioning could explain addiction. Wikler, a pioneering behavioural pharmacologist, used animal experiments to demonstrate that even after withdrawal symptoms had passed, longer-lasting physiological adaptations caused individuals who were exposed to cues associated with drug use to relapse.

In relation to these investigations, the chapter focuses on how the physiological nature of addiction shifted from an acute, easily-treatable condition, to a chronic disease that requires that attention be paid to the physiological risk factors involved in relapse – the return to drug use by an abstinent individual. It examines how the inability to remain abstinent came to be identified as a scientific problem, and how it became possible to define and study relapse in objective, physiological ways, in terms of the 'biomedical problematization of relapse.' Setting the stage for future chapters, it concludes by examining how, as biomedical explanations of relapse became first utterable, then reasonable, then factual, ideas about the nature of addiction as a disease, the addict as a subject, and the physician as a healer began to change.

Chapter 4 – Addiction biopolitics and the neurobiological imagination

This chapter investigates the emergence of molecular neuroscience understandings of addiction by looking at how it became possible to state, as a matter of scientific fact, that addiction *is* indeed a disease that manifests itself at the neurobiological level. It examines the development of research into the molecular, laboratory sciences of addiction, not in terms of the unified theories and explanatory models that emerged in the late 1980s and 1990s, but in terms of a will to know addiction as a neurobiological condition. It asks how it has become possible to think about addiction in terms of the molecular structures of the brain; and how these structures, and the processes in which they are involved, have been brought into existence. The chapter suggests that the discovery of the neuroscientific truths of addiction cannot be understood independent of social rationalities, material considerations and realities, and scientific entrepreneurship. Rather, the interplay between cultural, political, economic, and technological factors often influenced which particular ways of going about studying, thinking about, and researching addiction have been pursued most actively.

To support this claim, the chapter demonstrates that even according to scientists themselves, the facts of addiction neuroscience, and the means for producing those facts, have been made possible only in relation to an alignment of interests between the US government and a community of scientific researchers which involved:

- *The novel biopolitical strategies of the US government.* The US had historically dealt with addiction by simply incarcerating addicts, and prohibiting the use of narcotics within maintenance programs. Beginning in 1965, however, the US government began to fight a War on Drugs that was based upon 'science, rather than ideology or anecdote.' This involved the approval of a federal program of methadone clinics, and the development of

new funding programs for basic and clinical research into the neurobiological basis of addiction.

- *The emergence of a 'neurobiological imagination.'* Amongst researchers, there emerged in the 1960s a project – significantly facilitated by the support of the US government – to position addiction science within the field of legitimate biomedicine. This, of course, required producing evidence that addiction could be explained in terms of neurochemical models, and could be treated as a disease of the brain.

The chapter focuses on how the lines of research undertaken in this period were partially specified by a range of material, technological, political, and epistemic contingencies, and how some key research initiatives – most notably, the demonstration of opiate receptors, the discovery of endorphins, and the development of an opiate antagonist that could block the effects of heroin without producing euphoria – led to the positing of new neurochemical facts about addiction, and also raised new problems for researchers and clinicians. These new facts and problems become the focus of the following chapters.

Chapter 5 – Pathologies of desire and the science of craving

This chapter traces how the brain disease of addiction came to be characterized by bioscientists as a matter of craving. While craving and pleasure-seeking had long been considered important problems by psychologists, it was not until the 1970s, in the wake of discoveries of the opiate receptors and the development of naltrexone, that craving and pleasure began to appear as neurobiological phenomena. This chapter describes how the elucidation of the neurochemical reward systems, and the unexpected finding that naltrexone could reduce cravings, helped make it possible to conceptualize craving – which for so long had been an entirely subjective

phenomenon not considered by physiologists – as objectively related to a neurochemical event, something that could be scientifically defined, observed, and acted upon. It also explores how new representations of addiction that focus on endogenous brain chemistry (rather than on exogenous drugs) have drawn formerly disparate types of drug addiction together with a common underlying explanation (the 'dopamine hypothesis of addiction'), thereby making possible new rationales and objectives of medical treatment.

The chapter examines how, as craving has come to be understood as a result of changes in parts of the brain that are involved in the control of emotions and motivation, a significant proportion of the research and treatments of addiction science have come to focus on managing pathological drug cravings with medications that modulate the neurochemical processes within these areas. It suggests that from the 1970s onwards, as the inability of chronic drug users to remain abstinent came to be understood as resulting from abnormally intense cravings for drugs which are caused by malfunctions of the neurological structures and mechanisms that are involved in the control of emotions and motivation, addiction has come to be represented as what can be referred to as a *pathology of desire*. As the problem space of addiction came to centre around the brain, the fundamental problem of relapse ceased to be relapsing behaviour – that is, the actions involved in the return to drug-use. Instead, it became the mind/brain state of craving, which is understood to lead to relapsing behaviour – or at least to increase the risk of relapse.

Thus, this chapter suggests that neuroscience-based addiction treatment has come to involve a sort of risk-management that can be understood within an anatomo-politics

of the human brain: within contemporary biomedicine, the therapeutic problem has shifted from managing the relapse-related behaviour of the addicted individual, to regulating neurochemical processes. The addicted subject's neurophysiology is essentially, and more or less literally, treated as a *risky matter*. As the activity of the neurochemical reward system came to be identified as the locus of the pathological thoughts and behaviours associated with addiction, and as naltrexone was shown to modulate craving and reduce the risk of relapse, addiction became newly intelligible (drug addicts suffered from chronic brain disorders, and all major types of drugs, from heroin to cannabis, appeared fundamentally comparable). Correspondingly, the goals and logics of addiction treatment came to shift from augmenting will-power to mediating (neurochemical) desire.

Chapter 6 – Problems of conduct as diseases of the brain: behavioural addictions and the neuroscience facts

This chapter investigates how it has become possible to think of behavioural addictions as 'real' addictions, and to treat them with the same medications that are used to treat drug addictions (e.g., heroin and ethanol). More precisely, it investigates how some behavioural problems are coming to be (re)constituted as addictions within neurochemical styles of thought; and how the biological facts and psychopharmacological technologies of contemporary addiction neuroscience are giving rise to new ways of understanding and fashioning the self as a subject of neurochemical desire. It suggests that today, as representations of addiction that focus on the role of dopamine and the endorphins have come to occupy a central position in neurobiological styles of thought, the concept of behavioural or non-substance addictions is becoming much less controversial than it once was. The underlying neurochemistry of addiction makes it difficult, perhaps even impossible, to make a

distinction between drug and non-drug addictions that is based on neuroscientific facts.

The chapter focuses on a period of roughly one decade, from the mid-1990s onward, in which naltrexone was being introduced into the treatment of behavioural compulsions. While neurochemical models of and treatments for craving, which focus on the brain's own rewards, rather than the effects of drugs, may provide an intuitive link between drug addiction and behavioural addictions, the chapter argues that other forms of reasoning, as well as broader political and economic contingencies, were involved in the production of these new neuroscientific 'facts'. The most salient of these are identified as:

- *Psychopharmacological forms of reasoning.* Naltrexone has played a key role in bringing about a shift in thinking about behavioural addictions as neurological conditions: the fact that naltrexone works to reduce desires for gambling, sex, etc. has been used to justify seeing gambling addiction, sex addiction, etc. as – like other addictions – diseases of the brain. Thus, as social scientists have found with other medications, an effective treatment can help legitimize a controversial disease category.
- *Political economy of neuroscientific research.* The very fact that clinical experiments have been done to test the efficacy of treating behavioural addiction with naltrexone appears to be tied to the (indirect) funding of such research by parties that could potentially gain from scientific evidence suggesting that behavioural addictions exist – most notably, the gambling industry. While there is little evidence to suggest that such economically interested parties are involved in academic fraud, their support of research in

certain areas is obviously selective, and this has implications for the truths that are found by scientists.

While suggesting that the re-making of behavioural addictions as matters of neuroscience fact is a social and political process, the chapter also examines how the new, neurochemical truths of compulsive desire are providing the means for re-making aspects of society, politics, and individuality that relate to a range of behavioural problems, from kleptomania to binge eating. It examines how neurobiological theories have begun to change the ways in which such behaviours are represented as problems, especially focusing on how problematic behaviours that have traditionally been understood in relation to social problems are beginning to be reconfigured as problems that can be explained and dealt with as matters of neurobiology.

Chapter 7 – Logics of neurochemical control and the problem of drinking

This chapter looks at how alcoholism has been rendered newly intelligible and newly governable within neurochemical thought styles that explain addiction in terms of pathological desire, by examining how naltrexone has been incorporated into strategies for managing problematic desires and problematic drinkers. After a brief consideration of how it became possible to deploy a pharmacological treatment that was once thought to be a magic bullet for heroin addiction (because of its direct and specific actions on the opiate receptors) in the treatment of alcoholism and the control of problem drinking, the chapter focuses on elucidating some of the social and legal implications of this new means of treatment. It does so by describing the emerging

logics of control and forms of governance within which naltrexone therapies are situated. Two specific strategies are explored as case studies:

- *The ContrAl program.* This alcoholism treatment program situates naltrexone as an internal, pharmacological mechanism of control over drinking that replaces, or at least minimizes, the need for the intense forms of introspection and self-monitoring (associated with disciplinary forms of power) that constitute the basis of other treatment modalities. Thus, the control of desire becomes a routine matter of self-care, based on decisions about taking medication, rather than the confessional and revelational practices.
- *Drug courts.* American drug courts are increasingly offering individuals convicted of drinking-related offences to avoid imprisonment on the condition that they take naltrexone for a specified amount of time. This probationary strategy is based on the understanding that craving for alcohol prevents successful behavioural modification in traditional probation programs, and that naltrexone acts on the brain pathways involved in the pleasure-producing effects of opiates and alcohol.

Based on a discussion of Deleuze's concept of 'societies of control' and Foucault's work on 'disciplinary societies,' the chapter suggests that naltrexone is not deployed according to logics of discipline and normalization, but rather, according to a new logic of intervention and correction that might be referred to as one of 'neurochemical control.' The chapter demonstrates that at the same time that naltrexone has been deployed within the penal-therapeutic formations of the criminal justice system, it has also formed the basis of a range of other strategies for managing behaviour and desire which do not primarily involve punishment or the crushing of subjectivity. This

discussion suggests that naltrexone, as it makes possible new experiences and new means of governing conduct that are engaged in on a profoundly ethical and personal basis, allows certain types of subjectivity (which are perhaps novel) to flourish. Thus, naltrexone as a technology of neurochemical control that is based upon a constant modulation of the brain's activity may be used within a logic of legal discipline, but also within an ethical logic of *self*-control.

Chapter 8 – Knowing and caring for the neurochemical self

This chapter, as the penultimate in the study, moves towards a conclusion by opening out the scope of investigation to a consideration of how contemporary addiction science is being deployed within something like a 'biopolitics of the population' – that is, attempts to foster the health and biological well-being of societies by *preventing* the onset of addiction. Such operations focus not on managing specific problematic individuals, or the treatment of particular cases of pathology; instead they are geared towards targeting 'the public' or some element thereof in order to pre-empt thoughts and behaviours (e.g., opinions that there is nothing wrong with experimenting with drug use) that might lead to drug use or addiction.

The chapter provides evidence that, today, biopolitical strategies of addiction prevention govern, at least in part, through the production of a form of neurochemical subjectivity that is similar to those which provide an essential conduit of power in the regimes of naltrexone control described in the Chapters 6 and 7. However, in addition to the absence of direct pharmaceutical intervention, strategies of preventative governance do not involve the deployment of scientific discourses in unmediated form (i.e., as enunciated by scientists or other biomedical experts themselves). Instead, they rely on translations of technical science into ostensibly value-neutral

forms of advice and information that can be used in training and educational programs for a wide range of non-experts. Thus, as the focus of this study shifts from regulatory strategies involving direct therapeutic intervention into the lives of risky individuals, toward those strategies that place an emphasis on the preventative administrative management of populations 'at risk,' the sources of empirical data change as well. The texts that do the work of preventative governance are not those which are produced by experts for other specialists, but are instead those which are oriented towards the population at large – i.e., popular science.

This chapter develops a case study of popular neuroscience accounts of addiction provided by the American National Institute for Drug Abuse – the world's foremost source of information on drug use and addiction – in order to examine some of the ways that 'at risk' individuals are provided with particular ways of knowing themselves and thinking about their lives that are based on incitements to care for their brains. This material is analyzed in terms of its relation to a cultivation of neurochemical subjectivity among its target audiences. The material, it is suggested, provides a model for living a good and healthy life which is based upon knowledge about one's brain and about how one's actions and choices about substance usage may affect one's neurological functioning. Because the NIDA material represents the brain as an object of individual (and social) care, and offers neuroscience-informed techniques for living one's life on a day-to-day basis as a responsible, autonomous neurochemical subject, the material is considered as a technology of neurochemical selfhood which encourages individuals to effect, by themselves or in alliance with others, programs of self care aimed at bringing about certain neurological states that are associated with happiness, well-being, purity, and functionality.

Chapter 9 – Conclusion

The thesis concludes with a brief reflection on how the new forms of knowing and caring for the self according to neurologic and pharmacologic rationalities described in this study have begun to change how we think about ourselves and our desires as the subjects of regulation. It suggests that due to the emergence of new intellectual models and practical technologies, which allow the body to be represented at the sub-organic level, changes are occurring in the way desire is being imaged, imagined, thought about, and acted upon. It suggests that while it may not be the case that all individuals will be encouraged to undertake pharmacological treatment for problematic desires, it does seem that the widespread, popular discourses on myriad types of addictions indicate that, in contemporary Western societies, individuals are increasingly expected to engage in the monitoring of thoughts and cravings in terms of neurological function – and possible pathology.

CHAPTER 2: TOWARDS AN HISTORICAL ONTOLOGY OF DESIRE (AND A SOCIOLOGY OF THE BIOMOLECULAR ERA)

INTRODUCTION

This study arises in part as a result of a dissatisfaction with much of the existing social science literature on contemporary developments in biological psychiatry, psychopharmacology, and molecular neuroscience. The dissatisfaction with the literature is quite straightforward: there is not very much of it! Given the extent to which we, in the industrialized societies of the North Atlantic, have come to deploy what are understood to be brain-targeting drugs in the management of moods, personalities, affective states, thoughts, and behaviours, it is lamentable how little social science attention has been focused on the sciences and practices of contemporary psychopharmacology. This is especially problematic since, as Joan Busfield notes, the new brain sciences are bringing into question the social explanations of mental health and illness that sociologists have developed since the 1970s (Busfield 2000).

If the politics of the present are, to a significant extent, 'the politics of life itself' (Rose 2001) – a politics in which social, personal, and global questions increasingly come to involve conceptions and explanations of our selves, our societies, and our essences in terms of the biological – they are essentially also a politics of the human brain. For we are told, today, that we are who we are because of what our brains do; that we do what we do because of what our brains do; we feel what we feel, think what we think, like what we like, because of the structures of specific neurons and actions of neurochemicals inside our heads (cf. Churchland 1986). Notions of what it

means to be human, today, are largely bound up with the meanings, explanations, and theories of contemporary molecular sciences of the brain.

Why have social scientists, with only a very limited number of exceptions, remained so apparently unconcerned with these sciences? It would be hard to say without making almost groundless conjecture. But responses of some sociologists to my own work – to this very study – perhaps reveal a bit about the situation. Taken as a group, their collective (insistently sociological) refrain might sound something like this: "This work is interesting, but is it sociology? You've done no fieldwork, no interviews – where are the people? Wouldn't it be better to study addiction in terms of *social* processes (labelling theories offer good examples) and individual experiences, than to study in such detail the *science*?" But what do these very questions, with their implicit accusation of breaching disciplinary boundaries, tell us about the sociologists who ask them, if not that these sociologists accept the assertion that the formation of knowledges about the brain are *not* social processes?

This study is thus in part formulated to provide an empirical rebuttal to assumptions about the non-social nature of neuroscience: to demonstrate, in relation to a case study on pathological desire, that the production of neuroscientific facts is, even at the most basic and technical levels, inherently social. This endeavour goes beyond sociological approaches which restrict themselves to explaining how competing interests, successive ideologies, and the interplay of structural forces determine the nature of science. In this thesis, what is taken to be the 'social' aspects of neuroscience is not limited to its ability to dominate other types of knowledges and to

legitimate projects of social control; the assumption is that *everything* about neuroscience is, to some extent, social.

Approaching every aspect of the neuroscience of addiction – from the communication of ideas and theories, to the invention of laboratory instruments and the development of experimental methods – as at least in part a social event is, of course, not a groundbreaking innovation; for over two decades, a group of scholars associated with 'Science and Technology Studies' have been developing frameworks suited to such an endeavour (cf. Latour and Woolgar 1986 [1979]). Some of this work will be discussed below, toward the end of this chapter. But before this, it should be noted that while this study involves attention to a level of mundane and technical detail that many sociologists would rather avoid, the goal here is not only to make a case for (and present a case study of) the 'social studies of neuroscience.' The particular case study chosen was also selected with the intention of contribution to something like a 'libidinal sociology,' or a sociology of desire.

A SOCIOLOGY OF DESIRE AND SUBJECTIVITY

Although sociologists have, over the last few decades, begun to investigate how some forms of sexual desire (most notably, homosexual desire) have been pathologized by medical and scientific authorities, they have largely avoided taking up, as a focus of serious and sustained inquiry, the problematics of pleasure and desire in their essence. The latter have been, however, central themes for both psychoanalytic and philosophical theorists. From Freud onwards, desire has been a key topic of psychoanalytic concern. Freud's basic thesis – that the survival of human civilization requires the sacrifice of libido and the restriction of libidinal impulses to reproductive sexuality (i.e., primary genitality and relations between human individuals of the

opposite sex) (Freud 1989) – came to inform the theories and therapeutic methods of generations of psychological therapists. It also brought the problem of desire into focus for social and political theorists.

One of the earliest and most ambitious of such problematizations was Herbert Marcuse's (1966 [1955]) Marxist study of Freudian psychoanalysis, *Eros and Civilization.* Here, Marcuse argues that what Freud takes to be an 'inevitable' biological conflict between the gratification of individual desires and the fulfilment of civilizational needs is in fact only an *historically specific* opposition: while the 'basic repression' of instinctual drives may be necessary for the development of pre-industrial societies in which resources are scarce, modern, industrial production technologies are so efficient that sublimation of libidinal drives is no longer *necessary* in order to meet a society's material needs. Thus, Marcuse argues that in the advanced stages of industrial civilization, the possibility of an unrepressed Eros emerges – a return to polymorphous sexuality and the pleasure principle.

However, the liberation of Eros has not been achieved in industrialized capitalist societies, Marcuse suggests, because the *fact* of scarcity (which requires basic repression) has been replaced with an *organization* of scarcity. The uneven distribution of wealth and the hegemony of particular ideals and norms that structure social life ensure that the rising tide of productivity does not overwhelm the barriers that are in place to contain desires. As Marcuse puts it:

> The high standard of living in the domain of the great corporations is *restrictive* in a concrete sociological sense: the goods and services that individuals buy control their needs and petrify their faculties. In exchange for the commodities that enrich their life, the individuals sell not only their labor but also their free time. The

> better living is offset by the all-pervasive control over living (Marcuse 1966 [1955]: 100).

Marcuse suggests that the 'relaxed sexual morality' of the post-war era is possible because the 'surplus repression' of modern societies rule "not so much over the instincts as over consciousness" (Marcuse 1966 [1955]: 94).

Despite his suggestion, above, of a 'concrete' sociological investigation, Marcuse's study is explicitly a work of philosophy (the subtitle of his book is *A philosophical inquiry into Freud*), which focuses on political theorizing rather than empirical investigation. While it offers little in the way of empirical sociological evidence, what is interesting about Marcuse's political philosophy is its attempt to formulate desire (and repression) as the central political and societal issue of the post-war era. Marcuse closes his Preface to the 1966 re-edition of his book by informing his readers: "Today the fight for life, the fight for Eros, is the *political* fight" (Marcuse 1966 [1955]: xxv). Politics, for Marcuse, is a sphere constituted by technological, biological, and material disputes and conflicts over the human body, and what is ultimately at stake is the body's status as "an instrument of pleasure rather than labor" (Marcuse 1966 [1955]: xv).

Marcuse's concerns are echoed by Gilles Deleuze and Felix Guattari, whose work *Anti-Oedipus* (Deleuze and Guattari 1977) focuses on a 'political analysis of desire.' While their conceptualization of desire is in many ways radically different from that of Marcuse, their understandings of the political nature of desire remains close to that of Marcuse. For Deleuze and Guattari, desire represents the will to transform oneself, or the impulse to alter affective states by changing one's relation to social and material environments. This desire is kept in check – identities are kept stable – by

the territorializing influences of a variety of forms of social organization: individuals are tied down to roles, jobs, responsibilities, and other relations which restrict the potential of what they may experience and become. Although capitalism has a liberating potential, insofar as it subverts older forms of territorialization (such as religion and the family), it in fact creates in their place new forms of social organization which are equally, if not more, repressive. As with Marcuse, for Deleuze and Guattari contemporary politics are a politics of desire, in which individuals must fight the repression of desire in capitalist societies – the increasing control and manipulation of bodies and pleasures – in the hope of ultimately liberating desires from repressive territorializations.

For some contemporaries of Deleuze and Guattari who took an interest in investigating desire, however, the problems and politics of desire were not so clear-cut. In *Libidinal Economy*, Jean-Francois Lyotard (1993) was among the first to question the assumptions of prevailing radical accounts of desire, repression, and experiences of pleasure in capitalist societies. Lyotard suggests that for these (and other) theorists, the study of capitalism has, to a large extent, focused on the study of repression and pain – of discomfort and hardship, of emasculation and impotence – and has ignored, or failed to recognize, complementary aspects of pleasure and gratification. Caught up in their zeal for criticism and their sympathy for the most oppressed segments of society, the work of critical theorists who sought to document the suffering and agony produced by modern industrial capitalism obfuscates what Lyotard takes to be self-evident: that pleasures, passions, and desires are as prevalent in contemporary industrial societies as they were in pre-industrial ones.

For Lyotard, then, the political economy of capitalism is also a libidinal one; just as new forms of wealth are produced and distributed among individuals in society, so are new experiences of pleasure and desire created. However, these new libidinal forms are generally described by critical theorists as pathological – as when the pleasures of mass-produced goods and popular entertainment are understood to be symptomatic of a sort of false consciousness, or when desires for inanimate objects are described as a sort of malignant commodity fetishism. For Lyotard, a distinction between natural and pathological desires can only be made by claiming an illegitimate moral authority over the experiences of pleasures. Thus Lyotard asks his contemporaries:

> Why, political intellectuals, do you *incline towards* the proletariat? In commiseration for what? I realize that a proletarian would hate you, you have no hatred because you are bourgeois, privileged smooth-skinned types, but also because you dare not say the only important thing there is to say, that one can enjoy swallowing the shit of capital, its materials, [...] and because instead of saying this [...] you lean forward and divulge: ah, but that's alienation, it isn't pretty, hang on, we'll save you from it, we will work to liberate you from this wicked affection for servitude, we will give you dignity. And in this way you situate yourselves on the most despicable side, the moralistic side where you desire that our capitalized's desire be totally ignored, forbidden, brought to a standstill, you are like priests with sinners, our servile intensities frighten you, you have to tell yourselves: how they must suffer to endure that! (Lyotard 1993: 115-6).

Lyotard's accusation is quite simply that theories about the repressive nature of capitalism depend upon the establishment of limits to what pleasure can be taken to be, and upon the suggestion that pleasure, so defined, is suppressed in capitalist society. If it is difficult to recognize pleasure and desire in capitalism – in generic consumer goods, in the dulling pain of machine operation, in a sea of uncaring faces, or in workers' struggles – this is because critical theorists of desire have on the one hand emphasized the desirability of certain types of pleasures and fulfilments, and on the other hand have excluded other delights and impulses from consideration.

But since there is no objective position from which to determine which experiences and affects are intrinsically more pleasurable – or more valuable – than others, these discourses at best are blind to, and at worst consciously devalue, the pleasures and experiences of the non-bourgeois others they seek to liberate. Not only do they define and limit the realm of desire, but they also impose these definitions upon their subjects. Thus, Lyotard suggests that engaging in simplistic critiques of repression or in theorizations about the creation of 'false' desires puts one at risk of becoming, just like critics of homosexual or masochistic desires, a "crude bastard of the moral order" (Lyotard 1993: 110).[3]

Lyotard's sweeping, philosophical conjecture about the illegitimacy of theories of repression was followed, two years later, with Foucault's now famous rejection of the 'repressive hypothesis' (Foucault 1985 [1976]). In contrast to Lyotard, however, Foucault offers, in his three-volume *The History of Sexuality*, a grounded, empirical study suggesting that there are a wide range of possibilities for the experience of desire and the management of pleasure between the opposing (and indeed, he suggests, fictional) poles of complete domination and absolute liberation of the desiring subject. Foucault's insight is that although pleasure and desire are experienced as subjective and autonomous domains, they can only exist in relation to political, historical, and discursive factors. The experience of the desiring self, the formation of standards for the voluntary management of desires, the personal projects of self-discipline and

[3] All of this does not imply the redemption of capitalism and its exploitation and alienation of individuals and groups – Lyotard does not attempt to privilege the capitalist mode of production with the status of a unique means to libidinal fulfilment, but merely exhorts us to escape from an orthodoxy of needs and values that seeks to impose on its subjects a regime of libidinal truth. What Lyotard demands is that a theorization of desire does not begin its analysis with pre-formulated conceptions of what desire is – its meanings, its values, its forms.

government – none of these develop outside of relations of power, accounts of truth, perceptions of obligation, and the sociocultural generation of expectations of proper thought and behaviour.

Foucault's work on sexuality is not drawn upon in the present study for its substantive material – for example, his elucidation of the particular regimes of self care practised by the ancient Greeks – but rather for its insights on how the subject of desire is constituted by a range of expert understandings and techniques that are imposed upon and adopted by individuals. During the latter part of his career, Foucault came to make an analytic distinction between two recurring themes in his work on subjectivity. On the one hand, much of his early work focused on investigating disciplinary techniques – those procedures through which bodies are studied, mapped, and regulated by experts, and through which deviance is corrected and brought in line with normalcy (Foucault 1979; 1989). On the other hand, his later work focused on the ways in which individuals come to know themselves and to devise their own practices and standards of being, or 'techniques of the self' (Foucault 1990a).

Perhaps more than anywhere else in his writings or interviews, Foucault most explicitly discusses his understandings of how personal ethics relate to historical, cultural, and political issues in a chapter on "Morality and practices of the self" included in *The Use of Pleasure* (Foucault 1992). Here, Foucault teases out some of the ambiguities and multiplicities of the meaning of the term 'morality,' and relates them to the methodological orientations of his study. He begins by noting what are for us the two most obvious meanings of morality:

> By "morality," one means a set of values and rules of action that are recommended to individuals through the intermediary of various

> prescriptive agencies such as the family (in one of its roles), educational institutions, churches, and so forth. [...] "morality" also refers to the real behaviour of individuals in relation to the rules and values that are recommended to them: the word thus designates the manner in which they comply more or less fully with a standard of conduct, the manner in which they respect or disregard a set of values (Foucault 1992: 25).

In the first instance, morality can be understood as socially and institutionally sanctioned standards of and prescriptions for conduct. Morality, in this sense, posits a pattern of behaviour that is accepted in or expected of individuals, and provides norms of conduct against which individuals can be measured. But the term can also describe the judgment of an individual's behaviour in relation to such standards, so that individuals may be thought to possess their own, varying levels of morality.

Morality, in these senses, may at first seem somewhat irrelevant to an investigation of the contemporary neuroscience and pharmacology of addiction; but this is only because the notion of morality has today become so closely associated with *Christian* morality. We do not associate morality with biology and molecular science because we tend not to associate the latter with Christianity. But if we accept a more general definition of morality, such as that proposed by Foucault in the above passage, then it is not difficult to understand how neurological and psychopharmacological knowledges might be said to provide the basis for biological moralities – sets of values and rules of conduct that a variety of intermediary agencies might prescribe to individuals on the basis of understandings about the human body and brain. What physicians, biomedical researchers, and health education officials tell us about the facts of normal brain states and functioning comes to define what individuals ought to do. As these facts become embedded in the conventions and attitudes of societies, and also in the strategies and rationalities of social and political projects, they provide

a basis for evaluating and regulating the behaviours and dispositions of individuals (or groups of individuals). Individuals may thus be said to be characterizable as biologically moral to the extent that they measure up to expectations of how responsible persons who look after themselves (and their brains) would act.

In relation to standards and evaluations of conduct Foucault identifies a third aspect of morality:

> another thing still is the manner in which one ought to form oneself as an ethical subject acting in reference to the prescriptive elements that make up the code [i.e., social standards of conduct]. Given a code of actions, and with regards to a specific type of actions (which can be defined by their degree of conformity with or divergence from the code), there are different ways to "conduct oneself" morally, different ways for the acting individual to operate, not just as an agent, but as an ethical subject of this action (Foucault 1992: 26).

Foucault uses the term 'ethics' with a meaning that is similar to the ancient Greek usage, to specify the ways in which individuals come to involve themselves in the setting of standards and evaluations of their own thoughts and conduct. Ethics interest Foucault because no matter how pervasive and forceful external moral regimes may be, there is always a certain space of freedom for the individual to act according to his or her own prerogative. Within this space, the individual may very well still think and behave according to social standards; but this is not a matter of imposed discipline, because it involves *self*-regulation, rather than external control.

It should be clear, then, that what is ethical does not correspond to conformity with a law, a rule, or an obligation to others. It involves the identification of parts of the self that need to be taken into account in order to meet one's own aspirations, and the establishment of means of governing one's self in such a way that brings one's

thought and conduct in line with these aspirations. But it should be equally clear that the ethical cannot arise independently of the moral, and that the ethical subject, the governing self, always exists in relation to external standards.

The important question about (various forms of) ethics is not whether they allow individuals to escape the relations of power and knowledge that constitute society; it is, rather, how, and with what freedoms and responsibilities, individuals constitute their own relationships with themselves. Foucault makes an analytical differentiation between 'code-oriented' and 'ethics-oriented' moralities in order to distinguish those which emphasize the compliance with and enforcement of social norms of conduct, and those which depend principally upon formations of self-rule within individuals. This distinction has methodological implications for Foucault: with moralities of the former type, "the important thing is to focus on the instances of authority that enforce the code, that require it to be learned and observed, that penalize infractions; in these conditions, the subjectivation occurs basically in a quasi-juridical form" (Foucault 1992: 30). In contrast, a study of moralities of the latter type, where the observance of systematic codes and rules may be less important than the individuals' experience of him- or herself, "would be concerned with models proposed for setting up and developing relationships with the self, for self-reflection, self-knowledge, self-examination, for the decipherment of the self by oneself, for the transformations that one seeks to accomplish with oneself as object" (Foucault 1992: 29). We should note that Foucault does not recommend, nor does he dedicate much effort to, an analysis of the precise content of individual relationships with themselves; he encourages and pursues studies of the *models* for forming such relationships that are provided within authoritative discourses.

Towards the end of his life, Foucault came to believe that his early studies of the human sciences of the seventeenth and eighteenth centuries had overemphasized the extent to which discipline and domination constituted the experience of the self by simple imposition; but even a consideration of ethical domains, he thought, would relate to knowledge and power. The important ethical investigations which Foucault regrets not having pursued in his earlier work were, for example, "What are the games of truth by which man proposes to think his own nature when he perceives himself to be mad; when he considers himself to be ill; when he conceives of himself as a living, speaking, laboring being; when he judges and punishes himself as a criminal?" (Foucault 1992: 7). His conclusion is not that ethics can or should be a realm of mere aesthetics – of unimpeded self-fashioning and free choice – but that, in order to understand how certain experiences of the self are made possible, we need to relate aesthetics, ethics, and self-care to political, discursive, and technological contexts of broader societies.

Unfortunately, the analytical distinction Foucault formulated is often misconstrued as the positing of a simplistic dichotomy between discipline / domination on the one hand, and ethics / freedom on the other. As a result, it is sometimes claimed that Foucault's substantive shift in focus to the techniques of self of ancient Greek and Roman societies marks a "radical break with his previous conceptions of subjectivization", and that after this break Foucault was almost exclusively concerned with "technologies that allow individuals to create new modes of being, distinct from those imposed by the workings of power regimes" (Starkey and Hatchuel 2002: 647, 642). In the most extreme cases, Foucault is described as "a philosopher who had

always been concerned with the care of the self and whose project, despite its general application, was essentially individual" (Nehamas 1998: 169). The present study avoids using Foucault's *analytical* distinction as a theoretical imperative or an empirical claim about separate domains; instead, it seeks to understand how forms of discipline and techniques of the self are each implicated in the other, and how individuals are positioned in such a way that they simultaneously exercise freedom and are subjected to power over their desires. Indeed, this study suggests that, in important ways, the personal ethics relating to desire *depend* upon the presence of certain games of truth and regimes of discipline.

This study examines the forms of ethics – and the types of desiring subjects – that are made possible by contemporary neurosciences of desire. It examines how models of and techniques for care of the self increasingly involve the truths and treatments of the brain sciences. Expert knowledges about addiction are especially important here, because they have provided some of the most important and influential models that we have in the modern era for developing relationships with ourselves as healthy, desiring subjects. Sociological studies of addiction thus relate to this project in important ways.

ADDICTION: A CASE STUDY OF PATHOLOGICAL DESIRE

The vast majority of sociological work on addiction has focused on contributing to expert knowledge on how to understand and / or control compulsive thoughts and behaviours. Sociological theories about what addiction is, for example, attempt to define addiction in social terms. These accounts frequently incorporate medical or scientific understandings into their ontological accounts, but generally maintain that addiction arises out of social relationships and processes to such an extent that these

social elements constitute the essential 'nature' of addiction. A number of authors have argued that medical approaches to addiction often fail to grasp the fact that addiction is a 'biopsychosocial' condition (Orford 1985) that at least in part arises or manifests itself as a result of social and environmental factors (e.g., Larkin and Griffiths 2002; Miller 1996; Peele 1985). Others pay less attention to physiology, and explain addiction in exclusively sociological terms. Giddens (1992), for example, suggests that addiction results from the anxieties and uncertainties individuals face in detraditionalized societies; Truan (1993) posits that addiction exists simply because of the functionality of the concept – addiction is a social construction that, by establishing and maintaining a character structure of a certain type of (addicted) individual, makes it easier to think about and act upon behaviour. Since such formulations offer little insight into the development of scientific and molecular models of addiction, these are of only limited relevance to the present study.

Perhaps the most common form of sociological studies of addiction are those which fit within psych- or social-work approaches and which focus on what are understood to be the pragmatic issues of controlling addictions and addiction-related problems. These approaches do not engage with the issue of what 'addiction' might or might not be, but instead focus on such issues as estimating the various (social, economic, personal) costs of addiction, determining the effectiveness of different modalities of treatment, or elucidating the precise ways in which different modalities appear to bring about desired treatment outcomes (e.g., Lemmens 1996; McLellan, et al. 1998; Single, et al. 1998; SSRC 1983). These approaches, too, are only of tangential interest in relation to the focus of this thesis.

More closely related to this study are the various 'critical' or 'constructionist' investigations which have been developed over the past few decades. A number of these studies have investigated specific forms of addiction such as alcoholism (Alasuutari 1992; Denzin 1987; Gusfield 1996; O'Reilly 1997; Wilcox 1998), co-dependence (Rice 1996), internet addiction (Surratt 1999), or sex-addiction (Irvine 1995); in doing so, they have highlighted the contingency of notions about addiction, and have illuminated how addiction theories and treatments can be related to social and political issues. However, because of their focus on particular *forms* of addiction or behaviour, these tend not to engage with the conceptions of addiction *itself.*

There have been a small number of inquiries into general medical theories of addiction. Included among these are extreme examples of social constructionist arguments which are dismissive of the possibility that addiction is a biological issue at all. For example, Thomas Szasz, one of the first anti-psychiatric critics of the addiction concept, produced a series of almost polemic publications which assert that addiction is, like other forms of mental illness, a 'myth' (1961) which helps make possible the 'ritual persecution' of drug users by state and institutional authorities (1975; 1979). The tradition of explaining addiction medicine and treatment in terms of the functions they serve (most notably, social control) has continued with other reductionist accounts of addiction (Davies 1992; Hammersley and Reid 2002; Schaler 2000). While such accounts of addiction medicine have been of political value in struggles against psychiatric authority, they offer little insight into the concerns of the current project.

There has been a series of more nuanced accounts of the difficulties of distinguishing 'normal' drug and alcohol use from addiction (Derrida 1993; Keane 2002), and of the ways in which cultural factors play a role in determining how addictions are represented (Alexander and Roberts 2003; Friedling 2000; Gabe, et al. 1991; Quintero and Nichter 1996) and how addiction theories relate to understandings of the self (Alasuutari 1992; Denzin 1987). These astute analyses examine addiction as a condition that relates (but is not produced by) a series of political, legal, moral, and biomedical methods of regulation. For example, in an insightful but brief analysis, Eve Sedgwick (1992) examines the ways in which tropes of addiction have become a conduit for self-governance in contemporary Western societies. Reflecting on new perceptions of the risks of 'becoming' an addict and fears of 'losing control' over oneself, Sedgwick suggests that contemporary 'epidemics of the will' seem to necessitate pre-emptive regimes of monitoring and control at an individual level, as those who consume and behave in non-compulsive, unproblematic ways increasingly feel compelled to govern their own thoughts and conduct in attempts to prevent the loss of self-control that may develop into addiction or dependency.

Sedgwick offers an interesting and provoking cultural analysis of addiction, making it clear that the problematics of addiction require more serious, critical attention than they have been given by social scientists. However, Sedgwick does not consider in any serious manner how these 'cultural' forms of governing desires and the will – such as those engendered by a booming industry of self-help publication and the proliferation of 12-step and other recovery groups – have arisen. She suggests that the 'slippage,' or expansion, of the addiction concept may be related to the anomic conditions of 'the consumer phase of international capitalism:' commodity fetishism

and the unlimited trajectory of demand seem to be kept in check as self-helping individuals learn to shape their consumption (of alcohol and other drugs, but of all types of commodities and behaviours as well) within the realm of autonomous individuality. But this sketch is not an adequate explanation of the proliferation of addiction / recovery discourses in present day understandings of conduct and desire; especially since it fails to consider the rationalist and scientific approaches to addiction alongside cultural, non-specialist ones.

In *Diseases of the Will*, Mariana Valverde (1998) offers a much more concretely grounded analysis of alcoholism and addiction. In a detailed historical study, she documents how problems of desire and compulsion have been approached from the late eighteenth century onwards by medical and legal authorities. She especially focuses on the various attempts that have been made to medicalize or rationalize problematic alcohol consumption – and describes how each one of these attempts has successively failed. Valverde shows how time and time again the most respected physicians and scientists have proven incapable of creating a fully medical or biological explanation of addiction that is able to free itself from the moral and ethical stumbling blocks that impede a 'scientific' approach to alcohol (and other forms of) addiction.

However, we might question Valverde's presumption that failure among those who search for a science of addiction is inevitable. If her study limited itself to a discrete historical period rather than attempt to continue its analysis up to the present day, these presumptions would not be as problematic as they end up. An assertion of the failure of biomedical experts to have arrived at an adequate understanding of

alcoholism between the 1880s and the 1930s, for example, would be quite easy to defend. Unfortunately, Valverde seems to conclude that the past failures of scientific models of addiction will be repeated indefinitely, forever assuring the impotence of addiction experts. Addiction, she argues, has always been, and always will be, a 'hybrid' condition which is partly biological, but also partly moral. Given the contemporary developments in molecular addiction research, such a suggestion needs to be concretely examined, rather than taken for granted.

Although the critical and constructionist sociologies of addiction referred to above have produced many valuable insights, especially to the extent that they have implicated addiction treatment within regimes of political, social, and moral regulation, one of their main weaknesses has been the failure to consider, in earnest, the contemporary physiological sciences of addiction (Weinberg 2002). In the most extreme cases, researchers have tended to suggest that there is no real biophysical basis to addiction – that addiction is 'merely cultural,' a social invention that enables and justifies a variety of forms of social control. As Michael Bury notes, the idea that medical disciplines are simply discursive or 'ideological edifices' seriously underestimates the broad range of social, personal, and political issues at stake in contemporary understandings and practices of health and illness (Bury 1997).

In less extreme (and less problematic) studies, the biological tends to be brushed aside without comment: social, cultural, and political dimensions of addiction medicine and treatment are focused on to the virtual exclusion of scientific and physiological considerations. This is problematic, too, as scientists are increasingly asserting that the current shift from social and psychological theories about addiction to

neurobiological models constitutes a shift to a truly scientific approach, with a new objectivity that is based on the most fundamental, molecular facts of the human body. Due to advances that have taken place in a range of disciplines since the 1960s, from organic chemistry and pharmacology to behavioural psychology and biopsychiatry, a new way of studying, measuring, and formulating addiction has arisen. And this new style of thinking about addiction, precipitated by the emergence of neuromolecular medicine, may significantly alter the field of addiction studies. The present project builds upon a small number of recent sociological analyses (e.g., Bourgois 2000; Keane 2002; Rose 2003a; Valverde 2003; Weinberg 2002) which consider the possibility that the representation of addiction in terms of neurological dysfunctioning, which has significant implications for the field of addiction studies, also challenges conventional social science analyses of addiction and thus requires further examination. Because of their high relevance to the current investigation, these studies are discussed within the context of the findings reported in the substantive chapters that follow.

A SOCIAL STUDY OF NEUROSCIENCE

This thesis studies the emergence of neuroscientific models of and psychopharmacological treatments for pathological desires in order to contribute to the sociological understandings of addiction, desire, and neuroscience in a complex, rather than reductive, way. This is not to suggest that desire is repressed or identified as a problem to be eliminated, but to understand how the neurochemistry of desire actually came to be thought of as an integral element of human life that demands attention and study, and came to form the basis for a wide range of projects of investigation, regulation, and care. In between the positions of extreme constructionism (studying addiction as a myth) and biological essentialism (studying

addiction as a fact), it investigates addiction and desire as existing within a social field of 'multidimensionality' and 'indeterminacy' (cf. Laclau and Mouffe 1985); as things that exists on a molecular level, certainly – but only because they have been brought into being within a particular 'style of thought' that itself is related to a complex assemblage of personal, social, material, and scientific factors. In doing so, it significantly draws from the pioneering work of Ludwik Fleck (1977), as well as more contemporary scholarship on scientific styles of thought and, more generally, social studies of science and medicine.

For Fleck, scientific facts cannot be investigated as if they arise from the observations of an isolated individual; to do so would presume that the objects under observation can present themselves independently of human perception, and also that human perception consists of an innate capacity to process external stimuli. Fleck argues that facts can only emerge within the contingent structure of a 'thought style,' which itself is continually reproduced within epistemic networks which Fleck refers to as 'thought collectives.' Facts themselves must be considered social events, existing only as observations produced with particular fields of perception. Since 'facts' do not arise from the direct observation of nature any more than they do from the conscious construction of social actors, the opposition between 'nature' and 'culture,' so prominent in many sociological approaches to science, becomes less necessary.

Concepts such as 'reality' and 'fact,' for Fleck, do not describe any essential nature, but are in fact expressions of a sort of alienation from human thought itself. All objects have to be thought into existence through human thought, and this is achieved only within a particular, shared thought style, which determines how objects are

perceived. It is only within a thought style, as human perception is coordinated and directed, that an object can be uniformly observed as such. Once this coordination and direction cease to be consciously mobilized – when individuals are no longer conscious of their participation in perception – things come to be thought of as having a natural existence as one or another type of object. Thus, what we consider to be reality is largely that which we take for granted. When we become so used to 'seeing' objects or facts within a prevailing thought style that we lose awareness of our own historically contingent perspective, Fleck argues, these things assume an independent power over us that we passively accept.

By emphasizing the epistemological structuring of cognition rather than the ontological content of truth, Fleck's approach makes possible the investigation of scientific facts as *events* rather than as *things*. Since "*truth is the up-to-date stage of changes of thought-style*" (Fleck 1986: 111), truth is an event in the history of thought. Any fact can only survive as long as it continues to invoke the stylized perception of a member of a specific thought collective. Since the ability to directly perceive form [Gestaltsehen] can only be acquired after the induction (whether formal or informal) into a thought collective, all perception is 'stylized,' that is, limited to observations within the perceptual fields of a particular style of thought. Thus, induction into a thought collective in some ways limits the field of perception of the individual: incongruences with a prevailing thought style come to be formulated as impossibilities. All this suggests, of course, that thought styles and the truths they engender – such as the scientific thought style and scientific facts – act as a constraint on thought: observation is limited, perceptual awareness is reduced, technologies

lessen active participation. Thus, the more entrenched an individual is within a particular thought community, the less ability s/he has for disagreement or dispute.

However, Fleck considers this necessary, rather than lamentable: it is only by establishing limits on the possibilities of thought, by requiring the epistemological conformity of utterances that are allowed to 'count' within a field, that facts can develop at all. Fleck's overarching concern is not to emphasize the ways in which thought styles restrict human thought. On the contrary, he demonstrates that thought styles are necessary for certain objects to be brought into being. If they were not kept within a single epistemological framework, not only would solutions to or explanations of a problem be difficult to arrive at, but problems themselves could not be formulated. A style of thought provides a common set of intellectual principles and practical conventions that specify not only what counts as an explanation, but also what needs to be explained. Fleck maintains that if social scientists are to properly theorize scientific knowledge, "[t]hinking must be accorded a certain power to *create* objects [...] but, of course, *only if it is the style-permeated thinking of a collective*" (Fleck 1977: 181, emphasis added). Only 'style-permeated' thinking – that is, thought that adheres to the thought style of a collective – has creative potential, and this is precisely because it is constrained.

Fleck's book was originally published in 1935 with the subtitle *Introduction to the Theory of the Thought Style and Thought Collective* (*Einführung in die Lehre vom Denkstil und Denkkollectiv*). Fleck was not presuming to offer a sophisticated, clearly formulated theory, but was rather introducing, or moving towards establishing, the investigation of scientific knowledge in a novel way. Fleck is in fact explicit on this

issue: "[I]t is not the aim of this book to construct a complete theory of thought styles. All I want to do is point out a few distinctive properties of the communication of thoughts between collectives" (Fleck 1977: 108-9). Thus, some of the recent work on scientific 'styles' might not be as clearly aligned with or differentiated from Fleck – or one another – as they consider themselves to be.

Ian Hacking, for example, claims that Fleck's thought styles are restricted to a particular discipline or substantive field of knowledge that produced facts about a specific issue or set of related issues, and uses the phrase 'styles of reasoning' to describe his own approach. Hacking suggests that his approach is distinguishable from Fleck's work on the basis of its focus on general methods of inquiry which cross disciplinary boundaries and guide all branches of contemporary science (Hacking 1982; Hacking 1992; Hacking 2002b). However, Fleck decidedly does not restrict the conceptual or analytical elements of thought styles to disciplinary boundaries. When Fleck writes of a 'stylistic bond' that exists between 'many, if not all, concepts of a period' (1977: 9), he is writing generally, about the thought style that determines "*the given stock of knowledge and level of culture*" (1977: 39). Hacking's 'styles of reasoning' could quite easily be considered in terms of the 'stylistic bonds' of the "specialized thought style of science" (1977: 145) which Fleck's book is ultimately about.

Thus, both Fleck and Hacking are predominantly concerned with general social or philosophical theories of scientific objectivity. Fleck would agree with Hacking that social studies of science need to investigate scientific styles "not because styles are objective (i.e. we have found the best impartial ways to get at the truth), but because

they have settled what it is to be objective (truths of certain sorts are just what we obtain by conducting certain sorts of investigations, answering to certain standards)" (Hacking 1992: 4). Similarly, Hacking's work supports Fleck's claim that "nobody has either a feeling for, or knowledge of, what physically is possible or impossible" and that "[w]hat we feel to be an impossibility is actually mere incongruence with our habitual thought style" (Fleck 1977: 48). The sorts of investigations, standards of evaluation and so on that are particular to specific thought styles change an individual's readiness for directed perception which in turn offer new possibilities for discovery and the creation of new facts. Just as these standards of objectivity are the central concern of Hacking, so too does Fleck state that they are "the most important epistemological significance of the intercollective communication of thoughts" (1977: 110).

But while Fleck shares Hacking's concerns about broad, cultural styles of thought that *do*, in a sense, 'stand above' specific disciplines and locales (cf. Hacking 1992b), he is not satisfied with the production of a philosophical theory about scientific knowledge that is divorced from empirical investigation. Fleck believes that "[c]orresponding to any thought style is its practical application" (1977: 104), and that it is largely through the study of these applications that sociological understandings of science will advance. Through a concrete investigation of ways in which thought is organized, engaged in, and mobilized in relation to the social and historical circumstances of the development of the Wasserman reaction to syphilis, Fleck provides an indication of how sociological researchers might go about investigating the genesis and development of scientific truths within definite, substantive enterprises.

Fleck is not fundamentally concerned with the specific implications of the case of syphilis and the thought community which developed the Wasserman reaction, except to demonstrate the effectiveness of his analytical framework. However, as Rose notes, analyzing a particular problem or discipline in terms of styles of thought can provide a rich source of insights about the ways in which different forms of argument, observation, prediction, and demonstration intersect within an assemblage of intellectual and practical problems. Rose's work on understandings of depression within the style of thought of biological psychiatry (2000; 2003a) not only contributes to an understanding of the ways in which criteria of objectivity are subject to historical and cultural change, but also how classificatory regimes, experimental procedures, and other truth-making technologies reshape how mental illness is imagined today.

Like the substantively-focused studies of Fleck and Rose, the present investigation of how neurochemical conceptions of addiction have been brought into objective being through the specific formulations of molecular biomedicine empirically investigates the practices and problems of a specific thought community. This study identifies and analyzes the ways in which expert discourses and practices produce the truths of dependence and addiction that effect the constitution and regulation of individuals on a local, contingent basis. It does so by examining how the specific style of thought of neurobiological addiction science, through the establishment of new criteria of proof and demonstration, translates, territorializes an addiction which is in some form 'already there,' into a more particular (i.e., both distinct and molecular), fixed reality (Rose 2003; Hacking 2002), and which lends itself to certain rationalities of governance.

METHODS AND MATERIALS

My principal interest is *contemporary* representations of and interventions for pathological desires as matters of the brain. However, it would be difficult – or rather impossible – to demarcate what exactly constitutes 'present' or 'current' debates in academic and professional discourse, since contemporary issues about addiction have come into being only as a result of interactions between pre-existing cultural, medical, and scientific notions about disease, desire, health, individuality, and so forth. Thus, my work might be described as an investigation of present conceptions of addiction; but one with a necessarily historical component, namely, an examination of the linkages between present conceptions of addiction and their conceptual precursors. This is fundamentally a genealogical approach (cf. Foucault 1994, 1984): the endeavour is not to show how all past thought on addiction has progressed chronologically to give rise to present theories, but is rather the more modest endeavour of linking today's understandings with specific elements of the past. Thus, I do not attempt an account of how past ideas have led up to the current issues I am concerned with, but instead investigate how current ideas can be traced back to past ones. This difference is significant, insofar as the latter approach, in contrast to the former, does not demand an explanation of why it is that current conceptions have arisen as they have, but only seeks an elucidation of how current conceptions have become possible.

In taking a genealogical approach and looking back at addiction from the perspective of current neuroscience, this study focuses attention on the particular issues and developments that pertain to questions about the implications of new, molecular discourses on pathological desire. It should be noted that an emphasis on genealogical considerations in preference of traditional approaches does not impute a

lack of significance to actual events, happenings, and activities that occur within a specific historical context. This study largely consists of an account of the practical, temporal, and material events that relate to these conceptual developments in addiction science: inventions of technologies, changes in treatment and regulation, and mobilizations of resources, for example. What I do avoid, however, is the temptation of suggesting that these events contain, in themselves and in relation to one another, an adequate basis for a causal explanation of contemporary styles of thinking about addiction.

The strategy taken here resembles what Bruno Latour (1987) refers to as 'following scientists through society;' but it would perhaps be more accurate to describe my methodological program as following *concepts* – and, more specifically, the concepts of a particular thought community – through society. The difference is slight, but significant: the former suggests something of an ethnographic approach, with scientists taking center stage as objects of analysis. And yet one of Latour's most insistent claims is that "fact construction is such a collective process that an isolated person" – even a scientist – "builds only dreams, claims and feelings, not facts" (Latour 1987: 41). Statements cannot be established as either fact or fiction by the person uttering them or by their intrinsic qualities; their fate is determined only as they enter into wider networks of discourse and practice that may not always include scientists.

Of course, 'following concepts through society' is not a simple – or perhaps even possible – task, since 'society,' whatever we take it to consist of, will be composed of an almost unlimited number of chains of association. My investigation has therefore

been limited to following concepts through a specific thought community: through the networks of scientific, neurological addiction research; although this does include a consideration of attempts made by these experts to expand their style of thinking so that it includes larger and larger segments of society. I have further limited my undertaking by focusing more on the concepts, or elements of concepts, that are particular to my specific interests – molecular addiction science – and are traceable through official and technical texts. Thus, my project is primarily based on archival research, that is, on the analysis of a set of texts which describes a set of rules that define what, in molecular addiction discourse, can be counted as a fact or object; and which correspond to a set of practices that operate in accordance with these rules to produce facts and objects that count.

These slight modifications to Latour's method suggest a concept-driven investigation of molecular styles of thought on addiction: a method of documenting how the representations, inventions, and classifications of a particular thought community gradually produce scientific facts that are not entirely dependent on scientists. This method assumes that there are no unmediated, intrinsic perceptions, interpretations, or experiences of desire, compulsion, and drug use; and that these are influenced by how we imagine them in relation to our bodies and ourselves. It asks what molecular biosciences of compulsive desire 'make of' the human body: what they define the desiring body as, how they configure it, including some elements, excluding others, and how they go about acting on it. Such an approach does not attempt to describe what the body, or what addiction is, but what it is thought to be – even more precisely, how it is thought into being within a particular style of thought, and how this style of thought extends beyond a small collective of research scientists.

Because my concerns are with concepts that originate in expert and scientific discourses on addiction, my primary sources are principally obtained from archives that are collected by addiction experts themselves. Specialty journals of addiction studies, of which there are over seventy-five (Babor 2000), themselves constitute a significant resource – one so significant, in fact, that it is potentially unwieldy, given the breadth of its content, not to mention its sheer size. Rather than attempt any sort of randomly-controlled sampling of specialty addiction journals, my method of selecting materials resembles the 'interdefinitional' method described by Bruno Latour in his account of the revolutionary impact of microbe sciences. Latour notes that "[i]f we open up the scientific literature of the time, we find stories that define for us who are the main actors, what happens to them, what trials they undergo." We do not have to attempt to 'independently' ascertain what is of importance and what is not because "studies of the texts of the time will do the job of *interdefinition* for us" (1988: 9).

This thesis' account of the development of neuromolecular styles of thought on addiction explores a wide range of contemporary texts on addiction and its nature, to examine the degree to which contemporary molecular accounts of addiction establish their scientific imprimatur on the basis of the suggestion, establishment, and maintenance of a singular conception of addiction, i.e., addiction as a (relatively) sutured totality that can be located within the biochemistry of the body. These texts reckon with themselves and one another in an attempt to achieve the coherent and consistent accounts of the neuroscientific facts of addiction.

The process of interdefinition for this project began with a close analysis of the pre-eminent serial publication in addiction studies, the journal *Addiction*, which was established in 1884 (with the original name *The British Journal of Addiction*) by the (formerly "British") Society for the Study of Addiction, and is the longest established journal in its field. The journal's editorial board prides itself on the journal's reputation for scientific quality, for the diversity of material it publishes, and for its pioneering role in stimulating and leading debate. For these reasons, *Addiction* is especially useful as an expert archive per se: a collection, founded well over a century ago, of records which have been selected by a respected professional organization on the basis that they possess permanent value because they provide important information about addiction.

Addiction contains editorials that provide overviews of and opinions about the most current issues in their fields, and publishes contributions not only from academic researchers, but also from therapeutic professionals, and even policy-makers; thus it offers a means of considering the practical, therapeutic, and social implications of the expansion of molecular thought styles. Moreover, it provides, interdefinitionally, links to other publications and materials that have been integral to the contemporary addiction science: reports on conference proceedings, citations of articles from general scientific journals, such as *Science* and *Nature* which report on 'breakthroughs' in research on the brain, and professional reference works and textbooks which contain indications of the impacts of academic and scientific thought on how addiction is conceptualized and intervened upon at a molecular level. While it is certainly the case that only a small percentage of articles, mostly of relatively recent origin, directly pertain to molecular models of addiction, the contents of

Addiction provide an overview of how, when, and to what extent such discourses have appeared and how they have been received by the general field of addiction studies. They also shed light on the adoption of emergent concepts and explanations within fields of other life sciences (e.g., neuroscience, endocrinology, pharmacology, molecular biology, genetics, etc.) by addiction experts, and of the ways in which these discipline-specific utterances have been translated into enunciations of addiction discourse. Thus, by beginning from an analysis of *Addiction*, this study interdefinitionally gains access to a wide variety of sources that are used to explore the implications of recent developments in the life sciences for the ways in which forms of desire are thought of and acted upon.

CHAPTER 3: BIOMEDICAL PROBLEMATIZATIONS: THE CONSTRUCTION OF A CHRONIC, RELAPSING DISEASE

INTRODUCTION

> Addiction used to be defined as tolerance to and physical dependence on a drug of abuse. [...] We now know that detoxification is, at best, a first step in beginning treatment and that achieving the drug-free state is not a particularly significant accomplishment. The more difficult aspect is *prevention* of relapse.
>
> Charles O'Brien, *Science* (1997: 66, emphasis added)

> Drug dependence generally has been treated as if it were an acute illness. Review results suggest that long-term care strategies of medication management and continued monitoring produce lasting benefits. Drug dependence should be insured, treated, and evaluated like other chronic illnesses.
>
> McLellan et al., *Journal of the American Medical Association (2000: 1689)*

Today, addiction is described by biomedical scientists as a chronic illness because even after extended periods of abstinence, addicts are prone to return to compulsive drug use. As the American National Institute on Drug Abuse tells us, "Once a patient goes through withdrawal, there is still considerable risk of relapse. Patients may return to taking drugs even though they no longer have physical withdrawal symptoms" (NIDA). The understanding is that it is a relatively easy thing to get addicted individuals off drugs (all one needs to do is minimize the discomfort and anxiety caused by short-lived withdrawal symptoms); but it is quite another matter to *keep* individuals drug-free. The most problematic aspect of addiction is thus thought to be "the high risk of relapse to drug use that persists even in abstinent addicts long after any withdrawal symptoms have abated and perhaps for a lifetime" (Hyman and Malenka 2001: 695).

As the quotations above indicate, however, the conceptualization of addiction as a chronic, possibly lifelong medical condition is a relatively novel development. Such

understandings contrast dramatically from the opinions held by biomedical experts sixty years ago. In the 1940s, there appeared very little doubt that addiction was an acute condition that was relatively easy to cure. Addiction was considered to be a state in which, over extended periods of drug use, the body gradually adapted to the presence of drugs and could no longer function properly without them. If an individual stopped drug-taking, the mechanisms that the body had developed to maintain its equilibrium against the effects of drugs would have nothing to counteract, and would themselves produce a range of pathological symptoms. Consciously or unconsciously, the individual whose body had become dependent on drugs would attempt to prevent such symptoms from arising by continuing drug use indefinitely. So the problem for physicians, when they dealt with addiction at all, was to help the addicted subject achieve abstinence by reducing the pain and distress associated with withdrawal. Once the withdrawal symptoms subsided, the individual was no longer considered an 'addict' – he or she was in fact referred to as a 'postaddict.' And the postaddict was cured: he was not thought to differ in any physiological way from 'never-addicts' (a term we might use to describe individuals who have never taken drugs and never been addicted). While there was an anecdotal awareness that postaddicts seemed to have a greater chance than never-addicts of becoming addicted, relapse was not understood as a medical problem, and was not treated by physicians.

This chapter investigates how, between the 1940s and the 1970s, addiction came to be conceptualized and studied by biomedical experts as a chronic disease. It focuses in particular on how relapse – the return to drug use by an abstinent individual – came to be thought of as resulting from physiological pathologies. The implicit or explicit assumption made by most bioscientists of addiction today is that these changes reflect

advances in knowledge about the nature of addiction that were made possible as the field of addiction studies began to mature into a more objective science. The suggestion from bioscientists is that while the addicted subject had, in the 1940s, been *thought* to return to a normal state of functioning shortly after the withdrawal of drugs, this was never actually the case. It was only as scientific investigations became more precise that it became possible to correct such misperceptions, and to reveal the truth of addiction and relapse: that bodily changes brought about by drug use lasted far beyond detoxification, and that it was these persistent, physiological pathologies that caused a long-term threat to abstinence – and increased the risks of relapse.

According to such explanations, what has changed over the course of history is not addiction, but only our ability to know it. In the past there were imperfect means of studying addiction, and as a result, there were a lot of confused ideas about the condition (indeed, prior to the 1940s, there was a lot of doubt about the very biological *existence* of addiction). Relapse had been thought to be due to psychological or moral problems; or at most, a sort of 'disease of the will.' But the experimental and objective life sciences have, over the last half-century, verified that addiction and relapse are bodily matters. We now know that relapse is not a matter of the will; instead, it is explicable in terms of the matter of the body.

This chapter suggests that while addiction can be known in terms of objectively knowable, physiological facts, these facts, and what the biology of addiction is taken to be, are not immutable. It suggests that the interplay of cultural, political, economic, and technological factors influence what comes to be identified as legitimate means of arriving at truth, and thus also what comes to appear as truth. Since criteria for

objectivity determine what counts as evidence about addiction and what facts about addiction are found in the laboratory, in the clinic, and in the human body, our facts change in relation to the formation of new methods and rules for knowledge-production.

As part of a genealogical study which endeavours to shed light on how we have come to arrive at our contemporary neuroscientific understandings of addiction, this chapter focuses on providing a genealogy of relapse. It asks how current knowledge about the physiology of relapse has been informed and shaped by previous scientific work on addiction. The goal here is not simply to document the historical and social contingency of the scientific truths of addiction at a particular moment in time; it is also to investigate how the emergence of specific truths have helped make possible new ways of doing science which have a bearing on our own present.

Foucault's concept of problematization (Foucault 1991; 1992) can help us in this two-fold endeavour. For Foucault, problematization is a concept which highlights how an ensemble of theoretical discourses and applied investigations "make something enter into the play of true and false and constitute it as an object of thought" (Foucault, quoted in Rabinow and Rose 2003: xviii). An analytic perspective based upon forms of problematizations does not seek to define an object, but rather to understand how objects come to be knowable and potentially brought under control. This chapter, which examines how the inability to remain abstinent came to be identified as a scientific problem, and how it became possible to define and study relapse in objective, physiological ways, might thus be thought of as an investigation of the biomedical problematization of relapse.

The chapter particularly focuses on the emergence of two of the most fundamental concepts to contemporary scientific understandings of addiction, namely homeostatic adaptation and behavioural conditioning. It investigates how these concepts came to be adopted by addiction scientists, how they were used to structure early scientific research on addiction as a physiological condition, and how they shaped the directions for future research that would move from observing organic subjects – persons or organisms as indivisible wholes – to penetrating the physiological interiors of subjects in order to implicate precise biological processes and structures in the disease.

In order to understand how relapse – the inability to remain abstinent – became a scientific problem, the chapter begins (following this introduction) by describing the development of a scientific understanding of the abstinence in relation to the work of the first physiologist who pioneered methods for measuring and observing the 'abstinence syndrome' experienced by individuals withdrawing from a dependence on opiate drugs. The first half of the main body of this chapter, then, looks at how abstinence came to be studied and explained within bioscience discourses, and provided a basis for conducting quasi-experimental studies about drug and withdrawal effects which enabled addiction to be known as a matter of physiological fact. This section focuses principally on the influential work of Clifton K. Himmelsbach, whose work in the 1930s and 1940s helped constitute addiction as a physiological condition which could be studied according to the rules of experimental science. It describes how Himmelsbach's introduction of the concept of homeostasis into addiction studies helped change what researchers counted as relevant information about the physiology of addiction, and led to particular prescriptions for the medical treatment of addicts.

The second half of the chapter examines how the introduction of conditioning theories (and experiments on laboratory animals) by Abraham Wikler allowed researchers to bring into question the acute nature of the abstinent syndrome. By developing ways of studying withdrawal symptoms in animals, and demonstrating that such symptoms could be produced long after detoxification, Wikler's work suggested a 'protracted' abstinence syndrome which appeared only under particular environmental conditions. This evidence led to the biology of addiction being expanded to include mechanisms involved in relapsing behaviour in abstinent individuals. And as biomedical explanations of relapse became first utterable, then reasonable, then factual, once again ideas about the nature of addiction as a disease, the addict as a subject, and the physician as a healer changed.

DISCOVERING THE 'BODY'S WISDOM': HOMEOSTATIC APPROACHES TO ADDICTION

Although there is much within today's medical knowledge of addiction that is contested, one of the most basic and widely acknowledged facts of addiction science is that addiction involves changes in the body which result from extended periods of drug use. The understanding that the body is not merely acted upon by drugs, but itself anticipates, reacts and adapts to the presence (and signs) of substances, is one of the foundational assumptions that has guided the field of addiction science for the last half-century. And yet, at the moment of its inception this idea was extraordinarily novel, and its acceptance had long-lasting impacts on what addiction was thought to be, and how addiction was scientifically studied. Indeed, John Littleton, one of the world's most prominent addiction pharmacologists, recently suggested that the hypothesis that addiction could be related to the body's adaptation to the presence of

drugs was the 'single best idea' in drug-dependence research, and implied that the man who came up with this idea, Clifton Himmelsbach, might be as significant to addiction science as Charles Darwin has been to biology (Littleton 1998).

It was in the early 1940s that Himmelsbach proposed that drug addiction arose as a result of homeostatic mechanisms (1941; 1943). He reasoned that when certain types of drugs are introduced into the body they had disruptive effects on physiological functions. In order to cope with these disruptions, the body initiates its own counterbalancing actions which attempted to restore normal systemic balance. With prolonged or chronic drug use, the actions of the drug and the homeostatic adaptations of the body cancelled each other out. And when such a state of equilibrium was attained by the body, users became *tolerant* to the actions of drugs, and had to overwhelm homeostatic adaptations by increasing their dosages in order to obtain achieve the same drug effects.

The onset of tolerance marked the development of a new equilibrium in the body, as homeostatic mechanisms more or less neutralized the effects of drugs. This new balance was not disrupted by the presence of drugs, but was instead dependent upon it. If the body's supply of drugs was suddenly reduced or cut off, the homeostatic processes would have no external action to counteract; they would become redundant. And their effects, now superfluous, would produce their own disruptions to the body's balance. Himmelsbach theorized that it was redundant homeostatic mechanisms that produced the range of distressing withdrawal symptoms that addicts experienced when coming off drugs, and suggested that it was avoidance of this 'abstinence syndrome' that motivated the addict to continue taking drugs.

Since its initial formulation, Himmelsbach's original conceptualizations of addiction have been significantly modified, but his idea about the relationship between the compulsion to use drugs and the body's adaptations to drug effects has been extraordinarily influential. Himmelsbach remains identified as one of the key founders of current thought on addiction and dependence among bioscientists (Martin 1980; Paton 1969; Sharpless and Jaffe 1969). And although direct reference to his work has become increasingly rare, this is to be expected, as his idea has come to stand on its own, in abstraction from the conditions of its construction. Homeostatic drug adaptation has become a physiological fact of addiction that is so evident that one does not need to be persuaded to accept it through the appeal to authority that a citation provides. Indeed, to provide a reference to Himmelsbach may be seen as irrelevant – or even subversive, since an allusion to the historical conditions of the construction of the idea may appear to suggest that homeostatic mechanisms did not always constitute the physiological basis of addiction (cf. Latour and Woolgar 1986).

But since an objective of this thesis is precisely to demonstrate the social conditions and historical contingency of the neurobiological facts of addiction, it will not do to simply marvel at how Himmelsbach could have gotten such a good idea, and how it could have been so in keeping with the facts of addiction. Instead, the goal is to understand how Himmelsbach's work developed in the way that it did, how it influenced the field of addiction science, and how it helped shape the objective truths of the addicted body. We might begin by thinking about how even the very 'idea,' admiringly attributed to Himmelsbach by Littleton and others, can be understood in social terms. As Latour and Woolgar note, having an idea "represents a summary of a

complicated material situation" which includes institutional settings, interpersonal relations and group traditions, seminar meetings, and other elements (Latour and Woolgar 1986: 170). Thus, explaining the origins of contemporary theories of addiction in terms of Himmelsbach simply having an idea tends to obfuscate the localised, heterogeneous, and material set of circumstances in which the idea emerged, and in which social factors are clearly evident.

One might ask whether the 'single best idea' in addiction science would have been possible without a number of social factors. Would Himmelsbach have explained addiction as the result of homeostatic adaptations if the term 'homeostasis' had not been coined a few years earlier, and not by an extremely renowned Harvard physiologist? Walter B. Cannon invented the concept of homeostasis in the early 1930s to explain the biological form of 'organized self-government' that he presumed had naturally evolved within the biological makeup of humankind, and which he attributed with the power to explain how the seemingly fragile 'organic fabric' of the body could endure not only sudden, acute environmental changes, but also more long-term ones.[4] Would Himmelsbach have seized on Cannon's ideas if Cannon had not invited researchers in a wide range of fields to investigate homeostatic processes (which he believed demonstrated the almost perfect 'wisdom of the body'), and had not assured his readers that searches for the "governing agencies" of the human body "will be rewarded by their discovery" (Cannon 1932: 282)? And what if the link

[4] We might also note that while homeostasis has come to be understood as a natural feature of the human body within the biological sciences, of course, Cannon's idea were also historically contingent. This is made most clear through a consideration of the reception of Cannon's ideas about social and political homeostasis within the social sciences, as Cannon's work was taken up by a number of social scientists, most notably his Harvard colleague, Talcott Parsons. After a brief period of enthusiasm for structural functionalism, analyzing responses to social problems and political disturbances in order to reveal patterns of cultural stabilization was widely discarded because of its circular logic and evolutionary underpinnings. **Cannon, W. B.** 1932 *The wisdom of the body*, New York: Norton.

between Himmelsbach and Cannon had been strengthened by a personal encounter between the two men? In 1934, Himmelsbach visited Cannon at Harvard, and it was at this point that he became convinced of the potential of homeostasis to explain addiction (Acker 2002).

While it might be possible to trace the contingency of Himmelsbach's ideas indefinitely backwards through social and material networks, the main goal of this section is to examine how Himmelsbach's 'idea' that habitual drug use could be explained in terms of homeostatic mechanisms actually became a *fact*. Since the forging of an association between homeostasis and addiction was not made possible simply on the basis of ideas, we need to turn to a consideration of how the requirements of the physiological sciences for objective evidence of this relationship was met.

Inventing objectivity in addiction research

As a physiologist, Himmelsbach felt that valid evidence about addiction could only be obtained by observing his subjects's bodies – not by listening to their subjective reports. Thus, for Himmelsbach the only reliable indicators of addiction were the physiological symptoms which appeared when an individual stopped taking certain kinds of drugs. Shortly after his visit with Cannon at Harvard, Himmelsbach set out upon a program of research to provide evidence that the severity of such symptoms reflected, and could be explained in terms of "the degrees to which mechanisms for the maintenance of homeostasis have been affected by the drug" (1941: 829). This program, which was developed with the goal of making the study of addiction as objective and scientific as possible, nevertheless involved a wide range of social, political, material, and institutional factors.

Even at the level of 'methods and materials,' which might be assumed to involve mundane technical considerations, Himmelsbach's work can be understood in social terms. It is clear from Himmelsbach's own accounts[5] that he did not consider addiction to be something that was always obvious or readily apparent to the casual observer. He argued that scientific studies of addiction required that subjects be observed with methodological rigor, and observations had to be carefully recorded and analyzed. Himmelsbach reasoned that the withdrawal signs of addiction manifested themselves in different degrees of intensity, and that if a researcher's perceptual skills were not sufficiently developed she would fail to recognize them. Furthermore, if symptoms were not methodically recorded, researchers relying on general clinical impressions might overlook less pronounced cases of addiction that an analysis of recorded data would reveal.

Himmelsbach thus set out to invent standards and procedures for producing observations of the abstinence syndrome that could be counted on objectively. Drawing upon the incremental system used to measure the Wasserman reaction for syphilis testing, he created a classification system for categorizing withdrawal symptoms within four classes of severity, so that even marginal signs of dependence – such as watering eyes – could form part of the clinical picture of addiction. In addition, he devised a means of methodically recording these observations with a series of plus signs, from mild (+) to severe (++++). The operationalization of addiction symptoms not only allowed Himmelsbach's withdrawal syndrome to be

[5] Details about the refinements of Himmelbach's methods have largely been drawn from interviews reported by Acker (1995).

represented graphically, but also, by determining what could quite literally 'count' as a symptom, led researchers to record new, more subtle cases of addiction which previously could not have been recognized.

But the refined clinical gaze and specification of standards for the production of truths were not all that was necessarily for Himmelsbach's findings to have been made possible. Historical, legal, and institutional factors provided a context in which Himmelsbach's clinical research could attain degrees of experimental control that approached those of laboratory studies. In the 1930s and 1940s, individuals arrested because of drug-related crimes, and prisoners who were suspected of using drugs were not uncommonly placed into the Narcotics Hospitals of the US Public Health Services – so-called 'narcotic farms' commissioned by the government to house and supervise incarcerated addicts. And much of Himmelsbach's work was carried out after he was appointed to the newly established Narcotic Hospital in Lexington, Kentucky, from which the vast majority of subjects for his research was drawn (on a reportedly voluntary basis). Unlike other researchers who had to rely only on episodic clinical interactions with patients and the reports addicts themselves made about their condition, Himmelsbach had access to a captive population of research subjects who could be observed and experimented upon within the controlled environment of a total institution. Within the medico-correctional research facilities of the state sponsored narcotic 'hospital,' the addict came under almost complete experimental control.

Himmelsbach's research also benefited from his institutional and professional mandate to not only treat addicted prisoners (which would limit the extent to which he

could use his patients in laboratory-like investigations), but to also use prisoners as experimental subjects in order to produce findings that would help government agencies deal with the social and legal problems associated with drug use. For example, state regulators wanted information about the addiction potential of various drugs, so that they could classify or prohibit those likely to be abused. To assist regulators obtain this information, part of Himmelsbach's research involved testing the addictiveness of new medical drugs by administering them to subjects for a period of time, then cutting off the drug supply and observing whether withdrawal symptoms followed.

The power of Himmelsbach's procedures and standards to determine the facts of addiction was demonstrated in early disputes about contradictions between his own findings and those of other researchers. By meticulously reviewing the work of other studies, Himmelsbach was usually able to attribute contradictory findings to inadequate experimental conditions and observation and data-recording techniques. The explanation for conclusions that differed from those of Himmelsbach, then, was usually that other researchers had not used adequately rigorous experimental methods.[6] In most cases, when other researchers adopted Himmelsbach's methods and techniques, disputes were resolved in Himmelsbach's favour. Gradually, as

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[6] For example, a number of researchers argued that Himmelsbach overestimated the addictiveness of some of the newly synthesized derivatives of morphine, finding an addiction potential that others were unable to replicate. Why did Himmelsbach's subjects become addicted when those of others did not, they asked. Their answer was that (among other things), their drug-administration schedules were flawed. Different drugs within the morphine family were broken down by the body at different rates, and quickly-metabolized drugs would not produce addiction unless they were administered more frequently than longer-lasting drugs. With captive subjects, it had been a relatively easy thing for Himmelsbach to devise administration schedules so that the drugs were administered several times a day, and thus that drug effects were sustained more or less continuously. **Eddy, N. B. and Himmelsbach, C. K.** 1936 'Experiments on the tolerance and addiction potentialities of Dihydrodesoxymorphine-D', *Public Health Reports* 118.

Himmelsbach and his colleagues established methodological techniques and principles which became the model for clinical and experimental studies of addiction, evidence and reports began to fall into line with one another, eliminating most objections to Himmelsbach's basic approach to addiction research.

As they placed physiology and biology at the centre of clinical truth-making activities, these new standards of objectivity also made it easier to discount the validity of knowledge of researchers in other fields of addiction studies, and to describe the assumptions of competing disciplines as mere belief or opinion. Recalling the divisions between psychologists and physiologists in the 1930s and 1940s, Himmelsbach noted, in a 1972 interview, that:

> A lot of folks thought that morphine abstinence syndrome was purely psychological, that there wasn't anything basic or organic about it, and if you would snow a fellow with something that would make him feel comfortable and take his mind off it, there wouldn't be any abstinence syndrome at all. Well, this wasn't true (Himmelsbach 1972: 12).

After devising a method for quantifying withdrawal symptoms – a scheme for truth-production in the drug-research laboratory – Himmelsbach had been able to back these claims up with facts. His work made it possible to argue that the truths of addiction were physiological, rather than psychological, and helped strengthen the case for increased research into biophysical aspects of addiction.

As the abstinence syndrome came to be known as a physical reality rather than a psychological experience, it also came to be perceived as providing a causal explanation for the compulsive drug-taking behaviour of addicts. With regular use, homeostatic adaptations developed to counteract the effects of drugs; if an individual stopped taking drugs, these mechanisms would have nothing to counteract, and would

themselves produce unpleasant symptoms; and it was the attempt to avoid such withdrawal symptoms that explained why individuals were not willing to abstain from drug use. Addicted individuals came to be known, to a significant degree, in terms of their physiology.

Knowledge of the physiological subject also had implications for notions of appropriate medical care. Since the abstinence syndrome constituted the condition of addiction, treatment was directed at helping the body rid itself of drugs and making the detoxification process as comfortable and painless as possible while the body readapted to the absence of the drug. "The purpose of withdrawal treatment", Himmelsbach writes in 1941, "is to relieve the patient of physical dependence safely, yet without undue suffering or prolongation of treatment" (1941: 835). Drug addictions were treated as temporally discrete, entirely curable medical problems. This short-term, acute care, was achieved by administering sedatives or hypnotics to an addict while tapering off her access to drugs over a period of three or four days.

Because addiction was curable, individuals could move between states of addiction and non-addiction with relative ease. Addiction, in a significant sense, only lasted as long as withdrawal symptoms could be observed and recorded by a researcher, and so individuals ceased being addicts once observable withdrawal symptoms had been eliminated. The importance of this point should not be underestimated: it offered a basis for distinguishing 'addicts' from 'non-addicts' which, unlike most categorizations deployed today, did not require a permanent assignment to a pathological class of subjects. Complete recovery from addiction was possible.

Himmelsbach reflected on this matter in an interview after his retirement (close to forty years after he was assigned to the Lexington hospital): "A post-addict by definition, I think, was an individual who had been off drugs for at least six months". Those individuals who had gone through drug withdrawal and were positively free of symptoms of the abstinence syndrome were to be defined as non-addicts. Indeed, since an addict could completely recover from addiction within a matter of months, researchers concluded that "there'd be no harm in giving him [sic] a new addiction" (1972: 13) for experimental purposes. Provided that research subjects had six or more months left to their sentence, and could thus recover under the control of the hospital authorities, inmates were 'given' addictions – and had those addictions removed – as a regular part of scientific studies.

By the late 1940s, Himmelsbach had become one of the leading authorities on addiction, and was appointed as the first director the US Addiction Research Center (a forerunner of the National Institute for Drug Abuse). The Center, which was established at Himmelsbach's Lexington facilities and which became the most important research centre in the US, produced many of the pioneering researchers who led the development of addiction science over the next two decades (Kornetsky 2003; Musto 1996). Although Himmelsbach did not theorize addiction on a neurobiological level, the basic physiological principles he set out established some of the most fundamental principles of biomedical styles of thinking about addiction, and focused the attention of a generation of medical researchers to investigate addiction in terms of the body's own processes of adaptation and self-regulation. But conceptualizations of addiction by no means remained static – even in the period that preceded the advent of molecular neuroscience.

HOW RELAPSE CAME TO MATTER

As indicated at the outset of this chapter, addiction is today understood to be a chronic, relapsing medical condition. And yet for Himmelsbach and many other researchers of the 1940s and 1950s, relapse was *not* a medical problem: individuals who had recovered from the abstinence syndrome rejoined the category of normal individuals and were no longer considered addicts. So how did the notion of a "postaddict" disappear from the developing bioscience of addiction? How did the community of addiction research scientists, of which Himmelsbach was one of the most eminent pioneers, broaden its domains of objective inquiry and professional authority to encompass new aspects of its subjects' thoughts, behaviours, and experiences? More to the point, how did relapse become (part of) a medical condition?

The key researcher in the early biomedical problematization of relapse was Abraham Wikler, a junior colleague of Himmelsbach's at the Lexington Narcotic Hospital. It was Wikler who, in the late 1940s, began to suggest that the newly developing physiological investigations of tolerance and dependence needed to be broadened to include aspects of addiction that seemed obvious in other spheres – namely, the tendency of 'postaddicts' to relapse to drug use and become addicts once again (Wikler 1948). Although there had for many years been an awareness among clinicians and researchers of high rates of relapse following acute treatment for addiction, this had been seen as a psychological or moral, rather than medical, issue. Just as Himmelsbach had developed scientific means of explaining the apparently psychological phenomenon of compulsive drug use in physiological terms of tolerance and dependence, Wikler suggested that the problem of relapse – the return to drug use by abstinent individuals – also needed to be investigated as a physiological matter within the domain of legitimate scientific inquiry.

Although Wikler was originally trained as a psychologist, he strove to develop means of investigating problems of the 'mind' objectively, particularly by developing research that drew upon behavioural psychology. Wikler rejected psychoanalytic explanations of relapse on the grounds that they lacked scientific rigour, and were based, by necessity, upon information offered by patients themselves. He asserted that the psychological clinician "has tended to accept his patient's mentalistic interpretation of his addiction and his frequent relapses to drugs as a valid one" simply because psychologists have no source of information other than the subjective reports their patients give them. While this seems acceptable to psychologists who ask "who else would know better what his feelings are than the patient himself? And does not one behave in accordance with one's feelings?", Wikler argued that the data gathered from personal accounts is plagued by imprecision, incoherency, and that the psychoanalyst's explanations of human behavior, including drug addiction and relapse, could never "be tested for validity in the accepted manner of the natural sciences" (Wikler 1984 [1965]: 279). The reliance on their patients' self-reports could possibly explain, Wikler suggested, the fact that psychologists simplistically described relapse as a result of under-developed or maladjusted individuals who could not control their impulses for pleasures and gratification. Wikler believed that "few have ventured to question the decisive importance of euphoria as the main determinant of the addicts behavior, both in his initial addiction and in his subsequent relapses – for this is also the addict's explanation" (Wikler 1984 [1965]: 279).

From the 1950s onwards, Wikler helped provide evidence of a biological basis for relapse by developing experimental strategies for studying drug-taking, addiction, and

abstinence in laboratory animals. Noting that 'postaddicts' sometimes experienced symptoms resembling acute abstinence from morphine long after withdrawal, he hypothesized that:

> physical dependence may become conditioned to environmental situations specifically associated with availability of morphine and hence "abstinence distress," or something very much like it, may be reactivated after "cure" when the postaddict finds himself in a similar situation, thus providing an unconscious motivation to relapse and renewed self-maintenance of addiction (Wikler 1984 [1965]).

Wikler's suggestion was that the bodily changes brought about by drug use were more enduring than had previously been assumed. Even after dependent subjects had undergone detoxification, the homeostatic drug-opponent processes described by Himmelsbach could be activated by encountering certain objects, people, places, or situations. While the body had developed these response mechanisms to prepare for the destabilizing effects of anticipated narcotics, such conditioned homeostatic responses would in fact be perceived in individuals as a 'need' for drugs.

To investigate this hypothetical model, Wikler and others developed experimental strategies for producing addiction, withdrawal, and relapse conditions in laboratory animals. In experiments spanning over a decade (e.g., Siegel 1975; 1976; Wikler 1984 [1965]; Wikler, et al. 1962), the physiological responses of rats were observed under a variety of environmental conditions as they were 'addicted' (Wikler did apply this term to his non-human subjects) and withdrawn from dependence-producing drugs in a variety of ways. One of the earliest experiments was devised to investigate the urges to relapse that were frequently reported by abstinent addicts when they returned to environments associated with drug-taking. Wikler moved his rodent subjects from their 'home cages' to 'training cages,' in which rats were introduced to

drugs, and experienced, over several weeks, carefully manipulated stages of addiction and withdrawal. He found that even after rats had been returned to their home cages and had apparently recovered from addiction, signs of the abstinence syndrome would reappear when the rats were placed back in their training cages (signs of the abstinence syndrome in rats, for Wikler, consisted of an increase in 'wet dog shakes' – "so called because of their resemblance to those of a dog shaking water off its back") (1984 [1965]: 281). Moreover, the rats, if given a choice between unadulterated water and water containing narcotics, were more likely to ingest the drug-laced water in their training cages than they were in their home cages.

As Wikler and others noted, such laboratory conditioning experiments, which suggested that addiction was not quite as an acute physiological condition as had been previously thought, had far-reaching implications for the understanding of addiction if generalized to humans. The finding that drug-related environmental cues could significantly affect the body's autonomic system, and thereby re-initiate drug-seeking behaviour in what were thought to be 'post' addicts brought into question the most commonly held assumptions about the amount of time required for physiological recuperation from addiction – if not, at this point, the assumption that individuals could completely recover. It also raised the question of whether helping a dependent individual through drug withdrawal could be considered adequate therapy, since even after the abstinence syndrome had passed the addict suffered from pathological biological adaptations to drug-related cues. For Wikler, it became clear that effective addiction treatment would by necessity involve the prevention of relapse: if subjects were detoxified, but still sought narcotics due to conditioned responses, they would end up returning to drug use before the physiological addiction had been truly cured.

Bodies at risk

From Wikler's pioneering work onwards, increasing attention was paid by researchers to the study and management of the long-lasting physiological changes that result from the body's adaptation to extended drug use. In an obituary written for Wikler, Jerome Jaffe – one of the most prominent authorities in the field of addiction science today – noted that the conceptualization of addiction as involving the processes of learning and conditioning, "which Wikler was one of the first to champion, has now been accepted as a fundamental aspect of our understanding of the disorder" (1981: 431). Although Wikler and his contemporaries presumed that behavioural changes were the result of a 'pharmacological need' that involved biological mechanisms rather than psychological ones, they could not specifically identify any anatomical changes that occurred in the bodies of addicts (cf. Wikler 1948). The specification of physiological processes of addiction was in fact not essential to the model (or at least not necessary for the model to gain initial currency), since the use of animal subjects in early conditioning analyses of addiction was understood to circumvent the dualism between body and mind (Siegel 1999). However, this work had a profound influence on the direction of experimental addiction science by indicating the plausibility of finding biological mechanisms of relapse.

Wikler's work also influenced clinicians' perceptions of the problem of relapse. It was in the 1970s that addiction experts – and particularly those dealing with alcoholism – began for the first time to study systematically the extent and causes of relapse in human subjects (Connors, et al. 1996). At the beginning of the decade, one researcher noted that there appeared to be "no systematic study of the relationship between events in the lives of alcoholics and episodes of relapse into drinking although relapse is very frequent and at least in some alcoholics a relationship would

appear possible" (Hore 1971: 83). In the next few years, however, studies were undertaken that suggested that relapse was a serious issue: an early relapse study found, for example, that a majority of subjects had resumed addictive behaviours such as drinking, smoking, and the taking of narcotics within six months following the end of treatment (Hunt, et al. 1971); by the time researchers increased the longitudinal scope of relapse research to twelve months following treatment, relapse rates as high as 90 percent were reported (Orford and Edwards 1977).

As theoretical and empirical evidence indicated that the danger of relapse could be related to environmental stimuli and was not an issue for only a few exceptionally problematic addicts, clinicians began to investigate the 'external precipitants' of relapse – those factors that increased the chances of an individual's return to drug use. The gaze of experts began to follow the clinical subject as she left withdrawal treatment, to examine what happened to individuals who had presumably recovered. This avenue of inquiry, virtually non-existent before the rise of conditioning theories, was taken up with considerable enthusiasm by a number of researchers, the most influential of whom was G. Alan Marlatt. By administering questionnaires to alcoholics who had relapsed after treatment (e.g., Marlatt 1976) and observing associations between alcoholic drinking behaviour and various external conditions, Marlatt began to devise a scheme for systematically identifying and classifying 'high-risk' situations that significantly increase the likeliness of relapse – what are now colloquially referred to as external relapse 'triggers' (Marlatt 1996: s40).

Through the 1970s, Marlatt and his associates expanded their alcoholism 'relapse taxonomy' situations to include an increasing number of high-risk triggers, and

created a general taxonomy that could be applied to addictions other than alcoholism (Marlatt and Gordon 1980; 1985). While the substantial growth of identifiable risk-factors may have suggested that the dangers of relapse were ubiquitous, addicts were not to be considered lost causes; indeed, the purpose of identifying relapse triggers was to provide a basis for therapeutic intervention (Marlatt 1978). "The 'good news' about research findings on relapse precipitants", Marlatt notes, "is that the assessment of a patient's high-risk situations for relapse gives both the patient and the therapist a 'handle' on how to cope with relapse risks or actual lapses" (Marlatt 1996: 47).

In clinical settings, the early 1970s studies of Marlatt and others on relapse precipitants "shifted the focus from endogenous biological determinants [...] to exogenous factors such as environmental setting and stressful life events" (Marlatt 1996: s41). But while many clinicians focused on such cognitive and behavioural therapies which sought to teach addicts strategies for minimizing risk-factors associated with relapse, the question of how, exactly, the body and the brain were involved in relapse was not abandoned. There was an implicit understanding among many researchers that what clinicians and therapists had come to know about their individual subjects must relate, in their essence, to biological processes; and that the true risk factors of relapse were not to be found in environments and situations, but in the body and the brain.

Thus, what researchers had, in the late 1960s and 1970s, was a sort of black-boxed body-at-risk. This was not the 'Pandorian' black-box of the sort described by Bruno Latour (1987): the brain had not been actively put together by scientists and then sealed shut. Nor was it a black box that is analogous to the sort of "empty organism"

(Ellinson 1959) of behavioural psychologists; the internal operations of this subject were not inconsequential, and the brain could not be ignored simply because the summarizing input-output functions had been observed. Rather, this was a black box of problematization that had to be broken open and manipulated to divulge information in order to realize the promise of scientific understanding and treatment of addiction. The belief was that with enough resources, skill, and technology, the brain could be probed and explored, made to reveal secrets about the neurochemical transmission of compulsive impulses – and that eventually, the biology of addiction might be brought under control.

CONCLUSION

Today, a "key point for the clinician to realize is that the proneness to relapse is based on changes in brain function that continue for months or years after the last use of the drug" (O'Brien 1997: 66). Environmental factors are still understood to be important, and do pose risks to abstinent addicts; but their danger is a function of their impact upon the physiology of the brain, and the resulting psychological states. Indeed, virtually everything about addiction, from the initial to the end results of chronic drug abuse – e.g., from the development of pathological cravings to the decision to continue to use drugs despite negative consequences – are thought to be linked to the brain.

This chapter has not, of course, begun to explore the emergence of neurobiological explanations of relapse and craving. It has only explored some of the ideas and studies that today's addiction scientists themselves identify as keys to contemporary understandings of addiction as a chronic brain disease. But by having described how physiology came to be placed at the centre of addiction science truth-making activity,

we have attuned ourselves to some of the fundamental issues about the changing nature, and interrelatedness, of what we know about the body and how we know about it.

This chapter has examined the early quest for objectivity in addiction science. The ability to obtain the truths of addiction, as we saw, involved a search for truthful research 'materials' – and the invention of ways of knowing the body as an object, rather than listening to the individual subject, involved substantial amounts of social work (cf. Jasanoff 2004). It was shown that not only did objectivity have to be invented through social, material, and technical means, but that it also had to be *successively reinvented.* Moreover, it was shown that objective facts are social in both their construction and their effects: they are achieved through work, but are also put to work, insofar as they provide new possibilities for human choice and action, and insofar as they can be mobilized to justify new studies, treatments, and endeavours.

Thus, we saw that when Himmelsbach 'got the idea' that addiction could be understood in terms of homeostasis, he and his colleagues at the Addiction Research Center had to develop methodologies and experimental designs which could test a set of hypotheses about the body's homeostatic responses to narcotic drugs. New ways of not only measuring changes in the body – and new ways of bringing about those changes – were devised that focused the attention of a new generation of medical researchers on the body's own processes of self-regulation. As a result of these new experimental strategies and epistemological principles, objective facts emerged about

the nature of addiction (as an acute, physiological condition) and of addicts (who used drugs to avoid the abstinent syndrome).

However, as was described in the latter half of the chapter, these assumptions began to change in the 1950s, especially in relation to the work of Abraham Wikler. As the principles of Pavlovian conditioning came to form an important element of the biomedical conceptualization of addiction, it became more difficult to determine how long an addict might retain the changes brought about by drugs which put them at risk of relapse, and the notion of a post-addict was brought into question. It became clear to an increasing number of researchers and clinicians that the physiological facts made possible by the introduction of animal experiments indicated that addiction was a more complex, more enduring biomedical problem than brief therapeutic interventions (such as acute care during withdrawal) could resolve.

Having described how early addiction science and the biomedical problematization of addiction reshaped what counted as important investigations, proofs, and observations for researchers, the stage is now set for ensuing chapters. We leave off at the point at which, by the mid-1960s, scientists were saying, on the one hand, 'we know that addiction is a chronic, relapsing disease on the basis of the body's physiology,' and on the other hand, 'we don't really know the physiology of the body, but we need to in order to understand addiction as a chronic, relapsing disorder.' The addicted subject thus came to be attributed with a new, 'mysterious physiology' that indicated a need for new sorts of treatments, new ways of thinking about addicted individuals, and hinted at new ways of dealing with social and political problems associated with drug use and abuse. The following two chapters examine how this black-boxed

physiology became the object of increased scientific study; and how, as increased knowledge of the brain and biochemistry were obtained, addiction came to be knowable in molecular detail, and treatable through precise neurochemical interventions.

CHAPTER 4: ADDICTION BIOPOLITICS AND THE NEUROBIOLOGICAL IMAGINATION

INTRODUCTION

Within biomolecular styles of thought, addiction has undergone a fundamental transformation from previous conceptualizations. It is no longer attributable, except among outdated thinkers, to a pathological personality, a problematic will, a behavioural disorder, or to anything like an individual soul or psyche. Today, addiction is a disease of the brain. This conceptual shift is not only of theoretical interest, for it has brought about a corresponding therapeutic reorientation: addiction, as a disease of the brain, has become treatable with new sorts of brain-targeting medications, which are understood to act on specific parts of the brain affected by drug use.

The preceding chapter demonstrated how the most fundamental assumptions about addiction that are held by brain scientists today can be traced back to concepts and research strategies that were developed by pioneering physiological researchers in the middle of the twentieth century. It showed in particular how, as new ways of thinking about and studying addiction as a somatic problem emerged, the nature of the addiction itself came to be redefined. The chapter took us up to the mid-1960s, when experimental scientific research methods had begun to be developed which provided evidence that elevated risks of relapse, hitherto considered merely psychological, were physiological matters that could be investigated in the 'manner of the natural sciences' (Wikler 1984 [1965]).

We will see, in chapters to come, how relapse has come to be represented in molecular detail within neurochemical models. This chapter, however, investigates more basic issues relating to the development of the brain sciences of addiction, namely issues regarding how it became possible to state, as a matter of scientific fact, that addiction *is* indeed a disease that manifests itself at the neurobiological level. It asks how it has become possible to think about addiction in terms of the molecular structures of the brain; and how these structures, and the processes in which they are involved, have been brought into existence.

The chapter examines the development of research into the molecular, laboratory sciences of addiction, not in terms of the unified theories and explanatory models that emerged in the late 1980s and 1990s, but in terms of a will to know addiction as a neurobiological condition. Why, in the late 1960s and early 1970s, did it become important or desirable to invest in research that could produce knowledge about, and therapies for, addiction as a disease of the brain? How, in this period, did addiction neuroscience move from being a marginal, almost non-existent field, to a well-funded specialty that is today understood to have played an important role in the molecular revolution within the brain sciences? What factors contributed to the ability of neuroscientists and pharmacologists to begin to specify the neurochemical pathways and neurophysiologic structures involved in addiction, and to devise pharmacological solutions for the medical, personal, social, and political problems associated with addiction?

Following this introduction, the first section of the chapter suggests that this will to know addiction as a brain disorder can be understood largely in terms of a confluence

of the professional concerns of an emerging group of addiction science researchers, on the one hand; and on the other hand, the interests of US policy-makers who were developing new strategies for waging what came to be referred to as the 'War on Drugs.' Both of these groups, it is suggested, became committed to a sort of 'neurobiological imagination' – that is, a way of thinking about and imagining addiction in brain terms, not necessarily in order to do away with considerations of individual and social problems, but to envision new possibilities for explaining and managing them. The neurobiological imagination is not investigated in terms of abstract ideas and vague convictions; instead, it is considered in relation to the social, political, and economic conditions which made it possible. The section focuses in particular on the adoption, by the US federal government, of biopolitical strategy for fighting drug problems – that is, a strategy that is based on a scientific management of the vital characteristics of human existence. It describes how, in relation to the War on Drugs, addiction increasingly came to be represented as a biological condition to be managed with the tools and insights of biomedical science, rather than a form of criminality to be eliminated through prohibition and legal repression.

The chapter then turns to a consideration of the productive power of these neurobiological imaginaries, and the products of early addiction neuroscience: the new biological objects, the new medical treatments, and the new forms of politics and control that it helped make possible. It focuses on how the neurobiological imagination was able to lead to neurobiological facts in two particular areas: first, the production of more thorough understandings of the neurobiological basis of drug action, including the construction of the precise neurological mechanisms affected by drugs; and second, the development and testing of new addiction pharmacotherapies.

It does not explain these developments in terms of politics, but rather investigates how politics combined with intellectual and technological factors to make the contemporary science of addiction possible, and to make the community of molecular neurosciences an obligatory point of passage for addiction studies.

Biopolitics and the War on Drugs

The period under investigation in this chapter – roughly between the mid-1960s and the mid-1970s – is often described as marking the beginning of the contemporary era of addiction medicine, in which 'rational' approaches have been devised to deal with the harms and problems associated with drug use. The characterization of this era as one of rational addiction treatment is to considerable extent accurate and valid, insofar as it distinguishes a strategy of intervention based on deductions from general principles obtained through scientific inquiry, and not on moral or empirical forms of reasoning. However, 'rational' tends to be used synonymously with other adjectives such as 'value-free' and 'politically neutral' – adjectives which would be misleading if applied to this era. This period was one in which the US government sought to make addiction and drug-related social problems matters of the brain, requiring that its mechanisms be brought into the realm of explicit calculations. Thus, if we want to capture what is most distinctive about the contemporary era, it is perhaps more appropriate to describe the shift that began to occur in 1965 as a movement towards a biopolitics of addiction. For, as this chapter demonstrates, politics arise at every level of addiction neuroscience, from disputes between experts and decisions about the allocation of finite research resources, to the development of strategies for regulating the conduct of the subjects of addiction medicine and the direction of legal and public drug policies.

This biopolitics of addiction did not consist of a simple medicalization of social control; instead, it involved a broader realm in which truths about drug use and human physiology are produced, and in which these truths began to form the basis of operations of monitoring, optimizing, and organizing individuals and populations. Biopolitical considerations of course can not entirely explain the nature of the facts and treatments that emerged during the early years of the War on Drugs; but they can help explain why, as the US government sought to make the science of addiction an agent of social transformation, it spent large amounts of money to enrol and support an emerging community of addiction scientists.

By the mid-1960s, a group of clinicians and researchers from a diverse range of institutional affiliations, scholarly traditions, and geographical locations had become convinced that addiction could be thought about and acted upon rationally, in terms of neurochemical processes. Molecular models of addiction were still relatively undeveloped, and theories, explanations, and perspectives often existed in contradiction with one another. While this community had by no means come to agree on the nature of addiction, it did coalesce around assumptions that knowledge about addiction not only could, but should, be acquired through clinical and laboratory experimentation on physiological and neurobiological processes. Notions about what addiction was, exactly, may have been open to a certain amount of dispute and change, but in order to be proven as facts, any such notions were required to adhere to the rationalist principles of molecular science.

However, in the 1960s this community was relatively small, and was marginal to both mainstream neuroscience researchers and mainstream addiction therapeutics. On the

one hand, basic neuroscientists and psychopharmacologists did not consider addiction a very prestigious or interesting field, and support for experimental research in this area was not easily come by. On the other hand, biological perspectives on mental illness were still marginalized within American psychiatry and the clinically-oriented field of addiction studies (which were dominated by psychoanalytic perspectives). As one biological psychiatrist who was trained in that atmosphere recalls:

> A psychiatric trainee who expressed a strong interest in basic biological research was regarded as somewhat peculiar, perhaps suffering from emotional conflicts that made him or her avoid confronting "real feelings." An interest in science was regarded almost as sick, some sort of stratagem to avoid the psychoanalytic issues that *mattered* by fleeing to science" (Snyder 1989: 10).

So amongst addiction experts in the 1960s, there was not much agreement at all that addiction really *was* a brain disease, and amongst brain scientists there was no strong conviction that addiction was an important research area.

The fortunes of this community began to change significantly in the latter half of the 1960s, however, as drug abuse in general, and heroin addiction in particular, were becoming causes of increasing alarm both among public health officials and the American public, and as the US federal government began to orient its drug policies towards biomedical approaches. Although the case of methadone (to which we will soon turn) may have been an early indicator of a new approach to dealing with drug use by the US government, it was when Richard Nixon began his presidency in 1969 that federal agencies began to increase support dramatically for the emerging community of addiction brain scientists. Nixon entered office at a point when tumultuous social change in the US, as well as worsening political and military problems resulting from the Vietnam war, had led to a sense of national emergency.

Drug problems were identified as one of the main sources of crises on both of these fronts by the US government: the military had reports that over 15 percent of returning war veterans were addicted to heroin (Jaffe 1999), and as drug use among white, middle-class youth began to increase, addiction was considered by some to be reaching epidemic proportions. One scientist reflects that:

> Among national authorities, apocalyptic visions of opioid-dependent armed United States soldiers, as well as similarly afflicted anti-war, anti-American anarchists roaming the streets looking for a "fix," provided necessary impetus to both the Executive and Legislative Branches of the Government to authorize funding for expanded research and treatment of opioid dependence" (Julius 1976: 5).

Nixon made a public commitment to reducing drug use and crime rates – which he saw as interrelated – and in 1971, created the Special Action Office for Drug Abuse Prevention (SAODAP) to administer the resources of the federal government. Nixon appointed Jerome Jaffe – a former student and junior colleague of Abraham Wikler at the Addiction Research Center – as the first director of SAODAP, with the mandate to steer the US's national drug policy toward a rational, pharmacological approach to addressing issues of drug abuse and addiction.

Jaffe, already committed to a conception of addiction as rooted in an individual's biochemistry, seized the opportunity to direct national drug policies toward the position of his thought community. Although he was enrolled by the US government to help federal authorities achieve their goals, Jaffe also enrolled the federal agencies in support of addiction science researchers. US policy-makers made large amounts of funds available to make it possible to 'do something' about the addiction problem; but they were not very interested in supporting basic research that might or might not be of practical use at some unknown point in the future. When Congress created a budget for the vast expansion of medical treatment for heroin addiction in the 1972

Drug Abuse Office and Treatment Act, Jaffe managed to divert some of the funds marked for establishing treatment clinical programs towards the support of basic laboratory research. Jaffe recently reported that "[t]hat early funding put money into things that really had an amazing impact. [...] I think we accomplished something in really jump-starting the research activities in substance abuse and – not the least – we laid the foundations for the current treatment system" (1999: 23).

Jaffe's role, and indeed the role of the community of experts he represented, might be compared to Howard Becker's 'moral entrepreneur' (Becker 1963). But whereas a moral entrepreneur might be said to be concerned with identifying a particular group of deviant or "bad" individuals with a particular social problem, addiction experts were concerned with reconceptualizing a social and legal problem as a medical one, and were committed to the view that drug addicts should be studied and treated, rather than penalized and imprisoned. And insofar as Jaffe and his fellow addiction scientists were able to position themselves as part of the solution to the problem of drug use, they were able to generate unprecedented opportunities for the community of neuroscience and pharmacological researchers.

These researchers became embedded in a new sort of juridically-willed research economy, in which legislative powers provided a variety of resources that allowed the field of addiction science to develop at a rate it would otherwise have been incapable. The government's official support of scientific addiction research seemed to indicate that this field was an important, high-priority area of research, and both new researchers who had yet to decide on academic specializations, and mid-career scientists from other fields, began to be attracted to addiction studies as a result of

generous new funding schemes. As a prominent neuroscientist who was attracted to the field in the early 1970s notes, "neuroscience – like other dynamic, disputatious fields ranging from cosmology to cancer research – often has as much to do with staking out territories, establishing priorities, and defending reputations as with furthering the cause of science" (Snyder 1989: 3). The support and endorsement of the US government should not be underestimated in accounting for the dramatically rapid development of the field and its rise not only to legitimacy, but even prestige.

Thus, the commitment to realizing the neurobiological imagination appears to have resulted in part from a confluence of interests, rather than a shared and unified goal. The US government was looking at ways of controlling drug abuse and the social, legal, and economic problems associated with it, and was willing to finance basic and clinical research into addiction in the hopes of developing more technical, efficient means of dealing with addiction. The community of addiction scientists, on the other hand, was not as much concerned with reducing the negative social and economic impacts associated with addiction as it was interested in securing funding for their research, status for their specialization, and in developing biomedical solutions for what they understood to be a biomedical problem.

But to identify this confluence of interests is not to suggest that the interests of these two groups were one and the same, of course; rather, their interests were such that each was able to enrol the other in the pursuit of its own goals. The US government wanted at least part of its War on Drugs to be based upon scientific authority; and Jaffe and other researchers were able to increase their authority because of the resources they were able to access as a result of being brought on board as part of the

war effort. But mutual enrolment is *not* the same thing as conspiracy! There is no evidence to suggest that scientists and government colluded to fabricate the 'facts' of addiction in order to artfully legitimate new strategies of social control. The alliance cannot explain the nature of the facts of addiction that we currently have; but it *can* help explain how the brain sciences became an obligatory passage point for producing truths about addiction, and how parts of the brain came to be identified and targeted as the locus of drug-related problems.

The following sections examine how, within the context of the American biopolitical concerns of the 1960s and 1970s, researchers moved from imagining addiction as a disease of the brain, to obtaining neurobiological facts about the condition and its treatment. Today, models of and therapies for addiction are understood as inextricably linked together within a single explanatory framework (e.g., therapies act upon what is modelled). However, this logical integration only arose after the early neuroscience experiments described below were undertaken, as models of addiction changed over time to explain what effective pharmacotherapies were thought to do. Since this chapter focuses on the origins of the neurochemical style of thought – the point at which it was only becoming possible, in vague, often hypothetical terms, to suggest that addiction does indeed have a neurobiological basis – it describes the genesis of the brain structures implicated in addiction on the one hand, and the development of pharmaceutical interventions for addiction on the other, as they arose: namely, as events that occurred relatively independent from one another.

THE BIRTH OF A BRAIN DISEASE

This section examines how it has become possible to know addiction in terms of the biochemistry of the brain – and indeed, how the neurobiological 'stuff' of addiction

has been brought into physiological existence so that addiction can be thought about and acted upon at the molecular level. It focuses on the pivotal period in which it was becoming possible to identify the specific 'receptor' sites in the brain on which drugs act and to implicate these receptors in the actions of addictive drugs. Specifically, it provides a genealogical account of the work of the first laboratory study that is considered to have adequately proven the existence of the (up-until-then hypothetical) receptors. Tracing the origins of the demonstrated receptors back to the point at which they had not yet become objective, factual objects, the section suggests that the existence of receptors was not simply 'demonstrated,' but that receptors were actually brought into existence within a medium of technological, political economic, and theoretical contingencies.

It was in 1973 that researchers at John Hopkins Medical School reported in *Science* (1973) that, thanks to the new experimental procedure of radioligand binding, they had demonstrated the presence of opiate receptors in nervous tissue: that is, the neurological structures to which heroin and similar drugs attach themselves in order to affect the brain. The demonstration of the opiate receptors contributed considerably to the consolidation of the biomolecular thought style within the field of addiction studies. The ability to define exactly where chemicals interacted with the central nervous system met one of the fundamental criteria for establishing addiction as a disease of the brain, and thus helped to secure the future of basic neuroscience research on addiction. But the demonstration of the existence of receptors was also a significant event for neuroscience in general. It seemed to indicate the possibility of moving from knowledge of the brain as an organ, to knowledge of the brain in terms of molecules; and it is considered to mark the beginnings of the molecular revolution

in neuroscience – and an era in which neuroscience could obtain knowledge of the workings of the mind in the smallest possible detail.

With the publication of their article, "Opiate receptors: demonstration in nervous tissue", Solomon Snyder and his graduate student, Candace Pert, brought to an end decades of speculation about the existence of receptors, and the reality of addiction as a neurobiological condition. Finally, it seemed, the truths of the brain had been brought to light, indicating that, indeed, addiction was a matter of the brain. But for anyone who might wish to explain these developments as the result of a discovery of the unmediated nature of the brain, Snyder's publication of a book-length account of the opiate-receptor research of the 1960s and 1970s would be disconcerting.

For Snyder, the development of knowledge about the molecular anatomy of addiction and the brain cannot be dissociated from either the political economy or the intellectual and technological history of the field of research. In *Brainstorming: The Science and Politics of Opiate Research* (Snyder 1989), he suggests that the facts obtained about receptors are best understood as contingent upon a range of historical and local factors. Indeed, Snyder's account can be read in support of the claim that Pert and Snyder had not been attempting to let the neurological truths of addiction appear through an experimental process designed to let the brain reveal its essential nature, as much as they had been engaging in the production of facts along lines already partially specified by (as Snyder's subtitle indicates) the science and politics of the era.

The introduction of this chapter touched upon some of the general political issues relevant to the emerging field of addiction neuroscience (including the Vietnam War and the countercultural movement). Snyder demonstrates how 'politics matter in science' on an individual level by relating his own entry into the field of addiction neuroscience to the concerns of the US government. Snyder asserts that his own interest in opiates "cannot be divorced from American political events in the early 1970s" (Snyder 1989: 6), when Nixon's recently-declared War on Drugs led to the funding of drug abuse research centres, one of which was awarded to Snyder's group with a specific mandate to study opiate receptors (Snyder and Pasternak 2003). Snyder and his team had not chosen either addiction as a field or the opiate receptors as an object of investigation because of a particular interest in or concern about opiate addiction; indeed, Snyder admits that in the late 1960s, he 'hardly knew heroin from horseradish' (Snyder 1989). Opiate receptors were chosen as the object of investigation simply because funding had been readily available for researchers whose projects could be aligned with the US government's War on Drugs.

But while Snyder's account attests to the relevance of politics to science, it by no means suggests that politics determine the ultimate appearance of scientific truths. Snyder also demonstrates an awareness of how the actual conceptualization and execution of scientific research on opiate receptors was reliant on the theoretical and experimental resources provided to him by his peers and predecessors. He notes that:

> Unlike the President of the United States, an investigator cannot simply call in newspaper reporters and photographers, declare war on a scientific problem, then walk into the laboratory and solve it. To carry out a program of experimental research in a given area, the scientist must have, at minimum, some strong hunches about what is going on and what experiments might be successful (Snyder 1989: 9).

Although Snyder is clearly aware that pre-existing understandings, procedures, and technologies played highly significant roles in determining the direction of his research, his account of the science of the receptors focuses mainly on developments of the late 1960s and early 1970s, especially those relating to the experimental laboratory procedures of radioligand binding that made it possible to produce results that were understood to demonstrate the existence of receptors. However, it is possible to trace the genealogy of the receptors back much further than the 1960s – back to a point when receptors appeared only as part of vague suspicions about the mechanisms of drug actions.

Receptors, before the fact

When the concept of receptors was invented in the early 20th century – long before the advent of molecular neuroscience – 'receptors' did not exist as the same physiological objects that exist today. Receptors were originally conceptualized as a purely hypothetical site at which potent drugs were hypothesized to bind to living tissue in the nervous system. The notion of a receptor was devised to explain how minute quantities of some drugs could have dramatic, general effects on an organism: since in such cases there did not appear to be enough of a substance to spread throughout the body, it was reasoned that particles might only initiate a first step in a chain of biological events within the body.

Despite lack of direct evidence of the receptive site or structure, receptors became an increasingly important concept through the first half of the 20th century, as biochemistry and pharmacology were rising to prominence in the brain sciences, and materialist conceptions of life began to replace vitalist ones. This was especially the case as scientists came to believe that the brain controlled bodily functions through

the process of neurotransmission – the sending of information through the release of endogenously-produced chemicals, i.e., neurotransmitters. If chemical messages (rather than, for instance, vital, electric pulses) were sent to and from the brain, this seemed to require structures that could receive such information (and could in turn activate other processes).

While pharmacologists could imagine, and even demanded, that receptors existed as distinct structures, the exact nature of receptors that began to appear in the 1970s was not immediately evident or obvious to the earliest addiction neuroscientists. Prior to the 1950s, when the expectation was that the molecular properties of drugs would contain the key to explaining addiction, a sort of 'addiction receptor' upon which all the addictive classes of drugs acted was imaginable. But as knowledge of the molecular properties of drugs of abuse increased, it became all but impossible to discern any significant structural similarities between the different classes of substances presumed to be addictive. As one pharmacologist reflected:

> If one enumerates the common drugs of addiction: alcohol, barbiturates, cocaine, cannabis, mescaline, opiates and synthetic analgesics, there is no basic structure common to all that I can see, except that they are organic or carbon-containing compounds. One cannot take a molecule of possibly-addiction-producing drug and strip from it its trimmings, to leave an active core in which the potency for addiction resides (MacDonald 1954: 21).

Because drugs of addiction were so structurally diverse that they precluded the possibility of addiction being explained in chemical terms, the burden of causation needed to be shifted elsewhere. If the addiction-producing potential of drugs of abuse could not be located in the physical properties of the drugs themselves, it seemed likely that understandings of addiction would require more detailed knowledge of the interactions between drugs and biochemistry. During the 1950s, cutting-edge

pharmacologists began to suggest that on a molecular level, drugs of addiction could be understood as 'enzyme inhibitors' whose pathological effects related to their ability to disrupt the normal functioning of neurotransmitters (MacDonald 1954).

By the 1950s, the observation of interactions between addictive drugs and neurotransmitters could be made fairly easily in laboratory experiments. Experiments on guinea-pig smooth-muscle tissue, for example, indicated that morphine could inhibit the effects of acetylcholine (Paton 1957; Schaumann 1957) and serotonin (Medakovic 1958). But it was not possible to observe the exact molecular process by which a drug could cancel out the effect of a neurotransmitter (much less cause addiction). By dissecting test subjects, it could be demonstrated that both chemicals were still present and intact in the subject, and this led to the conclusion that the drug did not cause the neurotransmitter to be destroyed or eliminated. But the precise process through which the drug interfered with the neurotransmitter's action was not understood.

Before precise molecular relationships were formulated between interacting chemicals and brain parts, this relationship came to be represented in vague terms of a competition for influence over the body's processes. A drug or chemical that produced an observable effect in a research subject was called an agonist, and a chemical that interfered with the effects of an agonist was called an antagonist (etymologically, of course, the terms refer to opposing combatants in a struggle). It was reasoned that if an antagonist could counteract the action of an agonist, the mere presence of a drug in a subject was not enough to produce observable effects. The agonist, then, had to form a physical bond or interaction with a specially receptive

part of the biological host; and the antagonist was assumed to interfere with the actions of an agonist by preventing or disrupting the agonist-host interaction at this receptive site. The specific nature of the site, however, remained unspecified for some time.

The induction of receptors into theories of addiction

In the early- to mid-1960s, a growing number of researchers began to speculate about how the homeostatic abstinence syndrome described by Himmelsbach might be explained in biochemical terms (e.g., Goldstein and Goldstein 1961; Jaffe and Sharpless 1965; Shuster 1961). Emmelin (1961) and others suggested that when the regular supply of neurotransmitters have been suppressed by an antagonist drug, the brain adapts to the resulting disruption in normal functioning by increasing its responsiveness to the effects of neurotransmitters (i.e., by becoming more responsive to neurotransmitters, the brain could function normally with fewer molecules of the substances). Although the exact biological mechanisms involved in such adaptations remained in dispute, the neurobiology of addiction came to be understood as a result of the brain's attempt to counteract the drug-induced interference with neurotransmitters. Drug tolerance and withdrawal both came to be modeled in neurobiological terms: as the brain's sensitivity to neurotransmitters increased, drug effects became less pronounced, and thus tolerance to the drug developed. When the drug was withdrawn, the 'supersensitive' brain mechanisms were overexcited by the return of neurotransmitters to normal levels, and this overexcitation led to withdrawal symptoms.

In 1963 a review of the current pharmacological approaches to tolerance and dependence reported that such hypothetical accounts of tolerance and dependence

were plausible given then-current scientific evidence (Seevers and Deneau 1963). At the time, however, the precise structures and mechanisms involved could only be posited hypothetically. This was the state of affairs even when, in 1965, British pharmacologist Harry Collier published an article in *Nature* that offered a theoretical explanation of addiction in terms of receptor activity. Based on the concept that "a chemical substance acts on a living system through its molecules becoming attached to particular sites (receptors) on cells", Collier explained that the brain's homeostatic adaptations involved these receptors (1965: 181). The brain compensates for interference with neurotransmitters, he hypothesized, by increasing the number of receptors on brain cells that respond to neurotransmitter activity.

Collier's "general theory of drug dependence by the induction of receptors" (Collier 1965) became one of the most influential and widely cited papers in addiction science research, despite the fact that it appears to have relied upon the force of imagery and rhetoric more than substance or proofs. Over thirty-five years after its publication, the renowned pharmacological researcher John Littleton noted that Collier's theory was based on tentative, almost unsupportable claims. In 2001 Littleton writes that:

> Since in 1965 "receptors" were only a vague concept [Collier's] hypothesis was outrageous and untestable, as well as brilliant. Its publication at that time does credit to the imagination of the Editor of *Nature*, and to Collier's powers of persuasion. After this, regulation of receptors rapidly took center stage as a potential explanation for the [biological basis of addiction] (2001: 89).

Retrospectively, Littleton seems surprised at the readiness with which Collier's work was embraced, and points out that the neurological receptors Collier deployed in his explanations did not demonstratively exist until almost a decade later. He also notes that other cellular and biochemical explanations for addiction had been suggested at the time, but captured the popular imagination much less. Collier's hypothesis was

attractive because it was tidy and elegantly simple; and Littleton warns us that the 'beauty' of the proposal should not be underestimated in understanding why it was seized upon so readily.

And so, from 1965, receptors became a major focus for researchers and research-funding agencies even though proof of the receptor remained indefinite up until 1973. Nervous tissue, in this period, became subject to a series of 'phenomenotechnological' operations (cf. Bachelard 1984): brain tissue was brought into laboratories, probed, measured, and experimented with until it yielded to Pert and Snyder the results that brain scientists, since at least 1965, had imagined would be produced. Even before Pert and Snyder's successful experiments, the discovery of opiate binding sites seemed virtually inevitable, and the existence of receptors was taken more or less for granted. In 1972, for example, the author of a review of leading pharmacological explanations of addiction stated that in order for such explanations to appear plausible:

> It is necessary [...] to accept that a drug is a chemical which when administered gains access to a cell and affects the function of the cell by one of a variety of means, bio-physical, bio-chemical, etc. This alteration is triggered off by the drug molecule being attached no matter how tenuously to a part of the cell capable of binding it (the receptor). The drug molecule produces changes as a consequence of its adhesion which may start a complex chain of events involving temporary or permanent change in the function etc. of the affected cell (Graham 1972: 83).

Despite the hypothetical status of the 'receptor' – in 1972 it was not a definite physiological entity – it was necessary to accept its existence for pharmacological theories to make sense. And thus it is not entirely surprising (though neither was it entirely necessary) that once such conceptualizations had arisen, the experimental

means developed to select, purify, filter, and shape the physiological phenomena that came to be identified as receptors.

The taken-for-granted reality of receptors before the experimental facts helps explain why, although the demonstration of opiate receptors was undoubtedly an important historical event, it did not immediately precipitate a drastic change in neurological theories about drug addiction. These theories had already been based upon the existence of receptors; and thus, there was not a clear break between literature on 'hypothetical' neurological receptors that was written before the demonstration of opiate receptors by Pert and Snyder and the literature on 'real' receptors. But indeed, it might be exaggerating to refer to the receptors as having taken on a distinct physiological reality in 1973, for they still remained relatively undefined in terms of structure and function. The 'discovery' of opiate receptors provided virtually no information about the physiological objects themselves, except that there was something in the brain to which the opiates attached. The radioligand binding technique of Pert and Snyder basically consisted of bathing brain tissue in radioactively-labelled opiates, rinsing the tissue off, and seeing what quantity of opiate molecules had binded with the tissue.

And so three years after Pert and Snyder's experiment, it was still acknowledged that the "final and conclusive evidence for an opiate receptor site must await its isolation and structural determination" (Hughes 1976: 201). At this point, then, all that existed of receptors, really, was the interpolation of structure: that is, the structure of receptors had only been called into enough of an existence that it could imperfectly fill the factual void between what was known about the chemistry of drugs and the

chemistry of the brain. Nevertheless, this was enough to precipitate the beginnings of a molecular revolution in the brain sciences (Snyder 1989), and to make possible a legitimate neuroscience of addiction. It also helped bring about the therapeutic reorientation towards what in biomedical literature is referred to as 'rational addiction pharmacotherapy:' addiction treatments which are understood to work not on the individual's *self*, but are instead understood to work at the most mundane and pragmatic level possible: on the molecular structures of the brain.

MOBILIZING MEDICATION RESEARCH AND DEVELOPMENT

The same year that Collier published his receptor theory of addiction also marked the beginning of the contemporary era in which the US government relies on "science, not ideology or anecdote" (NIDA n.d.-h) to provide the basis of dealing with addiction as a problem of personal and public health. It was in 1965 that the US government legally sanctioned a national program of clinics in which physicians could prescribe the opiate drug methadone to heroin addicts as a form of treatment. This regulatory shift reversed the federal prohibition of such uses of narcotics which had existed for decades (Jaffe 1999), and marked the end of what Courtwright (1989) and Acker (2002) refer to as 'the classic era of narcotics control.'

Methadone was a narcotic analgesic that was invented during the second World War and which, in the late 1940s, came to the attention of US Public Health Service researchers at Lexington. Although it was originally investigated as a drug that could reduce the withdrawal symptoms during the acute detoxification of heroin addicts (Isbell, et al. 1948), in the 1960s, Vincent Dole and Marie Nyswander theorized that methadone could be used as a long-term treatment. Dole and Nyswander had come to the same conclusion as their colleague Abe Wikler about the chronic nature of

addiction, and argued that detoxification did not constitute an adequate form of treatment for addiction. They reasoned that heroin use caused 'synaptic imbalances' in the brain that lasted long after detoxification, and that these imbalances created a persistent, visceral 'need' for drugs. With no known means of restoring normal neurological functioning to drug addicts, Dole and Nyswander proposed that the best treatment for heroin addicts was one which restored social functioning by pharmacological means.

Methadone was known as an opiate antagonist because it blocked the effects of opiate drugs – an action that prevented individuals on methadone from obtaining any pleasure from the injection of heroin (presumably because methadone competitively displaced heroin at receptor sites). Because of its partial antagonist properties, methadone would prevent the individual from getting high should he or she attempt to self-administer heroin. But methadone was only a 'partial antagonist;' it also had partial *agonist* properties, producing some narcotic effects of its own (though these were much less pronounced than the effects of heroin). Methadone was therefore understood to be a drug of addiction itself – but one that was considerably more benign than heroin. It could be prescribed at dosages that were low enough to produce relatively little euphoria, but that could still 'maintain' the individual's physiological dependence without causing the withdrawal symptoms that normally resulted when an individual stopped taking heroin.

Dole and Nyswander suggested that continual administration of methadone to addicts would maintain the balance of the addict's dysfunctional neurology and thereby allow him or her to avoid the drug-seeking thoughts and behaviours that interfere with a

normal lifestyle (Dole and Nyswander 1965). In doing so, it thus enabled the clinician to "free the patient from heroin-oriented hustling, allowing participation in the treatment and rehabilitation program and development of less destructive interests and activities" (Blaine and Renault 1976a: 2). Thus, 'methadone maintenance therapy' (MMT), as Dole and Nyswander's treatment came to be known, was formulated as a form of indefinite treatment that was "corrective but not curative" (Dole 1988: 3025). Its effectiveness was evaluated in terms of its ability to produce behavioural changes in patients, rather than its ability to eliminate addiction to narcotics.

In the late 1960s, methadone provided a means of satisfying the political imperative of 'doing something' about the increasingly urgent 'problem' of addiction. Moreover, as formulated by Dole and Nyswander, it approached the problem from a rational, biochemical logic. It was thus seized upon by policy makers, who developed a national program of clinics in which physicians could legally prescribe methadone to individuals on a continual basis. However, within the first few years of instituting a national program of methadone clinics, several problems were identified with the treatment approach.

The duration of methadone's effects was not long enough to protect the individual effectively from the vulnerabilities of craving or the effects of heroin for a full twenty-four hours when administered at doses low enough to avoid pleasurable effects from arising; in order for daily administrations to prevent relapse to heroin use adequately, methadone had to be prescribed in euphoria-producing quantities. This was a problem because by the end of the 1960s, politicians had become sensitive to

"anti-dependence" sentiment that had arisen in some US constituencies which questioned the government sponsorship of the administration of pleasure-producing, addictive drugs as a form of treatment (Julius 1976: 9).

The fact that methadone maintenance was based on regularly-scheduled, daily administrations supervised by clinicians was also identified as a problem, since these appointments often interfered, both practically and psychologically, with the efforts of patients to attempt to lead a 'normal' lifestyle. A compromise was made that allowed individuals who had successfully remained in therapy for at least three months to receive a four-day supply of methadone, which they could self-administer in order to reduce visits to the clinic. But it soon became clear that these supplies were often diverted away from therapeutic use, as individuals could take several doses at once to obtain a more intense euphoric state, or could sell their supplies to other individuals. Misuse of methadone was associated not only with illegal activity (i.e., drug trafficking), but also with several hundred deaths a year, especially as a result of ingestion by non-tolerant individuals, who were many times more susceptible to the effects of narcotics (Blaine and Renault 1976a; Gardner 1970). Moreover, by the early 1970s a number of studies began to question the claims of the general effectiveness of methadone maintenance altogether (Connor and Kremen 1971; McLeod and Priest 1973).

In efforts to overcome the problems associated with MMT, the US government sponsored research into levo-alph-acetyl-methadol (LAAM). Like methadone, LAAM was a partial agonist, but it resulted in less pronounced euphoric and dependence-producing effects, and had a significantly longer duration of action which

required only three maintenance doses per week. However, LAAM still had a potential to be misused and diverted, and was still, as a pleasure-producing drug that created dependence, considered a drug of addiction (Blaine and Renault 1976b). Thus, as with methadone, the therapy was vulnerable to criticism on the grounds that it simply replaced one addiction with another, and moral and political concerns about the legitimacy of both of these partial agonists arose.

In order to overcome the problems associated with agonist treatments, agencies of the US government and allied scientists increasingly focused on the development and testing of pure opiate antagonist treatments – that is, medications which could neutralize the effects of heroin and other narcotics, but which would produce no effects of their own. Although some opiate antagonists such as nalorphine had been synthesized in the 1940s, it was nearly two decades until one was discovered that did not produced unpleasant side effects so severe as to limit their use in human subjects. It was only in the mid-1960s that, as a result of progress in research and development of opiate antagonists, as well as in theoretical innovations, the first steps toward the practical application of these medications were made (Jaffe 1976).

The development of theoretical rationales and clinical testing of antagonist medications as addiction treatments began within a closely-linked and influential community of researchers. In 1965, the research group of William Martin, a junior colleague of Wikler's at the Addiction Research Center, began to report research which suggested that an adequate narcotic antagonist therapy was close to being found. Martin's team had found that a new antagonist, cyclazocine, differed from others opiate antagonists studied up until this point, insofar as it did not produce side

effects severe enough to preclude the possibility of their clinical use (Martin, et al. 1965). Such a medication would not only be politically desirable, but would also represent an ideal candidate for treatments based upon the conditioning theories that were rising to prominence in the 1950s and 1960s. Martin's work on cyclazocine provided one of the first rationales for the use of antagonist medications in addiction treatment – a rationale based on Wikler's (1984 [1965]) conditioning theory of relapse:

> On the basis of our studies, 4 mg per day of cyclazocine will provide protection against the euphorogenic actions of large doses of narcotics, prevent the development of physical dependence, and will thereby control the pharmacological actions which are held responsible for narcotic addiction [...] Wikler stated that two of the most important reasons for relapse of the abstinent narcotic addict are conditioned abstinence, which may be evoked by stimuli that have been associated with the addict's hustling activity to acquire drugs, and reinforcement of drug-seeking activity through repeated reductions of abstinence by drug. It is possible that in subjects who attempt to readdict themselves while receiving a narcotic antagonist such as cyclazocine, there may be extinction of physical dependence and drug-seeking behavior" (Martin, et al. 1966)

Martin thus reasoned that antagonist therapies could render the taking of opiates unpleasurable, and therefore allow drug-taking cues and activities to be disassociated from reward responses.[7]

Although cyclazocine was ultimately discarded due to side effects, by the late 1960s, narcotic antagonists had generated a great deal of enthusiasm among Martin, Wikler, and many of their civil servant colleagues who were employed in government-run research facilities and laboratories such as those of the Addiction Research Center, working with state-apportioned budgets, and who are today considered "the giants who defined the theories of addiction that continue to shape the research agenda of the

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[7] Martin's formulation of the therapeutic problem is here still rather general, and refers to 'the addict' in behavioural terms that do not indicate much of a concern with neurochemical mechanisms. But elsewhere he explains addiction and the use of antagonist therapies in neurological terms, which link up theories and findings of molecular pharmacologists and basic neuroscientists.

field" (Meyer 2003: 1465). The narcotic antagonists did, indeed, come to be considered the most promising agents for addiction pharmacotherapy, but the search for the key to rational treatment was not guided, or made possible, by intellectual, professional, or scientific factors alone. Indeed, researchers themselves point out the central importance of political and economic factors in the development of antagonist medications.

Drug testing and development was an expensive, time-consuming, and resource-intensive process (especially in the days preceding the automation and industrialization of pharmaceutical laboratories), and through the 1960s, significant amounts of funding for researchers had not been readily available from the US government. Moreover, pharmaceutical companies were not very interested in this research, since (given the relatively small population of consumers of addiction treatments) the potential returns on investments for bringing narcotic antagonists to market for addiction treatment were perceived to be very limited. The opiate antagonists were "shunned by the pharmaceutical industry, due primarily to the ever-increasing developmental costs associated with new drugs" (Willette and Barnett 1980: v), and thus were in danger of becoming members of the class of "orphan" drugs which are left undeveloped because of their low commercial value.

As the future of these pharmacotherapies was brought into question, so too were the theoretical formulations of addiction associated with them. Demetrios Julius notes that in fact, the early studies of Wikler, Martin, and their colleagues on the therapeutic use of narcotic antagonists in addiction treatment "might have faded into textbook obscurity had it not been for a number of concurrent social and political events that

were rapidly developing" (1976: 5). These social and political events were the same ones that, as described above, inspired the US government to invest heavily in scientific research into the opiate receptors. Addiction had been identified as a crisis of almost epidemic proportions both among soldiers returning from Vietnam and among an increasingly large proportion of experimental and radical youth. Although there were some indications of a slight decrease in the rate of addiction in the US in the early 1970s, intense governmental pressure remained focused on expediently achieving promising solutions to this political problem.

On the basis of Martin's preliminary investigations, and the more general convictions of the neuroscience thought community, pure antagonists seemed to offer the potential for a new sort of therapy that could quickly and easily be deployed in the War on Drugs. Thus, during the 1970s, attempts to develop a "new magic bullet" for addiction treatment intensified significantly. Leo Hollister, one of the scientists involved in government-sponsored antagonist research, somewhat cynically described the government's rationale: "What could be simpler than to block completely the action of narcotics and thus make their use unrewarding? And what could be simpler than allocating a great deal of money to get these magic bullets into the patients?" (Hollister 1976: 45). Government officials created special provisions for the fast-tracking of research into opiate antagonists which allowed for the circumvention of the lengthy procedural and experimental requirements that were standard for drug development. Hollister notes that "[s]o great was the rush that ordinary administrative procedures were not considered adequate. One could not take time to write grant applications for the study of narcotic antagonists, nor even to solicit ordinary contracts" (Hollister 1976: 45).

Whereas the normal procedure for developing antagonists would have involved providing funding to numerous independent researchers, SAODAP appointed the National Academy of Sciences (NAS) – a scientific body of such high esteem that it could act as the 'sole source' of evidence – to coordinate a series of antagonist trials. Hollister, who was appointed to NAS as a member of its Committee on Evaluation of Narcotic Antagonists (CENA), noted that as a result of political interventions the conditions of the study "were certainly far removed from those which would ordinarily be done by deliberate, thoughtful clinical scientists" (1976: 46). But while these conditions of truth-production may have raised some questions about the objectivity of the findings of these studies, they nevertheless made possible the generation of enough evidence to secure the future of narcotic antagonist therapies in addiction treatment.

During 1973 and 1974, NIDA and SAODAP contributed more than five million dollars to the investigation of a variety of different antagonists, including naloxone, cyclazocine, pentazocine, and naltrexone, in order to discover an optimal candidate for antagonist therapy. By the end of 1973, naltrexone had been singled out as a the most promising therapy: a powerful, long-lasting antagonist which neither produced any physiological or psychological effects in patients (which would indicate a medication's agonist properties) nor any of the dysphoric side-effects of earlier antagonist candidates (Martin, et al. 1973).

Naltrexone was considered to be "almost a non-drug because of the lack of discernable effects other than its opiate-blocking capacity" (Julius 1976: 9). The

purely antagonist effects were of considerable importance in determining judgments about the desirability, even necessity, of pushing forward with naltrexone research. As the director of SAODAP, Jaffe noted that he felt pressured to support research efforts on the development of pure opiate antagonists such as naltrexone, regardless of his own scientific opinions on the matter:

> [E]ven if I had not felt that the development of naltrexone was worthwhile, I would have felt obliged nevertheless to bend every effort toward its clinical development. Influential members of Congress had become enthusiastic about the possibility of a non-dependence producing pharmacological alternative to the use of methadone (1976: vi).

Jaffe did, however, support naltrexone research: he, along with many other clinical and basic scientists, thought that partial agonist therapies such as methadone were useful, but insufficient, treatments for addiction.

With the goal-oriented governmental coordination of naltrexone research, it soon became clear that naltrexone could be considered a pharmacological success. It was found to occupy and block the specific biochemical sites of opiate action, thereby preventing opiate agonist drugs such as morphine and heroin from producing any physiological effects.[8] Thus, the drug appeared to meet all of the major criteria that had been established by scientists for an ideal candidate for use in the rational pharmacotherapeutic treatment of addiction, according to Wikler's (1965; 1976) principles of conditioning extinction. In rendering drug use pleasureless, naltrexone was thought to provide the key to eliminating any direct or conditioned neurochemical incentives for relapsing to drug use. Changes in scientific ideas about naltrexone, as

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[8] Although the idea was that a 'pure antagonist' worked by occupying the receptor, this explanation was of course speculative – since the receptor was still, at this point, hypothetical. Nevertheless, as we have already seen, theoretical principles supported by a variety of experimental evidence had, by the 1960s, allowed scientists to make the presumption that a substance that could block the effects of a narcotic was assumed to do so at specific molecular sites in the brain.

well as its role in the treatment of addiction and drug use, are investigated in depth in following chapters.

CONCLUSION

This chapter has focused on how, from approximately the mid-1960s to the mid-1970s, researchers began to situate addiction within a newly developing neurochemical problem space. This was a critical period for contemporary understandings of addiction – a period in which new ways of representing addiction as a disease of the brain, and intervening on diseased brains, were coming into being. The demonstration of the opiate receptors greatly intensified optimism that mental illness could ultimately be known and modelled at the biomolecular level, and the testing of the opiate antagonist therapy naltrexone was heralded as the beginning of an era of pharmacotherapies that held the promise of curing the relapsing condition of addiction, rather than simply maintaining addiction in less harmful forms (as methadone had been criticized for doing).

The addiction science of this period also helped produce the new understandings of addiction that, thirty years later, have come to dominate biomedicine. Today, it is understood by those whose opinions arguably count most – at least in terms of funding research and implementing policy – that addiction is produced by alterations in neuroanatomy and neurochemistry which disrupt the functioning of selected groups of neurons in the brain (Institute of Medicine 1996). It is not that non-brain considerations have been cast aside altogether; but that these have become rethinkable in terms of what goes on in the molecular biology of the brain and central nervous system. As a report by the (American) Institute of Medicine's Division of Neuroscience and Behavioral Health puts it: "A sophisticated understanding of how

the brain works recognizes that an individual's life experience and social context exert powerful effects on the brain and, therefore, behavior" (Institute of Medicine 1997: 39). Psychological and sociological factors ('life experience and social context') which contribute to what individuals think and do ('behavior') become considerations of what at its core "involves a biological process" (Nestler and Aghajanian 1997: 58). What goes on in the realm of the psychosocial affects what goes on in the brain, and what goes on in the brain determines individual experience.

Ultimately, this chapter has investigated the formation of a new, neurochemical problem space for addiction; and how the brain – and the scientist's laboratory – have become obligatory points of passage for those who wish to produce truths about addiction or deploy scientifically rational strategies to manage drug use and abuse. But it has not suggested, as many scientists do, that the theoretical and technical advances described were "based primarily on scientific logic" (Akil et al. 1997: 70). The chapter in fact challenges the assumption that our neuroscience ideas about addiction – as well as our related legal and therapeutic controls – flow from an unchanging nature of the brain. Certainly, as the laboratory sciences have extended their lines of penetration into the molecular makeup of the central nervous system over the last few decades, we have obtained facts about what the brain is (i.e., its structure), how the brain works (its processes), and what the brain does (its functions). Yet, this chapter has demonstrated that the conditions required for producing such facts are historically contingent, and dependent upon a range of social, political, and economic factors that play an important role in determining what becomes a problem, what is imagined as a possible explanation, and what possible explanations are actually investigated and brought into the realm of truth.

Both in the case of the development of naltrexone and the demonstration of the opiate receptor, scientific achievements were only made possible with the concurrent development of a new style of thinking about addiction on the molecular level. The 'logic' or style of reasoning involved emerged as a community of researchers and scholars from fields such as pharmacology, chemistry, biology, neuroscience, and psychiatric medicine worked to produce a series of innovations, both tangible and cognitive, which made it possible for addiction to be established within a new realm of neurochemical truth. Although it may not appear striking or remarkable today, the facts – and the very objects – of this realm, which are now virtually undeniable, did not exist a few decades ago. They only came to take shape with the introduction of novel truth-producing methods, techniques, technologies, and formulations, which opened new avenues of research, allowed new types of questions to be asked, and created new possibilities of existence.

Even according to researchers themselves, the development of these thought styles is understood to be inextricably related to social problems, political strategies, and economic factors. The facts of addiction neuroscience, and the means for producing those facts, have been made possible only through the allocation of resources that had not been available to this thought community until the mid-1960s, when the US federal government began to reorient its drug policies towards more biological – and biopolitical – approaches. Alliances with government regulators and funders provided material and regulatory support, without which the hypotheses and hunches of addiction researchers may never have even become possible candidates for truth.

This chapter suggests, thus, that to a considerable extent, the molecular truths of the brain flows from the social, political, and material matrix in which the neuroscience of addiction is situated. A significant amount of basic addiction science has been funded in order to determine exactly what the brain is made of, what it does, and how it works; and this science has developed new ways of studying, explaining, and acting on the brain that bring about new factual objects and processes – things like receptors, neurotransmitters, and homeostatic adaptations – that had no a priori existence (at least not as objective facts). And needless to say, these facts did not bring an end to questions about addiction and did not themselves remain unchanged. The next two chapters consider some of the ways in which neurochemical models and psychopharmacological treatments have further reconfigured the problem space of addiction, and have, by shaping the powers and acts of perception of researchers, turned the minds of addiction scientists towards questions about craving and desire.

CHAPTER 5: PATHOLOGIES OF DESIRE AND THE SCIENCE OF CRAVING

INTRODUCTION

> Drug abuse affects the way the brain experiences pleasure. Drugs make people "high" by invading and manipulating the brain's pleasure circuitry. They fool your brain into good feelings that are a reaction to chemicals, instead of to real experiences.
>
> The key word is "fool." Drug abuse can damage the brain's wiring for pleasure, making it unable to function in a healthy, normal way. You can become addicted, meaning that your craving for the feeling you get from a drug will become so strong that you'll risk serious consequences to get it. And your ability to feel pleasure the old-fashioned way—the real way—may be disrupted. Good food, real accomplishments—even true love—may leave you feeling flat.
>
> From Scholastic Inc.'s "Heaps Up" programme (Kukula n.d.)

As the above quote from an educational program sponsored by the National Institute on Drug Abuse indicates, addiction is today understood in terms of a pathological state of brain functioning that arises when the brain's pleasure system becomes affected by drug use. All addictive drugs are thought to produce rewarding effects due to the fact that they directly or indirectly affect the same pleasure circuitry in the brain (the dopamine and endorphin systems) which rewards 'natural' behaviours that make us feel good, such as having sex or eating. Because their effects rely on the brain's own 'feel good' chemicals, the brain cannot differentiate between *kinds* of pleasure, but can only distinguish differences in the intensity of pleasure. Because taking drugs can produce significantly more activity in the reward system than non-drug activities, over time, the rewards that result from 'real experiences' become relatively unrewarding, and the addicted individual is left craving only the intense pleasures that a drug of abuse can offer.

Today neuroscientists explain that drug cravings – urges so powerful that they propel individuals back toward drug use – are (as the director of NIDA testified to the US Congress in 1997:) "probably the single most important factor which can lead to relapse" (Leshner 1997). While craving is defined by the Oxford English Dictionary in rather unscientific sounding terms – as an 'urgent desire,' a 'longing' or 'yearning' – over the last thirty years or so, scientists have come to study craving as a neurobiological process, the understanding of which "provides the foundation for developing effective treatments to prevent or reverse the addiction process" (Leshner 1997). In other words, scientists have developed ways of thinking about and treating drug cravings as matters of neurobiological fact.

The scientific understanding is that craving results from the brain's pathological responses to drug-related stimuli, which are brought about by conditioned learning and positive reinforcement. Over time, as the brain comes to associate certain behaviours, thoughts, sights, and sounds with the pleasure induced by drug-taking, it learns to encourage drug-taking by rewarding these stimuli. For example, in a process that is described as essentially similar to the conditioned salivating response of Pavlov's dogs to a feeding bell, when an intravenous heroin addict sees a syringe, the brain responds by increasing the activity of dopamine. It is this neurochemical surge, experienced subjectively as craving, that is understood to motivate an individual towards behaviours that increase the risks of relapsing.

As craving has come to be understood as a result of changes in parts of the brain that are involved in the control of emotions and motivation, a significant proportion of the research and treatments of addiction science have come to focus on managing

pathological drug cravings with medications that modulate the neurochemical processes within these areas. Such medications, referred to as anti-craving pharmacotherapies, are celebrated by some as 'magic-bullet' drugs that target brain structures so precisely that they produce no effects other than reducing the intense desires associated with risks of relapse. They are understood simply to 'down-regulate' the activity of the brain's reward systems, so that the brain does not react too much to drug-related stimuli, and the individual does not experience cravings that lead to drug-seeking behaviour.

This chapter suggests that from the 1970s onwards, as the inability of chronic drug users to remain abstinent came to be understood as resulting from abnormally intense cravings for drugs which are caused by malfunctions of the neurological structures and mechanisms that are involved in the control of emotions and motivation, addiction has come to be represented as what can be referred to as a *pathology of desire*. As the problem space of addiction came to centre around the brain, the fundamental problem of relapse ceased to be relapsing behaviour – that is, the actions involved in the return to drug-use. Instead, it became the mind/brain state of craving, which is understood to lead to relapsing behaviour – or at least to increase the risk of relapse. Thus, this chapter suggests that neuroscience-based addiction treatment has come to involve a sort of risk-management that can be understood within an anatomo-politics of the human brain: within contemporary biomedicine, the therapeutic problem has shifted from managing the relapse-related behaviour of the addicted individual, to regulating neurochemical processes. The addicted subject's neurophysiology is essentially, and more or less literally, treated as a *risky matter*.

Biomedical experts tend to assume, implicitly or explicitly, that contemporary logics of addiction science and therapy have come into existence simply as the result of a better, more accurate, understanding of what addiction actually is. This chapter, however, offers an account of the development of contemporary neuroscience of addiction that brings these assumptions into question. It suggests that the biological nature of addiction and craving are historically contingent, and that the neurobiological facts of addiction have changed as addiction science has developed. Moreover, it suggests that anti-craving interventions are best understood not only as a product of scientific knowledge about addiction, but also as having played an important role in producing that knowledge.

By examining historical scientific literature on the neurochemistry of reward and the pharmacology of naltrexone, this chapter traces how the brain disease of addiction came to be characterized by bioscientists as a matter of craving. While craving and pleasure-seeking had long been considered important problems by psychologists, it was not until the 1970s, in the wake of discoveries of the opiate receptors and the development of naltrexone, that craving and pleasure began to appear as neurobiological phenomena. This chapter describes how the elucidation of the neurochemical reward systems, and the unexpected finding that naltrexone could reduce cravings, helped make it possible to conceptualize craving – which for so long had been an entirely subjective phenomenon not considered by physiologists – as objectively related to a neurochemical event, something that could be scientifically defined, observed, and acted upon. The chapter further examines how new representations of addiction that focus on endogenous brain chemistry (rather than on exogenous drugs) have drawn formerly disparate types of drug addiction together

with a common underlying explanation, thereby making possible new rationales and objectives of medical treatment.

This chapter in some ways provides the basis for a transition in the thesis, from a genealogical orientation (which has examined the historical roots of contemporary understandings of addiction and the conditions under which these became possible) to a governmental one (which places emphasis on the relevance of addiction science and treatment to the management of social, political, and personal life in the present day). If Chapter 3 focused on how addiction medicine came to know addiction as a chronic, relapsing disease, and Chapter 4 investigated how addiction science began to develop evidence that addiction was essentially a matter of the brain, the present chapter examines how developments described in those chapters have, since the late 1970s, culminated in our current neuroscience models and treatments. By elucidating exactly what sort of brain disease addiction has come to be (the underlying molecular mechanisms of drug effects, the neurochemical processes involved in relapse, the fundamental biological problems that addiction presents), the chapter provides a basis for following analyses (in Chapters 6, 7, and 8) of how neuroscience models and treatments are coming to be deployed in strategies of managing and preventing individual and social problems associated with drug use and other forms of compulsive conduct.

THE BIOCHEMISTRY OF PLEASURE

We will recall from the preceding chapters that motivation and learning in addiction had already become the focus of scientific investigation during the 1950s as the work of Abraham Wikler inspired researchers to examine the role of Pavlovian conditioning in relapse. However, these early studies did not focus on the motivating

power of drug-induced pleasure – quite the contrary. Through most of the 1960s, these researchers agreed that relapse was due only to negative processes of conditioning: in their models of addiction, withdrawal-like symptoms (and the homeostatic mechanisms that produced them) became conditioned to drug-related stimuli, and it was these symptoms that led to relapse, since addicts learned that drug-taking relieved abstinence distress. Wikler explicitly rejected incorporating factors relating to euphoria and pleasure in scientific explanations of relapse, dismissing these as subjective, psychological phenomena.

However, positive reinforcement gradually came to be seen as an important factor (and indeed, as we shall see, the most important factor) in the scientific study of drug addiction, in the wake of announcements by James Olds and his associates of the discovery of the brain's 'pleasure centres' (Olds and Milner 1954). In finding that rats that could press a lever in order to administer electrical stimulation to certain regions of their brains would go on pressing those levers to the point of exhaustion, and even death, Olds' work suggested that pleasure had a physiological basis. This was a pioneering discovery in the neurobiology of motivation and reward: rat brains, and presumably our own, had particular areas that were responsible for making behaviour rewarding, and these centres motivate us to behave in ways that maximize our pleasure. And in the late 1950s, it began to inspire researchers to develop experimental methods of studying the positive reinforcement of drug-taking behaviour in laboratory animals.

Although Olds' original experiments were electrophysiological (rewards were produced by electrically stimulating parts of the brain) there soon emerged an

important group of researchers that found that the same sorts of positive behavioural motivation could be produced by administering narcotics and amphetamines to those pleasure centres. As the field of behavioural pharmacology developed, researchers created new technologies that helped implicate the brain's own chemicals in drug rewards. Studies demonstrated, for example, that the behavioural responses of laboratory animals to psychostimulant drugs could be reduced or eliminated by interfering with the activities of certain neurotransmitters. Through the 1960s, researchers began to propose that the euphoria produced from drugs resulted not exclusively from the drugs themselves, but was mediated – in ways that were still unclear – by the neurochemical action of the neurotransmitters (Jonsson, et al. 1971; Stein 1968; Way, et al. 1968).

These observations were of considerable importance for two reasons. First, they suggested that the rewards associated with drugs were not merely psychological issues, but in fact had a biological basis that could be studied in an objective manner. Second, they suggested that drug-induced euphoria might be neurochemical rather than pharmacological: drugs appeared not to produce pleasure directly, by coming into contact with the brain, but indirectly, by somehow interacting with neurobiological processes. As drug effects came to be understood as dependent on the chemistry of the brain, the task of researchers could no longer be to simply investigate the pharmacological properties of drugs themselves; the newly relevant molecular anatomy of the brain had to be explained as well. Interest in drug addiction thus came, to a significant extent, to focus on the chemistry of the brain, rather than the chemistry of addictive drugs.

Over the course of the 1970s, a range of laboratory tools and methods were developed which allowed researchers to move beyond behavioral experiments and crude dissection techniques to study *in vivo* the neurotransmitters involved in drug reward and motivation. For example, improvements in intracerebral microinjection techniques enabled researchers to introduce substances into specific areas of the brain with high levels of precision, and the introduction of microdialysis technology allowed researchers to measure the *in vivo* flow of neurotransmitters in brain tissue by inserting a semi-permeable, capillary-like probe into a living animal's brain. Through these and a wide range of other laboratory techniques and technologies, new information emerged that suggested that all of the dependence-inducing drugs produced their rewarding effects through one or more neurotransmitter systems. As already indicated, the most important of these have come to be identified as dopamine and the endorphins.

However, the neurotransmitters scientists were able to find in fluids extracted from brains were not substances that appeared in 'nature.' Their existence, as distinctive compounds, depended fundamentally on technologies which could purify and sort extracted neurohumours into an array of substances; and the development of such purification techniques in turn depended on what scientists thought it plausible to look for. For example, before the endorphins could be discovered, the perceptions of brain fluids had to change in such a way that it made it reasonable to look for endogenous opiate-like substances. And it was not reasonable to do so until research efforts (sponsored through agencies fighting the War on Drugs) provided evidence of the opiate receptors.

As described in the preceding chapter, Pert and Snyder's research – devising experimental innovations that could demonstrate the existence of structures which for a number of years had already been presumed to exist – was approached as a technical problem, rather than a theoretical one.[9] However, once the existence of opiate receptors came to be taken as an indisputable fact, researchers realized that they were facing a more fundamental problem, namely, explaining why the body has highly-specific receptors for chemicals not found in the body, and not easily obtained in nature. Researchers reasoned that the 'opiate' receptors in the brain must have developed not for opiates, but for an endogenous ligand – that is, a morphine-like chemical produced by the brain itself which was capable of binding with the receptors. If this was the case, then the ability of synthetic opiate drugs to activate preceisely the pleasure system of the brain would be easily explicable in terms of neurochemical mimicry: opiate molecules could be understood to bind with receptors that were normally activated by the brain's own, endogenous chemicals.

Thus, it was not until the opiate receptors had been demonstrated that a search for the neurotransmitters that acted upon those receptors seemed like a reasonable and worthwhile endeavour. And indeed, after the discovery of the receptors, an endogenous, opiate-like substance seemed so apparent that scientists rushed to beat others to its discovery. A hunt for morphine-like neurochemicals that the body naturally produces on its own soon began (Hughes 1976; Simon 1980), and within about a year of Pert and Snyder's (1973) report of the demonstration of opiate receptors, Hughes, Kosterlitz and their associates devised a series of chromatographic

[9] Leading up to and during their research, Pert and Snyder had, like most scientists involved in basic research in this field, not given much thought to the conceptual implications of a receptor for opiates **Snyder, S. H.** 1989 *Brainstorming : the science and politics of opiate research*, Cambridge, MA: Harvard University Press..

procedures, which allowed them to locate endogenously-produced brain chemicals that acted upon the same receptors as opiate-related drugs (Hughes 1975; Hughes, et al. 1975a). But it is far from certain that these neurotransmitters would have emerged as distinct substances if the receptors had not provided scientists with the motivation to devise complex technological means of producing them in the laboratory. The fluids extracted from brains did not appear to contain a distinct opiate-like chemical until scientists were prompted to develop tools capable of extracting data from brain fluids that could indicate the presence of such a substance. Such substances could not take on an existence independent of the technology that made them appear, in laboratories, as distinct compounds.

Shortly after the first 'enkephalins,' or morphine produced 'in the head' (Hughes, et al. 1975b), were identified, a number of different endogenously produced, opiate-like chemicals were discovered, and the generic term 'endorphin' (a contraction of 'endogenously-produced morphine') came into wide usage to describe this group of neurotransmitters. By the end of the 1970s, discussions of endorphins and the 'endogenous opioid systems' had become ubiquitous in neurological discourses on addiction and dependence. There was lingering uncertainty as to what specific roles these played in addiction;[10] but it was excitement, promise, and expectations about the enkephalins / endorphins, as much as evidence about them, that helped create new ideas about what needed to be explained in addiction science. For example, when it became possible to isolate endorphins from brain fluids, the purified substance could

[10] Many of the earliest assumptions about endorphins had to be revised or qualified. For example, efforts to link endorphin levels directly to clinical observations of addiction were largely unsuccessful, and it was discovered that the highest concentrations of enkephalins – the 'in the head' endorphins – were actually to be found in the adrenal gland **Snyder, S. H. and Pasternak, G. W.** 2003 'Historical review: opioid receptors', *Trends in Pharmacological Sciences* 24(4): 198-205.. Indeed, it had not even been proven, beyond doubt, that these morphine-like neurochemicals *did* act upon the opiate receptors.

create, in laboratory rats, the same responses as those produced by morphine – jumping and squealing, and a higher tolerance of painful stimuli. The ability of these naturally produced (but artificially concentrated) substances to elicit such dramatic, drug-like effects was taken as evidence that endogenous chemicals supplied by the brain could themselves have a fundamental role to play in explaining drug effects on the processes of motivation, reward, and conditioned learning. In 1980, one of the leading authorities on neurochemical models of addiction noted that:

> The discovery of opiate receptors and their *supposed* endogenous ligands, the endorphins, has kindled the excitement and imagination of many scientists and, through ample coverage in the news media, of the general public as well. Hopes have been raised that these findings may contribute to the solution of a number of human pathologies ranging from intractable pain to mental disease (Simon 1980: 307, emphasis added).

At this early stage, it seems that verifiable truths of endorphins were not necessarily the most important factor in the intensification of the focus (by drug abuse researchers, as well as policy makers, media professionals, and lay individuals) on the neuroanatomical and neurochemical interiors of human beings.

Modulating the endogenous reward system

The discovery of the endorphins also raised new questions about naltrexone, and specifically about its relationship to the neurochemical reward system. Since naltrexone was thought to interfere with the actions of heroin by occupying the opiate receptors, the question predictably arose as to whether it might also block the activation of the opiate receptors by the endorphins. Findings that naltrexone did indeed have such effects led to a significant reorganization of the facts of naltrexone's pharmacological actions.

In the early 1970s, virtually all scientists and clinicians had agreed that as a 'pure' narcotic antagonist, naltrexone had no effects of its own beyond the blocking of narcotics. Indeed, it was considered an undisputed fact that naltrexone's *only* action was the blocking of opiate drugs. As Avram Goldstein (the pharmacologist who developed the radiolabelling technique used by Pert and Snyder to demonstrate the existence of the opiate receptor) noted:

> The pharmacologic basis of naltrexone therapy is not in question. This antagonist, given in adequate oral dosage (e.g. 120 mg) three times weekly, maintains a sufficient degree of blockade of the opiate receptors to prevent the psychotropic effects of opiate agonists like heroin. Naltrexone itself appears to be without pharmacologic action other than to block opiate effects (Goldstein 1976: 158).

Naltrexone's action was assumed to be a blockade of the opiate receptor, and nothing else. The therapeutic effect of narcotic antagonists such as naltrexone were thus not thought to affect the neurochemical systems involved in an individual's reward system directly, and it was concluded that "[a]ll narcotic antagonists suffer the drawback that they do not relieve the 'hunger' for narcotics but merely interfere with gratification" (Ng, et al. 1975: 320). No other effects could be imagined. Naltrexone's therapeutic use was therefore limited to "protecting the patient from the effects of selfadministered [sic] opiates" (Resnick and Schuyten-Resnick 1976: 85). By blocking the effects of opiates, naltrexone allowed the brain to 'heal' itself, or at least recover from pathological states of conditioning; but that was all.

However, once the endorphins were found, certainty of naltrexone's actions diminished significantly. Goldstein concluded the paper (referred to in the preceding paragraph) in which he stated that 'naltrexone itself appears to be without

pharmacologic action other than to block opiate effects' by bringing into question that very assertion:

> I should like to mention the newly discovered endogenous opioid peptides (endorphins) in pituitary [(Cox, et al. 1975; Teschemacher, et al. 1975)] and brain [(Hughes, et al. 1975a)]. Could their existence have some bearing upon the use of antagonists? We know now that the opiate receptors, which are blocked by naltrexone and other antagonists, are really endorphin receptors. It must be assumed that they play some physiologic role in the nervous or endocrine systems. How can we block these receptors without interfering with their normal function? The answer to this question is not yet clear (Goldstein 1976: 160).

Once the endorphin system was assigned a role in motivation and reward, it became possible to ask whether an opiate receptor antagonist that blocked narcotics might also block narcotic-like neurotransmitters, and whether these effects on the reward system had implications for addiction treatment.

The emergence of these new possibilities led Wikler to revaluate reports he had previously discounted from patients who suggested that naltrexone reduced their 'hunger' for opiates. Although these subjective self-reports had once constituted anecdotal, and hence almost insignificant, evidence, they suddenly seemed capable of indicating naltrexone's pharmacological effects on the endorphin system. Based on these reports, Wikler came to believe that "[t]he question of whether or not the opioid antagonists exert a hitherto unknown agonistic, 'satiating' effect on opioid-deprived receptors is very important from both the theoretical and practical standpoints" (1976: 120). Thus 'subjective' information provided by subjects' accounts were used to strengthen an association between craving and 'objective' processes going on in the brain, and to speculate on a biological basis of drug-related desires.

Thus, by the mid 1970s, anecdotal observations of and reflections on craving, previously considered to be of only marginal importance to neuroscientists, began to move toward the centre of research priorities. It came to be hypothesized that "pure" antagonists such as naltrexone "have no known effect on normal organisms, but they may have important effects in organisms whose endogenous morphine-like substance (endorphin) system has been disequilibriated by chronic administration of opioid drugs" (Renault 1976: 2). Ensuing research provided evidence that patients receiving naltrexone reported significantly less craving than those treated with placebos – particularly if naltrexone was administered at dosages higher than the minimal levels required to block narcotics.

The indications of naltrexone's anti-craving properties had significant implications for its use in addiction treatment. Final clinical trials of naltrexone were underway in the late 1970s, and, in the early 1980s, naltrexone was approved by the US Food and Drug Administration for general clinical use in the treatment of opiate addictions as an antagonist therapy – i.e., a pharmacological means of blocking the effects of opiate drugs. These trials had been based on the assumption that naltrexone could allow conditioned responses to drug use to be extinguished, as individuals learned that drug-taking would not result in rewarding effects. The hope was that over time, since naltrexone rendered drug-taking activities unpleasurable, individuals' psychological and physiological responses to stimuli could be eliminated.

But as Wikler noted, the possibility that naltrexone had anti-craving effects would render the pharmacotherapeutic approaches to treating addiction that were currently being investigated "both impractical and useless" (1976: 120). If naltrexone reduced

the desire for drugs, drug-seeking behaviour would cease without anything having being (un)learned. An anti-craving therapy would not influence behaviour by changing patterns of learned response, but rather through a more direct control over neurochemical processes. Accordingly, Wikler suggested that an anti-craving therapy would involve prolonged maintenance on the medication, rather than a brief period in which naltrexone helps the subject correct pathological responses to drug-related cues (Wikler 1976: 120).

As a result of these developments, Pierre Renault noted that "even with the marketing of naltrexone, several important research issues will remain" (1980: 20). One of the most important areas he identified was the linking of the work of pharmacological researchers with that of basic neuroscientists, who, at this time, were beginning to explain addiction more in terms of the functioning of endogenous neurochemical systems than the relationship between exogenous drugs (i.e., 'drugs of addiction') and their effects on receptors. "An exciting area of research", Renault predicted,

> will be on the effects of naltrexone on the endorphin system. The fact that in the Phase II double-blind study, naltrexone appeared to decrease craving suggests that it may have a "healing" effect on the endorphin system. It may well be that endorphins serve an important function in the regulation of mood and sense of well-being. It also seems possible that prolonged use of heroin may atrophy this system and the subsequent disruption in mood and sense of well-being may be the basis of prolonged craving in addition to the known pharmacologic phenomenon of prolonged abstinence. It would be of the utmost importance if naltrexone proved to be not only a protection against impulsive heroin use, but also directly beneficial by helping to reverse heroin-induced atrophy in the endorphin system (Renault 1980: 20).

Certainly, such statements were highly speculative. But that is precisely the point: new possibilities in addiction research and treatment were brought into view by the interrelated basic and clinical research of this era. And indeed, even as naltrexone

was approved for addiction treatment as a narcotic antagonist, the interest of researchers in naltrexone began to shift from its targeted action on the receptors to its modulation of the brain's endorphin system.

As Renault and Wikler both suggested, understandings of naltrexone's actions did, through the 1980s and 1990s, come to include an effect on the endogenous reward system. And craving, so long considered an entirely subjective phenomenon – one not amenable to scientific investigation or targeted biological intervention – appeared to be knowable and treatable in ways that had hitherto been thought impossible. While the strategies and logics of anti-craving treatment are explored in depth in Chapters 6 and 7, the remainder of this chapter focuses on how, in the wake of their appearance in studies on opiate drugs as something that could be studied objectively, craving and relapse began to be explained in terms of pathological neurochemical processes that were involved not only in addiction to narcotics, but also in addition to the entire range of abused drugs.

THE DOPAMINE HYPOTHESIS OF ADDICTION

As interest in the neurochemistry of drug addiction grew in the wake of the discovery of endorphins, researchers began to focus on mapping out the neurotransmitter pathways affected by drug use with increasing specificity. Through the 1990s, as an increasing number of drugs were studied, the range of mechanisms and regions affected were found to vary widely between different classes of substances. However, it became possible for researchers to identify some commonalities among drug effects, and it was these similarities between the various drug classes, rather than the differences, that scientists focused on. Most significantly, researchers found that all drugs thought to be addictive – not just the opiates – influenced the processes of

neurotransmission within the brain's reward systems involved with motivation, learning, memory, and the sensation of pleasure.

It came to be understood that drugs interfered with the biological system of rewards, which is presumed to have evolved to encourage activities that increase an organism's survival chances, such as eating and having sex. This system is understood to motivate these (and other) behaviours by releasing euphoria-producing neurotransmitters when the behaviours are engaged in. As the individual or organism learns to associate particular activities with pleasurable sensations, it becomes motivated to engage in those activities again and again. This process is facilitated by conditioned learning, which "enables the organism not just to experience the reward [...] but to learn about it, so that they can get back to what they perceive to be the good things again or avoid the bad things" (Childress, quoted in Penn Medicine 2001). The association between an action and a reward constitutes a basic form of memory that enables the organism to remember how to achieve rewards.

Although it was confirmed early on that the endorphins played a significant role in the rewarding effects of opiate drugs, the most significant brain chemical in the reward system came to be identified as dopamine. Through the 1980s and 1990s, despite their dramatically different chemical structures, hallucinogens, narcotics, amphetamines, opiates and other drugs all began to appear to be essentially similar in their ability to motivate individuals to use drugs by influencing the activity of the dopamine system:

Psychostimulants. The rewarding effects of psychostimulants such as cocaine and amphetamine have been, since the 1970s, linked to the brain's dopamine levels (Stein and Wise 1973). The dopamine system was more closely implicated in addiction by a series of experiments demonstrating that the self-administration of psychostimulants decreases significantly when dopamine neurons in the nucleus accumbens (NA) were destroyed (Goeders and Smith 1983; Roberts, et al. 1980), and that the intravenous administration of cocaine results in elevated levels of the neurotransmitter (Pettit and Justice 1989). Psychostimulants have not been found to act directly on dopamine receptors – that is, they did not appear to mimic dopamine – but instead are understood to interfere with the 'reuptake' of dopamine released by presynaptic neurons. Because dopamine, once released, is not reabsorbed, the neurotransmitter collects around the postsynaptic receptor sites and results in the hyperactivity of dopamine neurons (Cohen 1988; Kuhar, et al. 1991).

Opioids. Although initially it was hypothesized that the dependence-inducing effects of opioids resulted solely from their mimicking endorphin (and over-stimulating endorphin receptors), it was discovered that one of the three sub-types of endorphin receptors also reduces the activity of other neurons which normally regulate the release of dopamine (Morita and North 1981). Morphine was also discovered to be most rewarding when administered to the ventral tegmental area (VTA), a region of the brain associated with dopamine reward (Bozarth and Wise 1981; Phillips and Le Piane 1980). Thus, the reinforcing properties of opiates began to be attributed to the same dopaminergic system responsible for the action of the psychostimulants (as well as additional, dopamine-independent sites of action) (Johnson and North 1992; Koob and Bloom 1988).

Ethanol. Although the precise neurological actions of alcohol on the reward system have proven difficult to distinguish, as ethanol produces wide-ranging, non-specific cellular changes throughout the body (Littleton 1978), ethanol has been found to increase the firing of VTA dopamine neurons by suppressing other, dopamine-suppressing neurons (Gessa, et al. 1985; Mereu and Gessa 1985), thereby elevating extracellular DA concentrations in the NA (Di Chiara and Imperato 1988).

Nicotinics. In the early 1980s, researchers established that administration of nicotinic drugs elevates brain levels of dopamine (Sakurai, et al. 1982) and that the destruction of dopamine receptors reduces self-administration behaviour of nicotine (Singer, et al. 1982). In addition, nicotinic receptors have been discovered on dopaminergic neurons (Clarke and Pert 1985), and were observed to elevate dopamine levels in the nucleus accumbens (Imperato, et al. 1986) by directly stimulating dopaminergic cell firing (Mereu, et al. 1987).

Cannabinoids. The identification of the sites and mechanisms involved in cannabis dependence had been one of the slowest-progressing areas of research almost until the mid-1990s (Wise 1996). The cannabinoids had been considered anomalies among drugs of abuse, apparently lacking pharmacological interaction with brain reward substrates – which in part explains why the very notion of cannabis dependency has been so hotly debated. By the early 1990s, however, researchers had begun to find indications that tetrahydrocannabinol (THC), marijuana's principal psychoactive component, acts on the brain "in strikingly similar fashion

to noncannabinoid drugs of abuse" – both by acting on dopamine activity and also by affecting certain opioid receptors (Gardner and Lowinson 1991). Through these discoveries, it became possible to compare cannabis with heroin, because the two drugs "exert similar effects on mesolimbic dopamine transmission through a common [m1] opioid receptor mechanism" (Tanda, et al. 1997). Researchers have also discovered cannabinoid receptors and endogenous cannabis ligands (Devane and Axelrod 1994; Vogel, et al. 1993).

It was in the early 1990s, when the activation of the dopamine system in the limbic region of the brain was identified as the 'common neural substrate' of drug reinforcement (Wise 1990; Wise and Bozarth 1987), that the 'dopamine hypothesis' of addiction emerged (Di Chiara 1998; Kuhar, et al. 1991; Melis, et al. 2005; Miller and Gold 1993). In a 1999 review article in *Trends in Neuroscience*, it was noted that dopamine-centred, neurochemical understanding of addiction had guided the field of addiction science research for over a decade (Spanagel and Weiss 1999). And indeed, since the emergence of the dopamine hypothesis, it has come to be suggested that prominent differences between drug effects beyond the activation of the reward system could be relegated to the status of 'side effects' (Bozarth 1990).

While dopamine (in addition to other neurotransmitters) provided the basis for establishing a unified biological model of addiction based on the neurochemistry of rewards, this common denominator in motivating drug use was also identified, of course, as a primary source of motivation in general – that is, for behaviours unrelated to drug use as well. Since dopamine has been identified as a common reward currency for all motivated behaviours,

> [t]he direct chemical activation of these reward pathways does not in itself represent any severe departure from the normal control reward systems exert over behavior. Inhalation of a substance (e.g., nicotine) is no less natural than the ingestion of sugar, although the former has no direct survival value to the organism nor to the species. But both involve activation of brain reward mechanisms and both may be subjectively experienced as pleasurable in humans. [...] Simple activation of brain reward systems does not constitute addiction! (Bozarth 1994).

Thus, addictive drugs could not be distinguished by a distinct kind of pleasure that was particularly dangerous or artificial. And so the question as to how, if at all, drug rewards differed from other rewards emerged as an important problem. What was special – or pathological – about pleasures derived from drugs that made them different from pleasures derived from eating, having sex, or exercising?

Since the mid-1990s, the answer to this question has come be based upon the assumption that while the pleasures produced by drugs and by normal activities do not differ in kind, they do differ in degree. Addictive drugs, it is proposed, can initiate far more dopamine activity – and thus far more rewarding experiences – than can everyday behaviours. And it is the 'supraphysiological' release of dopamine that is understood to play an important role in causing drug addiction (Volkow 2005). The idea is that under normal conditions, a balanced dopamine system motivates behaviours in a balanced way (not too much pleasure for any one particular activity, or one would not want to do anything else besides that). But when the dopamine system is invaded and manipulated by drugs of abuse, the resulting pleasure is far more intense than naturally-occurring pleasures – and all of a sudden the rewards we get from our brains for "normal" behaviours pale in comparison to those we get for behaviours associated with taking drugs.

When drug-taking is disproportionately rewarded by the dopamine system, it comes to increase in salience until it is valued above other behaviours. Over time, as the brain becomes accustomed to the artificially high amount of dopamine released by drug-taking, it establishes a new 'set point' or threshold for motivational rewards. This process culminates in a pathological state of brain functioning referred to by some as 'reward dysregulation' (Koob and Moal 1997). In such a condition, behaviours such as eating or exercising are not able to generate enough reward, and are unable to satisfy the (newly elevated) requirements of the system. And so without the presence of the powerful rewards elicited by drug use, the reward equilibrium of the emotional system becomes disrupted.

Thus, drugs of abuse are understood to 'hijack' the reward system by producing pleasurable rewards much more intense than those that occur naturally. But the most serious effect of drugs is not simply their ability to make drug-taking more pleasurable than other activities; it is instead their capacity to make drug-taking more *desirable* than other things, and to produce intense urges – referred to as cravings – when drug-related stimuli create incentives to engage in drug-seeking behaviour. As the current director of NIDA notes:

> the association of the drug-induced pleasurable experience with the increases in dopamine will result in strong conditioning, not only to the drug but to the stimuli that predict the drug (e.g., the house of the drug dealer, syringes). Through conditioning, initially neutral stimuli become highly salient and produce neural responses (e.g., dopamine increases) that trigger the motivation to procure the drug (Volkow 2005: 1402).

As drug-related stimuli are consistently and powerfully rewarded over time, feelings, behaviours, sights, and sounds associated with drug use activate dopamine pathways. The activation of this "neurochemistry of desire" (Penn Medicine 2001) reminds the

individual of previous euphoric states achieved in connection with such stimuli, and gives rise to the cravings that encourage drug-seeking behaviour and increase the risk of relapse.

Thus, a fairly fine-tuned distinction between pleasure and desire emerges in contemporary definitions of the fundamental problem in addiction. There is nothing wrong with (and there are no judgments to be made about) liking the effects of drugs at the very moment that pleasure is experienced. Indeed, given that the pleasure experienced results from the brain's own chemicals, liking drug effects – which are in fact the effects of dopamine – is natural. The problem arises only when 'liking' is supplanted by 'wanting' (cf. Robinson and Berridge 1993). Whereas liking is a direct response to the pleasurable effects of a real, immediately present substance, wanting is a conditioned response to reminders of a former state and an absent substance. Through such cue-elicited memories, experienced subjectively as craving, "neutral stimuli can gain control of behaviour" (Ammassari-Teule 2001) by providing incentives powerful enough to override competing motivations.

Such understandings suggest, as it is indeed coming to be accepted, that addiction represents a state of "pathological learning" brought about when drugs and drug-associated stimuli exert undue power over an individual's motivations (Hyman 2005). Craving has come to be described as a sort of disordered, long-term memory acquired through conditioned learning (Institute of Medicine 1996: 84). Because "the brain remembers too much or too powerfully records pathological associations" (Hyman 2005: 1414) the incentives it provides for drug-seeking behaviours outstrip those

provided for other activities, and this "motivational toxicity" results in a "deterioration in the ability of normal rewards to govern behaviour" (Bozarth 1994).

The question of why one sort of quest for dopamine is more normal than another is left implicit in neuroscience accounts. Scientists may note that patterns of behaviour associated with addictive drugs can often be distinguished from other behaviours by their "power to supplant almost all other goals", but they do not very frequently indicate why other goals should *not* be supplanted if they are not as rewarding. They may point out that drug-seeking incentives can motivate "parents to neglect children, previously law-abiding individuals to commit crimes, and individuals with painful alcohol- or tobacco-related illnesses to keep drinking and smoking" (Hyman 2005: 1415), but they do not specify how motivations to disregard one's family, the law, or one's health that come from drugs differ from or are similar to disregard of family, law, or health that are motivated by other sources of potential reward (such as money, power, love, or status).

Of course, this should not be too surprising, since neuroscientists and psychopharmacologists do not generally concern themselves with the problematic behaviours associated with addiction – even the actual actions involved in the return to drug-use. Their concern is instead with managing the underlying problem – the mind/brain state of craving, which is understood to lead to relapsing behaviour – in order to enable the reinstatement of normal motivational processes to reassert their control over an individual's conduct. The problem has become: how do we deal with cravings that increase an individual's risks of relapse (and of self- and social harm) in order to let her brain govern 'naturally'? And the answer is increasingly, as has

already been indicated, with anti-craving pharmacotherapies – the most researched and widely used of which is naltrexone.

THE ERA OF ANTI-CRAVING PHARMACOLOGY

As the dopamine hypothesis of addiction began to emerge in the early 1990s, and as it began to be agreed upon by scientists that drugs are compulsively taken because they act on parts of the brain that produce craving, a new focus emerged on intervening into the general neurochemical pathways and molecular mechanisms that had come to be implicated in pathological motivations. The once black-boxed body-at-risk of the relapsing addict had become sufficiently opened up to allow the perception of a new era of medical treatment:

> The identification of neurobiological substrates in addiction has provided new targets for treatment. Drug-induced increases in dopamine in the nucleus accumbens are considered to underlie the reinforcing responses to drugs of abuse, and repeated drug administration is believed to result in a series of adaptations involved in the loss of control and the compulsive drug administration that characterize addiction (Volkow 2005: 1401).

Today, it is understood that the specification of neurobiological reward pathways common to all drug addictions allows researchers to identify and develop medications that are useful for treating the underlying, general mechanisms of addiction.

But it is not simply the case, as Volkow suggests, that the 'identification of neurobiological substrates in addiction has provided new targets for treatment.' At the same time as this has occurred, new treatments have themselves provided new ways to identify the neurochemical mechanisms that came to be explained as the cause of craving and relapse. The efficacy of naltrexone, today considered the prototypical anti-craving medication, is explained in terms of its ability to target the brain mechanisms associated with craving precisely; but it is not the case that

naltrexone was rationally engineered in order to target these mechanisms, in a manner predicated or dictated by neurological models of addiction. Before naltrexone was introduced into experimental and clinical research, an interaction between antagonist pharmacotherapy and craving had not been imagined; indeed, during the 1970s, craving was not considered as something arising from an objectively knowable, biological process. Naltrexone helped make it possible to conceptualize craving – up to this point, an entirely 'psychological' phenomenon – as a neurochemical event, and thus as something that could be rationally observed and acted upon.

Moreover, naltrexone was the medication that helped make it possible to imagine pharmacologically-specific 'cross-over' treatments for addiction: that is, medications that, since they acted on the same fundamental mechanisms of reward, could be used to treat addictions to dissimilar categories of drugs. As early as 1980, there emerged reports of evidence that naltrexone could exert a significant negative effect on alcohol preference in laboratory animals (Altshuler, et al. 1980), and by the early 1990s, experiments with humans were indicating that naltrexone reduced subjective reports of alcohol craving and could help prevent relapse among abstinent alcoholics (Volpicelli, et al. 1992). As it appeared capable of controlling desires for alcohol, naltrexone – hitherto, a medication for opiate addiction – became thinkable as a potential general treatment for craving that could be applied to a range of compulsive desires. Since virtually all addictions were understood to be reinforced by neurochemical reward activity, and since naltrexone appeared to reduce the activity resulting from opiate- and alcohol-related cues, it was suggested that perhaps naltrexone could reduce craving for other, or all, rewarding substances.

It became clear through the 1980s and 1990s, however, that naltrexone could not so easily or effectively reduce craving for cocaine (Carroll, et al. 1986). This was a puzzling finding, since researchers had found that alcohol administration enhanced dopamine transmission in ways that were similar to cocaine (Fadda, et al. 1984). This apparent contradiction (naltrexone's ability to reduce cravings for opiates and alcohol, but not cocaine) was later resolved when it was found that ethanol molecules activated, in some way, the opiate system. It thus came to be accepted that while naltrexone does reduce craving by decreasing the activity of the dopamine system, it does so indirectly, through its effect on the endorphin system.

While naltrexone has not proven to be a cure-all for *all* pathologies of desire, it has come to be regarded as a model anti-craving medication (O'Brien 2005), and has inspired a hope that more effective pharmacotherapies can be developed which will be capable of managing the pathological desires and risky cravings that increase an individual's likeliness to relapse and compromise the brain's ability to govern behaviour in a normal way. Through the 1990s and up to the present, research into and development of neurologically-targeted anti-craving medications have become a major focus of addiction medication development and pharmacological treatment (O'Brien 2005). As following chapters elaborate, the goal of these medications is not to enhance individual will-power in the face of the dangers of freedom (for example, the availability, at relatively low cost, of a wide range of drugs, or the decreasing influence of moral and religious constraints on behaviour). Instead, it seems to be to protect free choice and normal motivation from undue influences – urges, impulses, cravings, and so on – that are symptoms of a neurochemical pathology of desire.

CONCLUSION

This chapter has attempted to build upon the insights of the preceding chapters to elucidate further the contingency of our contemporary understandings of addiction – and the inseparability of what addiction appears to be, from how we come to know about it. As in previous chapters, it focused on understanding the looping interactions between scientific 'findings' and 'lookings:' that is, researchers investigating the neurobiological basis of addiction (looking for things in certain ways, with certain methods) frequently encounter experimental artefacts (evidence or findings) that lead to a shift in the focus of their investigations. New findings create new ways of studying addiction as a disease of the brain. It has especially focused on the ways that research into and theories about addiction have frequently taken new directions as researchers have attempted to integrate unexpected findings into their models.

Thus, we saw that the opiate receptors and the narcotic antagonist naltrexone, both of which provided a foundation for addiction neuroscience (as the last chapter discussed), led to new investigations of the brain and of brain-targeting medications – which in turn led to new understandings of the nature of addiction. The opiate receptors led to the discovery of the endorphins, and consequently became known, in fact, as the endorphin receptors; and with these findings, the neurochemical effects of naltrexone came to shift from blocking narcotics to modulating the endorphin system. As the endorphin system was linked to the brain's reward system, it became possible to look for, and make sense of, findings that naltrexone could have a direct, modulating effect on craving.

And so it may not be quite as amazing as scientists often suggest, that we have magic bullet drugs that work so precisely on the parts of the brain specified by our models of

addiction – because those parts of the brain have in large part been specified by the actions of such drugs. Indeed, one might argue that to a considerable extent, the neurobiology of craving has been identified as 'those processes that are targeted by anti-craving pharmacotherapies.' So naltrexone is not simply useful *because* of the neuroscientific facts of addiction; it was also useful in *arriving* at those facts, insofar as it created new problems and new possibilities for thinking about and acting on addiction.

The suggestion is not that individuals did not experience craving before naltrexone was invented; but that the neurochemical mechanisms associated with craving only came to be scientifically studied after naltrexone was found to be effective in reducing desires for drugs, and that these developments contributed to a change in how craving came to be experienced and managed. These reconfigurations of the problem space and physiological nature of addiction are not only of historical interest, but also relate to important changes in strategies for dealing with a range of social, personal, and legal problems in relation to drug use and motivation. The following chapters of this thesis examine some of the transformations that the neuroscience understandings and pharmacological treatments of addiction examined so far have been producing on contemporary strategies for managing social problems and controlling problematic subjects. In particular, three themes that have emerged in the thesis so far are investigated in more concrete detail:

New classifications of disease and desire. Today, addiction is conceptualized as a disease that involves long-lasting, and possibly permanent, changes in the way that an individual's brain experiences pleasure, reward, and motivation. The biomedical

problem is no longer understood to be the body's dependence on drugs – e.g., the withdrawal syndrome – but instead arises from a chronic impairment of the brain's ability to regulate desire. In an important sense, the 'substance' of addiction is no longer drugs themselves, but the brain chemicals that make drugs rewarding. All addictions, and all addictive drugs, have become comparable in terms of their relation to endogenous rewards; and indeed, all motivations and desires – not only those involved with drug use, and not only those that are considered pathological – have become comparable. Chapter 6 investigates the implications of the dopamine hypothesis of addiction for so-called 'behavioural addictions' which, in contrast to drug addictions, do not involve substance use.

New logics of intervention and control. Biomedical addiction treatment has shifted from the brief use of anxiolytics and sedatives to calm the detoxifying individual, to ongoing interventions into (i.e., prolonged medication for) the dysregulated – and relapse-inducing – neurochemical reward systems of the brain. Since the control of dysregulated craving mechanisms is undertaken to prevent relapse, rather than to reinstate normality, it appears that the logic of treatment today consists of the management of risky desires, rather than the administration of cures. Chapter 7 investigates how naltrexone is conceptualized and put to use in the management of risky desires understood to relate to problematic forms of drinking.

New subjectivities and responsibilities. Contemporary addiction science has implications for how individuals identify themselves, and are identified as, certain kinds of people and patients. Addiction scientists today are emphatic about the fact that addiction is not a moral or character problem caused by degeneracy or lack of

will-power, but is instead a brain disorder to which all individuals are susceptible. Chapter 8 investigates how individuals are coming to be encouraged to think about and care for themselves in terms of their neurochemistry, especially as they make decisions about whether or not to use psychoactive substances.

CHAPTER 6: PROBLEMS OF CONDUCT AS DISEASES OF THE BRAIN: BEHAVIOURAL ADDICTIONS AND THE NEUROSCIENCE FACTS

INTRODUCTION

This thesis has been investigating newly emerging ways of exerting control over desires, cravings, and the thoughts and conduct associated with these. It has focused so far on addictions to 'alcohol and other drugs' – addictions in which the development of pathological compulsions is associated with a foreign chemical substance interfering with the balance and proper functioning of the brain's own neurochemical reward system. And yet, this thesis has been framed in terms of an investigation of the problematization of desire *itself* – not of drugs or substances per se. Thus, it makes a certain amount of sense to ask what changes are taking place in the governance of desire in relation to thought and conduct that is not inextricably linked to the use or abuse of substances. This chapter investigates how the anti-craving drug naltrexone, which is understood to regulate disordered reward mechanisms in the brain, is being deployed in the management of compulsive desire that is not related to drug use (and the resulting 'supraphysiological' pleasure), but is instead related to behaviours. More precisely, it investigates how some behavioural problems are coming to be (re)constituted as addictions within neurochemical styles of thinking; and how the biological facts and psychopharmacological technologies of contemporary addiction neuroscience are giving rise to new ways of understanding and fashioning the self as a subject of neurochemical desire.

There is nothing terribly new about using the term 'addiction' to describe compulsive forms of thought and conduct that do not involve the ingestion of psychoactive

substances. But the possibility of describing behavioural addictions in terms of biological and neurochemical facts *is* a novel and significant development, and relates in important ways to the blurring of the boundaries between natural and artificial forms of pleasure, reward, and craving that began to occur as drug effects came to be investigated in terms of their 'primary' neurochemical rewards. From the mid-1970s onwards, with the discovery of endogenous, morphine-like neurotransmitters and evidence that such endorphins interact with the dopamine system to 'naturally' reward all sorts of behaviours essential for the survival and reproduction of the human species, models of addiction based upon endogenous brain chemicals began to change how addiction and pleasure were represented in scientific literature and also in public domains such as the commercial media (Snyder 1989).

The case of the endorphins is one of the most obvious examples here. We may not know a lot of detail about these chemicals, whose name was invented to identify 'the brain's own morphine;' but most of us know that they are the 'feel-good' chemicals that our bodies release when we are enjoying highly-rewarding activities. Most of us have probably heard about the involvement of endorphins in producing, for example, the euphoric effect that athletes experience during endurance training: the so-called 'runner's high.' And when we hear or read about explanations of behaviour that arise from the quest for endorphins, they are, to a certain extent, understandable and logical.

Consider the scenario of the title character of the internationally syndicated comic strip, Dilbert (Adams 2005):

Figure 1. A Dilbert comic in which neurochemical styles of thought appear in relation to 'e-mail addiction.'

The ridiculous and shameful behaviour alluded to in the third panel – Dilbert sending *himself* e-mail – is explained in terms of his desire for endorphins. The reader – at least the reader with the sort of neurobiological imagination that characterizes contemporary neurosocieties of the West – can easily make the logical connections that the joke relies on for its effect. Dilbert is addicted to the pleasurable effects produced by the endorphins that his brain releases when he receives an e-mail. He enters a sort of endorphin withdrawal when he does not get his e-mails, or the resulting endorphin 'fix;' thus, although he is too ashamed to admit it, he has even resorted to writing messages to himself.

The point here is not, of course, that we take this comic strip too seriously; the point is simply that the explanation Dilbert offers for his behaviour is *plausible*, and its truth-value is ambiguous. Does Dilbert *really* think that his behaviour and emotions can be explained in terms of his neurochemistry? For those of us who presume that certainly an explanation based on the activity of endorphins cannot be taken seriously, the neuroscientific knowledge about motivation, reward, learning, and addiction that has developed over the last twenty or thirty years might give us pause for thought. Today, it is not only possible to think seriously about behavioural compulsions as a form of

brain disorder that is essentially similar to the state of 'reward dysregulation' brought about by the use of addictive drugs; it is also possible to invoke scientific evidence in support of the claim that this way of thinking is a matter of biological fact. Moreover, it is possible to manage behavioural compulsions with the same sorts of brain-targeting, anti-craving medications that are used to treat heroin addiction and alcoholism.

This chapter investigates such developments and relates their emergence to a range of epistemological, technological, political and economic conditions. But it also examines how these developments are reconfiguring the very same scientific, clinical, and personal fields from which they have arisen. It argues that just as the contemporary neurosciences of addiction are creating new ways of thinking about and acting upon behavioural problems *as* addictions, these new discourses are feeding back into, and reconfiguring, the meanings of addictions and of behavioural problems, and are also changing the experiences and understandings of individual subjects. To at least a limited extent, there is evidence that novel forms of 'neurochemical subjectivity' are becoming possible – ways of thinking about the self (even the non drug-addicted self) through the brain, or at least linking what is going on with our thoughts, feelings, and behaviours to what is happening among the cells, neurotransmitters, and communication pathways inside our heads. This neurochemical subjectivity is most apparent when we think about a mental problem such as depression – which we understand as linked to our serotonin levels, and which we treat with selective serotonin reuptake inhibitors. But it is also evident in the ways that we think about our pleasures and desires today.

The chapter focuses on a period of roughly one decade, from the mid-1990s onward, in which naltrexone was being introduced into the treatment of behavioural compulsions. It argues that the technoscience of naltrexone (not only the pharmacological substance, but also the practices and understandings in which its use is embedded) has played a key role in bringing about a shift in the neurobiological truth-status of behavioural addictions. The fact that naltrexone, a brain-targeting addiction medication, has been able to manage behavioural compulsions effectively has led to the conclusion that such compulsions arise in the area of the brain that naltrexone targets. (Since the effective treatment works on the brain, the problem becomes, as a matter of presumed fact, a brain problem.) In order to highlight this shift, the chapter begins and ends by examining the portrayal of behavioural addictions in print media that report on cutting-edge addiction neuroscience in two different periods – first in the early 1990s, and then in the early 2000s. These dramatically suggest the power of contemporary neuroscience to make up new kinds of thought, action, and subjectivity. However, they do not form, nor are they essential to, the bulk of the chapter; they simply provide a way into and out of an analysis of scientific and clinical research, which is the focus of the present investigation.

Behavioural addictions before the neuroscientific facts

Over the last thirty years, it has become commonplace to describe persistent and problematic forms of behaviour such as gambling, sex, and shopping as addictions. And indeed, since the 1960s, addiction models have been used by psychotherapists and self-help groups. It is only in the last decade or so, however, that neuroscience experts have come to consider behavioural compulsions as genuine biological disorders that are essentially similar to drug addictions. It was in the early 1990s, as the pathways of neurotransmitter activity had come to be mapped for most of the

drugs associated with addiction, that scientists began to subscribe to a relatively unified conceptualization of addiction which focused on the activity of the brain's dopaminergic system. It was in this period that, as a result of the development of new understandings of the endogenous neurochemistry of pleasure and reward, a number of new questions emerged about behavioural compulsions. If drug addiction is a state of pathological desire that results from a 'reward dysfunction' in the brain circuitry, what about other pathological desires – that is, desires that do not involve drug use? If all pleasure and all reward is understood to have a common underlying basis in the molecular physiology of the brain, and if what is being sought in addiction is not drugs themselves but primary neurochemical rewards, could *any* pleasurable behaviour that is desired too much be thought of in terms of a neurochemical disorder – a disease of the brain?

In 1990, right around the time when the dopamine hypothesis of addiction was first coming to be formulated, Isaac Marks controversially suggested in the *British Journal of Addiction* that emerging scientific evidence indicated that the differences between drug addictions and behavioural (or 'non-chemical') addictions were not as great as it had frequently been assumed (Marks 1990). On the one hand, both involve compulsive behaviours. Since at least the 1970s, scientists had come to understand that drug addiction involved behavioural compulsions (e.g., drug-seeking behaviour) that persisted long after active drug use had been stopped. Thus, Marks notes that "[s]ubstance abusers usually become both behavioural and chemical addicts. They condition [sic] to cues connected with their drug taking." And the source of behavioural compulsion in substance addictions is the same as the compulsions

involved in non-substance addictions, since "[s]trong conditioning to external cues also occurs in behavioural addictions" (Marks 1990: 1392).

On the other hand, by the end of the 1980s it had become possible to think of drug and behavioural addictions in terms of a common neurochemistry. The psychological and behavioural problems associated with drug addiction had begun to be explained in terms of the endogenous structures and chemicals of the brain, and evidence was suggesting that there was significant overlap between the neurochemistry of natural rewards for (non-drug-related) behaviour and the neurochemical rewards elicited by drug-use. The implication was that at the most fundamental level, both substance addictions and behavioural addictions could be thought of as (neuro)chemical addictions. Thus, Marks notes that "[s]ome brain mechanisms may be common to the establishment and maintenance of all addictions, be they chemical or behavioural" (Marks 1990: 1391).

These novel biomolecular understandings of addiction (e.g., the positing of addiction as a condition involving the reward pathways of the brain) did not go unnoticed by the popular media. A 1993 science and technology report in *The Economist* provides us with a fairly representative (though somewhat more in-depth) example of press reporting. The article, entitled "High and hooked", describes how a better neuroscientific understanding of addiction may be providing benefits for medicine and, as the subtitle indicates, "for recreation" (Economist 1993). If recreation seems out of place in the title, the photo montage on the opening page helps to clarify things: images include drug paraphernalia – pills, vials, powders, a needle and spoon – but also a roulette wheel and a Nintendo Gameboy. As the article goes on to discuss, all

of these produce neurochemical pleasure in very similar ways; and all potentially cause addiction.

The article begins with a discussion of the recent neurochemical links scientists have been able to make between pleasure and drug addiction, which are described as being 'obviously connected.' It provides an overview of the ways that various drugs interfere with synaptic activity within the brain's neurotransmitter systems, which furnishes the reader with an understanding of the biomolecular relationship between pleasure and addiction: "It is by subverting some of these synapses, and thus some of the brain's pathways, that drugs produce pleasure. It is through changing them in a more fundamental way that the drugs cause addiction" (Economist 1993). In a discussion of the place of dopamine in the general neurobiological theory of addiction, the text explains that due to the complexity of the brain, drugs acting on one system of neurotransmitters (for example, heroin acting on the endorphin system, or nicotine acting on the acetylcholine system) have secondary and tertiary effects on other parts of the brain, and that the dopamine system is understood to be activated in such an indirect manner by most drugs.

The article then makes it clear that, on the basis of neuroscience discoveries, the range of things that can be addictive is now understood to be wider than the range of addiction-causing drugs. "Foreign bodies in the synapses are not an absolute prerequisite for an addiction", it explains, because *any* behaviour that results in intense pleasure can presumably "over-stimulate some parts of the brain's wiring – can produce similar effects" to those of addictive drugs (Economist 1993). The article insists that while many people still currently see the application of the addiction

concept to non-substance related behaviours as problematic, there seems no reason to believe that compulsive forms of sex, gambling, and so on are necessarily in a different class from 'compulsive chemical-taking.'

Indeed, since there is evidence that many, if not all, addictions affect the dopamine system, this common denominator may provide the basis for rethinking pleasure and addiction in altogether neurochemical terms. The article suggests that we might think of crack heads, heroin junkies, pathological gamblers, and others similarly addicted to the pleasure-inducing chemicals in their brains as 'dopamine heads.' All individuals rely on dopamine for normal rewards and functioning; and all individuals, it seems, can possibly become dependent on elevated dopamine levels. After all, "the effects that cause pleasure in the short term are those that cause addiction in the long term" (Economist 1993); and thus, addiction it seems, can be understood as a (bio)logical result of overexposure to intense pleasures. Although individuals likely have varying degrees of susceptibilities to the enjoyment of behaviours and the pleasures of dopaminergic rewards, this may be a difference of degree, rather than of kind.

It might be suggested that as the dopamine hypothesis of addiction emerged as a new way of understanding compulsive thoughts and behaviours, the 'dopamine head' emerged as a new sort of species-type: a class of people whose experiences and actions had become newly intelligible – and, at least in theory, essentially similar. Now of course, the term 'dopamine head' has not come into common usage, and one should not overestimate the significance of the term. But neither should its significance be underestimated: the emergence of the very possibility of enunciating

such a concept, which renders *all* sorts of experiences of compulsive desire common, is in itself a notable development.

Such media representations, derived from neuroscience theories and models, may contribute to the making up of neurochemical selves by contributing to a process similar to the one referred to by Joe Dumit as 'objective self-fashioning' (Dumit 2003). Dumit suggests that in contemporary societies, an increasingly important component of personal identity is the 'objective self,' which "consists of our taken-for-granted notions, theories, and tendencies regarding human bodies, brains, and kinds considered as objective, referential, extrinsic, and objects of science and medicine" (Dumit 2003: 39). For Dumit, the objective self is fashioned through individual encounters with the "received-facts" of science, which provide persons with ways of understanding themselves, their bodies, and their brains as things that can be known and acted upon in (apparently) precise, concrete, and non-subjective ways. Dumit suggests these 'objective' selves are actually dependent on how bodies and brains have come to be known by scientists and other experts (processes that this thesis have been investigating in relation to addiction), and how we, as individuals come to know our bodies and brains. And while he investigates the ways that neuroscientific knowledge about depression – and especially representations of depression obtained from positron emission tomography (PET) scans – influence the subjective understanding of individuals diagnosed with depression, the person-with-depressed-brain is just one of many possible forms of objective selfhood. Self-understandings that arise as a result of our reception of scientific facts of course come from a variety of sources.

Thinking in terms of Dumit's concept of objective selfhood enables us to approach questions about neuroscience, brain chemistry, and identity from a perspective that highlights the essential contribution of neuroscience (within particular cultural, historical and institutional contexts) to our current understandings of our selves. However, Dumit's discussion of the process of objective self-fashioning is limited to the sphere of representations – ideas, discourses, and statements – and he has little to say of how *doing* things to ourselves may provide another, possibly more powerful, means of objective self-fashioning. For example: although he discusses how PET scans provide a powerful way for visualizing one's objectively depressed brain (through the generation of images that represent the self), he does not consider the relationship between this particular form of objective self-fashioning and the aspects of objective selfhood that arise from individual experiences on selective serotonin reuptake inhibitors (SSRIs) such as Prozac. Dumit argues that "we fashion our objective-selves with [...] received-facts in our own continuous and often creative manner, no matter how skeptical we are" (2003: 40). But there may be a significant difference between self-fashioning with received-facts and self-fashioning with what we might call 'received-acts and technologies.'[11]

This is to suggest that we need to consider the process of self-fashioning not only in relation to *representations* that we receive from science, but also the role of more material *interventions* into our selves – ways of acting upon our selves that we receive from science. These include guidelines, instructions, advice, but also, essentially, medications. There may be a significant difference between seeing a scan

[11] Of course, the distinction between the two is not as simple as this dichotomy suggests (since representations are always involved in a sort of intervention); but it still seems useful to think about the types of pharmaceutical actions that the contemporary neuromolecular science of addiction make possible, and the ways in which such actions interface with notions of selfhood and identity.

representing one's depressed brain and assimilating that image as part of one's identity, and, in contrast, experiencing oneself shifting from a depressed person to a non-depressed person as a result of taking (what are understood to be) brain-targeting pharmaceuticals. Representations may "function as particularly powerful resources because they bear the objective authority of science" (Dumit 2003: 44); but then again, they might not be quite as powerful as scientists would hope – or as Dumit would suggest. Plenty of scientific facts – especially those of biopsychiatry – are intensely disputed; and the meanings of PET scans in relation to mental illnesses are the topic of heated, ongoing debates. And indeed, one might ask whether PET scans would so easily be accepted as evidence of depression being a brain condition if the neurobiological facts of depression had not already been so well-established by (among other things) the experiences of millions of individuals taking SSRIs and having their symptoms of depression ameliorated. Certainly, neuroscientists themselves have noted that the perceived efficacy of the SSRIs has played an important role in thinking of depression as an imbalance in the serotonin system – despite the fact that a causal link has never been established between serotonin levels and depression (Healy 2004: 11).

This is to suggest, then, that pharmaceutical technologies may play a considerable role in redefining illness and refashioning selfhood. Quite simply, sometimes actions (treatments) speak louder than words (theories). It may have been the case, as Marks and *The Economist* suggested in the early 1990s, that a theoretical distinction between substance- and non-substance-related addictions could not be maintained on the basis of chemistry (since behavioural addiction *could* be shown to involve the chemistry of the brain) or behaviour (since drug addictions were to a significant extent dealt with

as behavioural compulsions). Because at one level all addictions (to drugs or anything else) could be thought of as the same, it may have become possible for a new type of brain-based identification to be adopted by individuals suffering from a wide array of compulsions and problematic behaviours. But it also may not have been the case that these new sorts of identifications would have been adopted on the sole basis of these theoretical (and highly contested) propositions. These theories were developed by extrapolating from established neuroscience facts, but did not become anything like facts themselves until naltrexone began to be used in the experimental treatment of a range of compulsive behaviours. It was only after this point – the point at which the neurochemical origins of compulsion became locatable, targetable, and controllable through brain-based interventions – that behavioral compulsions began to appear as neurochemical facts.

It was at the end of the 1990s that reports began to suggest that naltrexone targets pathological desires for rewarding behaviours in ways that are essentially similar to the ways it targets the cravings associated with drug addiction. Over the past decade, naltrexone has been studied and reported as an effective treatment for a range of behavioural compulsions and urges that have come to be associated with dopamine activity:

Compulsive buying. In a 2003 review of 'the psychopharmacology of compulsive buying,' researchers in Stanford's Department of Psychiatry and Behavioral Sciences state that current evidence, tentative and anecdotal though it is, suggests that pharmacologic interventions may be helpful for treating 'compulsive buying disorder' – or in lay terminology 'shopping addiction' (Bullock and Koran 2003).

Also in 2003, Grant reported observing that treatment of compulsive buyers ('shopaholics') with high doses of naltrexone led to "partial or complete remission of urges to shop and compulsive shopping behavior" in three patients (2003: 223).

Compulsive Sexual Behaviour. Anecdotal reports have also recently emerged on the potential effectiveness of naltrexone in treating 'compulsive sexual behavior' (CSB). Researchers have hypothesized that CSB and drug addiction are comparable conditions insofar as they seem to be 'urge-driven disorders,' and that naltrexone might therefore reduce both the urges associated with CSB and the problematic sexual behaviour itself. They report case studies which provide evidence for the possibility of treating some cases of CSB with naltrexone (Raymond, et al. 2002). A clinician working with convicted youth sex offenders has also observed that daily doses of naltrexone administered to patients resulted in lower self-reports of sexual preoccupation and masturbation, and suggested that these effects might be of use for treating non-criminal sex addicts (Ryback 2004).

Eating Disorders. Compulsions that relate to eating, too, have recently been treated with naltrexone, with promising results. The severity of bulimia nervosa, as well as 'Binge Eating Disorder,' reportedly decrease when naltrexone is deployed as an adjunct therapy to psychological treatment (e.g., Marrazzi, et al. 1995), and a recent review by experimental psychologists at the University of Sussex indicates that there is enough evidence available to support the claim that naltrexone's effects on eating behaviour result from the medication's ability to mediate the brain's reward mechanisms (Yeomans and Gray 2002).

Kleptomania. Evidence has been produced that naltrexone may be an effective therapy for managing urges to steal and compulsive stealing behaviour. In a preliminary, short-term study (Grant and Kim 2002), as well as a three-year trial (Grant 2005), University of Minnesota researchers found that clinically significant reductions in the severity of kleptomania could be produced with high doses of naltrexone.

This chapter suggests that naltrexone has played an essential role in the 'objective' redefinition of behavioural addictions as neurobiological disorders; to many, naltrexone's perceived efficacy has been considerably more convincing than the mere claims of neuroscience experts that behavioural compulsions may be diseases of the brain. In a sense, the fact that a brain-targeting addiction medication works to treat behavioural problems has made it increasingly reasonable to assume that such compulsions have their origins in neurochemical processes, and has begun to resolve debates about behavioural addictions as neurological conditions.

It is not, however, a straightforward matter to refer to the disorders listed above as 'behavioural addictions,' for they are not consistently classified as such or uniformly defined. They *are* referred to as behavioural addictions by some, but the concept of a behavioural addiction is still widely contested – as is, of course, the concept of addiction itself. The current edition of the Diagnostic and Statistical Manual of Mental Disorder, for example, avoids the term addiction altogether, and uses the diagnostic category of 'drug dependence' for problems that many scientists and lay individuals would refer to as drug addictions; while it uses the category of 'impulse control disorders' to encompass behavioural pathologies such as pathological

gambling and compulsive sexual behaviour that are referred by some experts as behavioural addictions (APA 1994).[12]

So an important question might be: even if these behaviours are *brain* problems, can they be classified as *addictions*? This chapter suggests that the answer is a qualified yes. Compulsive behaviours that are treatable with anti-craving pharmacotherapies are addictions – but only within neurochemical styles of thinking about addiction, and only because the rules for the classification of addiction have changed to include any form of intense cravings or urges that can be treated with anti-craving medications. Whether described as addictions, dependencies, impulse control disorders, or something else, these behavioural problems are bound up together (along with drug addictions) in the contemporary biomedical problematization and psychopharmacological management of desire.

Using the term 'addiction' in this specific sense may be misleading, given the lack, even within neuroscience discourses, of such a strict and explicit definition. The remainder of this chapter will escape the morass of multiple and often contradictory expert terminologies and classification systems by avoiding relying upon any of them for analytical or descriptive purposes. Instead, it will provide its own. As a class of problems, the disorders under investigation can be usefully thought of as *pathologies of desire*: disorders in thought, affect, and conduct that have been conceptualized in terms of dysregulated activity within the pathways of neurochemical reward.

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[12] Of course, despite its considerable authority, the DSM is only one classification system, and clinicians and researchers by no means orient their work or their writings to it. And indeed, impulse control disorders, while classified as distinct from dependence, have in fact been most commonly treated with the tools, technologies, and understandings of the field of addiction studies. **Tavares, H., Zilberman, M. L. and el-Guebaly, N.** 2003 'Are there cognitive and behavioural approaches specific to the treatment of pathological gambling?' *Canadian Journal of Psychiatry* 48(1).

Pathologies of desire include traditionally defined addictions to licit and illicit substances, but also compulsive thoughts about and pursuits of rewarding activities such as gambling, sex, and eating. They all entail, in terms of clinical description, forms of unwieldy desires (such as harmful preoccupations, misguided urges, difficult to control impulses, and / or unwanted cravings), and most can be treated with anti-craving medications.

The following section investigates how has it become possible to use naltrexone – originally a treatment for the most feared of addictions, e.g., morphine and heroin – as a means for dealing with an expanding array of pathological desires and the behavioural problems with which they are associated. One way of doing this might be to provide a general overview that attempts, briefly, to summarize each of the fields in which naltrexone has been deployed – from eating disorders to sexual compulsions, for example. However, such an approach might imply a story about naltrexone that is *too* general. In order to avoid painting these developments with too broad a brush, a case study approach is taken in the following section, with one particular pathology of desire – namely, pathological gambling – investigated in detail.

PATHOLOGICAL GAMBLING: A CASE STUDY

Among the compulsive forms of conduct that have been linked to addiction, the most sustained and significant biomedical research has been done on pathological gambling. Since the early 1980s, researchers have begun to draw comparisons between physiological aspects of gambling on the one hand, and drug addiction or dependence on the other. In a study that is still frequently cited, Wray and Dickerson (1981) observed that some high-frequency gamblers exhibit withdrawal symptoms similar to

those exhibited by individuals withdrawing from narcotic and stimulant drugs. Over the last two decades, a small number of physiological investigations have led many researchers to argue that pathological gambling is best thought of as an addiction or a dependence without a drug that has a biological basis (Coventry and Brown 1993; Dickerson 1989).

While the field of pathological gambling treatment has been, and continues to be, dominated by psychotherapists who eschew biomedical approaches to the disorder, a limited number of research clinicians and scientists have investigated the use of psychopharmaceuticals in the treatment of pathological gambling. The most studied of these have been medications already used in the management of other affective disorders such as depression, mania, and bipolar disorder. Mood stabilizers such as lithium and valproate (Depakote) were found to provide some positive effects on mitigating pathological gambling, but through the 1990s the selective serotonin reuptake inhibitor drugs such as fluoxetine hydrochloride (Prozac) came to be seen as the most promising class of pharmacotherapies for gambling disorders (Grant, et al. 2003). The efficacy of the SSRIs in treating pathological gambling led some researchers to suggest that serotonin was the most widely implicated neurotransmitter in pathological gambling and impulse control disorder (Potenza 2001).

However, since the late 1990s, naltrexone – which acts upon an entirely different neurotransmitter system from serotonin – has also been shown to be effective in the treatment of pathological gambling. The first published suggestions of naltrexone's efficacy in managing behavioural addictions appeared in 1998, when clinicians reported in a letter to the *Canadian Journal of Psychiatry* their success in treating a

compulsively gambling patient with naltrexone (Crockford and el-Guebaly 1998). In the same year, the University of Minnesota's Dr. Suck Won Kim published a peer-reviewed article in the *Journal of Clinical Psychiatry* in which he reported promising results from an open study of naltrexone in the treatment of pathological gambling (Kim 1998).[13]

The deployment of the tried-and-true SSRIs for the treatment of pathological gambling was not in itself a particularly notable development in the field of addiction biopsychiatry – especially since an incredibly wide range of psychiatric disorders, including schizophrenia, panic disorder, dementia, narcolepsy, and attention deficit and hyperactivity disorder (ADHD), have been noted to improve as a result of treatment with this class of drugs. But the hypothesis that naltrexone could be used to treat pathological gambling, and findings in support of that hypothesis, have been described as a development of almost paradigm-shifting proportions in the fields of addiction studies and gambling treatment.

While the discovery that some pathological gambling responds to treatment with SSRIs is not of much interest in the context of the present study, the distinction amongst pathological gamblers between SSRI-responders and naltrexone-responders is a more interesting development. With the observed efficacy of naltrexone, it has become possible for researchers to distinguish between two sub-species of pathological gamblers, each of which benefits from a different regime of treatment. Gambling problems that improved by treatment with SSRIs came to be seen as a

[13] It should be noted that while Kim describes pathological gambling as an impulse control disorder in this article, he also refers to gambling as an addiction.

result of problems such as loneliness or depression, whereas gambling problems that improved as result of treatment with naltrexone came to be understood as arising from pathological cravings. This new means of distinguishing classes of depressed gamblers from desiring gamblers will be discussed towards the end of this chapter; for the moment, the focus will be on the birth of the latter subspecies of problem gambler – the subject of pathological desire.

Pathological gambling and urgent desires

In an interview conducted by a representative of the North American Association of State and Provincial Lotteries, Kim, who is the most prominent scientist involved in naltrexone studies for pathological gambling, notes that there is considerable scepticism among those who first learn of his innovative treatment:

> almost every time the general public hears about this, they wonder, "How can it be possible?" It's almost unimaginable, because gambling is traditionally viewed as a psychological problem or weakness of willpower. If I didn't study the brain, even I would have thought it's a primarily psychological disorder. But, anybody who studies the brain and understands that there are specific brain regions that process human craving and human motivation will understand this approach (Kim 2002).

It is interesting to note that, indicative of his neurobiological thought community, Kim implies that the truth about pathological gambling is indissociable from facts about the brain, and that these truths are self-evident to those who actually do understand the brain. But the element of Kim's response that we will focus on is the question he describes himself as being frequently confronted with when non-experts hear that pathological gambling can be treated with an anti-craving medication: 'how can it be possible?'

How did naltrexone treatment for behavioural compulsions move from the unimaginable to the imaginable, to the possible, and, ultimately, the logical? In his

own account, Kim explains that his theory resulted from an intensive quest for knowledge about the relationship between dopamine and human urges that involved "living in the library six months – literally day and night", and immersing himself in scientific literature on addiction, impulse control disorders, and the neurobiology of craving. During this period, he began to conceptualize urges as the central problem in impulse control disorders (ICDs), to relate the urges of ICDs to the cravings involved in drug addiction, and to search for ways to apply these ideas to "non-drug addictive behavior such as gambling" (Kim 2002).

That naltrexone had already been developed, studied, and shown to be effective in managing a number of behavioural problems (associated with urges and dopamine activity) is at least implicitly acknowledged by Kim as an important factor in making his therapeutic approach imaginable. Citing a number of studies that date as far back as the mid-1970s, Kim and his colleagues note in one of their papers that:

> Support for the use of naltrexone in the treatment of pathologic gambling comes from evidence of its effectiveness in the treatment of alcoholism, bulimia nervosa, drug abuse, borderline personality disorder with self-injurious behavior and other psychiatric disorders in which urges are the dominant symptom (Kim, et al. 2001: 914-915).

Kim focuses almost exclusively on abstract ideas, however, and does not make any reference to the array of truth-producing technologies and methods that make certain ideas thinkable and researchable – psychometric scales for measuring craving, observational techniques, standards for evaluating treatments, and of course, pharmacological substances themselves. Nevertheless, he acknowledges, to an extent, that the existence of a thought style which had already developed understandings of urges, pleasures, and desires as neurochemical phenomena was an essential condition for his ideas to become possible. It was not simply a matter of careful logical

reasoning that led Kim to propose using naltrexone to treat pathological gambling; naltrexone had already been posited as a means of managing problematic impulses and desires. Studies indicating as much provided a pharmacological rationale for attempting to use naltrexone to treat gambling disorders – especially since, by the mid-1990s, it had been observed that a side-effect of naltrexone was the reduction of desire for eating, sexual intercourse, and other pleasurable behaviours.

But if we are to answer the question of how it has become possible to treat pathological gambling with naltrexone, our investigation has to proceed beyond an examination of the conditions of possibility within the neurochemical style of thinking about addiction, and the invention of anti-craving medications. We also have to consider the political and economic factors that are relevant to the research and experiments of Kim and other biological psychiatrists actually having been done in the first place. For these innovations depended not only on ideas and technologies, but also on a range of tangible resources in the form of institutional support, laboratory and clinical space, researchers themselves, and of course funding.

As already mentioned, pathological gambling has for many years stood out as the behavioural compulsion most readily accepted as comparable to addiction, and as the most exemplary form of non-chemical addiction. Nevertheless, in the mid-1990s, gambling problems were still not considered an important area of basic and clinical research in the biomedical sciences. It is only in the last decade or so that research into pathological gambling as a neurological disorder has come to be considered important; and this has coincided with the emergence and continued support of such research by industrial, institutional, and funding organizations. If we want to be more

specific, we might suggest that neuroscience research into pathological gambling became an important area of investigation in 1996 – the year that the US National Center for Responsible Gaming (NCRG) was formed.

The NCRG was established as the first national organization in the US dedicated to funding peer-reviewed scientific research on pathological gambling. Its $875,000 start-up funds and the vast majority of its multi-million dollar research funding program have come from donations from individuals and corporations involved with the gaming industry, and its board of directors includes a number of gaming industry executives (including, as President, William Boyd, the Chairman and CEO of Boyd Gaming Corporation). But the NCRG describes itself as a tax-exempt, non-profit, and 'independent' organization, and formulates its mission as increasing interest in, and improving the quality of, scientific research into pathological gambling. It aims "to be the leading source of science-based research and information on gambling and health, advancing education, prevention, treatment and public policy" (NCRG n.d.-c).

And indeed, after nearly a decade, the NCRG can boast of having wielded significant influence on research into pathological gambling, and of having helped transform the field of gambling research:

> Today, with the contributions of the casino gaming industry, equipment manufacturers, vendors, related organizations and individuals, more than $13 million has been committed to the NCRG, an unprecedented level of funding for gambling research. This financial support has enabled the NCRG to attract the best minds from the most prestigious institutions to conduct research in this uncharted field (NCRG n.d.-a).

Between 1996 and 2003, 23 academic institutions received NCRG funding for gambling research, including the University of Michigan, McGill University, and the

medical schools of Harvard and Yale. The most significant alliance between researchers, academic intuitions, and industry sources of funding was established in 2000, when the NCRG provided the Harvard Medical School's Divisions on Addiction with $2.4 million to create, under the direction of Howard Shaffer, the Institute for Research on Pathological Gambling and Related Disorders (IRPG). The Institute subsequently took over from the NCRG the responsibility of awarding the NCRG's research funds to scientific projects.

A major initiative of the IRPG has been, like the NCRG, to fund research on the biological causes of pathological gambling, and on pharmacological treatments for the disorder – and here we return to Kim's naltrexone studies. NCRG research funds were awarded to Kim to conduct the first ever double-blind randomized study for the naltrexone treatment of pathological gambling (HMS 1998). The successful results of this study were published in seven prominent journals, and have received significant attention from both the media and biomedical researchers. But it represents only a small portion of the output and impact of the NCRG. NCRG-funded studies have led to more than 100 peer-reviewed publications, including articles in some of the most prominent academic and professional journals such as *Neuron*, *The American Journal of Psychiatry*, *Psychiatric Times*, *Biological Psychiatry*, and *The Journal of the American Medical Association.* According to PsychINFO and Medline indexes, journal article citations using the term "gambling" increased by 77% between 1996 and 2003; and references to "pathological gambling" have increased by 123.5% (NCRG 2003). Moreover, the NCRG has provided funding for the production of *The Wager*, a weekly research report that has a circulation of 14,000 and was recommended in the Technology Section of the New York Times.

The political economy of neuroscience truth-making

In its 2003 report on the "Impact of NCRG on the field of gambling research", the NCRG suggests that the field of gambling research "pre-NCRG" was characterized by "junk science" that was "[d]riven by political or personal agendas" (NCRG 2003) rather than the pursuit of knowledge through rigorous scientific methods – with the implication that since the NCRG has entered the fray, much of this has changed. Of course, one can easily discern that the agenda of the gaming industry may be better served by some types of research than others (for example, research that indicates that the condition is best thought of as a neurological pathology rather than a social problem – resulting from exposure to gambling, for instance – may deflect blame from the gaming industry). But this does not mean that science driven by political agendas is incongruous with rigour and objectivity.

There is actually very little evidence to suggest that the scientific and therapeutic developments described in this chapter can be explained as resulting from the unilateral influence of large gambling corporations, or from any sort of academic fraud or misconduct that would bring into question the objectivity and validity of NCRG-funded research. There is no evidence that the gaming industry has 'bought out' leading researchers and institutions that were willing to sacrifice the ideals of scientific objectivity and independence in order to secure financial rewards; and the research that has been funded by the NCRG has been reported in peer-reviewed journals, with full disclosure of the support of the NCRG. In the specific case of naltrexone, the medication had already been identified as an anti-craving therapy that could reduce rewarding behaviours, and experts interested in non-gambling behavioural addictions have formulated similar hypotheses and rationales for the therapeutic use of naltrexone. Moreover, Kim's work in using naltrexone to manage

gambling desires has been lauded by a wide range of scholars as innovative and rigorous – not decried as suspect or questionable.

Refusing to make interest-based explanations does not, however, mean that we must accept uncritically that "facts speak for themselves" – the maxim that, in the late 1990s, was featured prominently on the front page of the weekly research bulletin published by Harvard Medical School's Division on Addiction. Facts do not, of course, speak for themselves, any more than they exist independently of material and political interests, or beyond specific spheres of discourse and practice. Facts arise as events, dependent on the enunciations and definitions of qualified spokespeople according to accepted norms of objectivity, and only insofar as such spokespeople have the resources to engage in fact-production and the recruitment of allies. The neurochemical and pharmacological facts about gambling and other behaviours that are emerging are *made*, to be sure – but not by the mere presence of corporate interests and generous research grants. The latter only fulfil certain conditions that make possible the generation of truths that are consistent with the beliefs and practices of a broader scientific community.

In the contemporary field of gambling studies, resources for research into certain types of explanations about and solutions for gambling problems are vastly more plentiful than others – for example, investigation into the neurobiological factors involved in the onset of pathological gambling. Such research, which is oriented towards finding solutions for gambling problems that are based upon interventions into neurochemistry rather than the regulation of the gaming industry, is more likely to receive funding than other types of research (e.g., studies which focus on social and

legal approaches to dealing with pathological gambling). The money made available by the NCRG in 1996 was the first ever significant pot for scientific research into pathological gambling; and several years later, as one of the beneficiaries of the NCRG's funding schemes has noted, "[t]he NCRG has helped bring the study of pathological gambling into a new era" (Ken Winters, quoted in NCRG n.d.-d). Within this new era, the interests of the gambling industry do not determine the outcomes of research; but projects which seem likely to produce findings that support the agenda of the NCRG, and the gambling industry, are those which become possible to carry out.

Kim and his associates, for example, did not simply construct findings that were convenient for gaming corporations, and they certainly did not propose any completely new hypotheses, theories, or treatments for behavioural addictions. What they did do – what they were able to do with the financial support of the NCRG – was to move beyond the anecdotal evidence and conjecture acquired from a few case studies, to the production of more objective, double-blind studies of the effectiveness of naltrexone in treating pathological gambling. In so doing, they moved the neurobiological basis of pathological gambling closer to the status of a fact, reconfirming the validity of neurochemical styles of thinking about human experience and behaviour, and providing further support for understandings of and treatments for compulsive behaviours that can be traced back through decades of scientific research.

It thus seems apt to think of the influence of the NCRG in terms of what Paul Rabinow describes as 'biosociality:' a state of affairs in which nature is not simply a given that provides a basis for social processes, but in which social processes are

involved in the technical knowing and remaking of nature (Rabinow 1996). The truths about and treatments for the human brain, as produced in NCRG-funded laboratories, provide (to paraphrase Rabinow) another vehicle for science, capitalism, and culture's infiltration into 'nature.' An NCRG report celebrating corporate donations expresses one aspect of contemporary biosociality nicely, when it describes donors as the "venture capitalists of gambling research" (NCRG 1998: 18). Such donor firms do not manipulate or subvert legitimate scientific inquiry. Instead, they provide pools of capital to an organization with which they have a limited partnership (the NCRG), which in turn puts this money into promising scientific projects that would not be viable without external sources of funding. The dividends on ventured funds are not certain (because findings are not determined in advance), but if investments are made wisely (in the right sorts of projects, researchers and institutions), the return on financial capital is certain types of facts and truths about the brain, the problem of gambling, and the pathological gambler.

Since scientific knowledge is inextricably bound up with contemporary biopolitics in the promotion of the health and well-being of advanced liberal democracies, there should be no difficulty in discerning that such facts, once produced, constitute a form of *biopolitical capital*. This capital is mobilized in struggles over determining, among other things, what sorts of legal and regulatory mechanisms are required to deal with the social problems associated with the gaming industry. It is not a coincidence that the gaming industry representatives formed the National Center for Responsible Gaming in the same year that the US Congress created the National Gambling Impact Study Commission to conduct a three-year, comprehensive study of the social and economic impact of gambling on governments, businesses, families and individuals in

the United States. The Commission received testimony from two NCRG representatives who suggested, among other things, that the most current and rigorous (NCRG funded) scientific research indicates that the prevalence of gambling problems is lower than most previous studies have estimated; that there is not as strong a relationship between the increase of gambling problems and the expansion of legal gambling establishments as is often assumed; and that in any case, gambling problems are medical problems, and thus that affected individuals "deserve to benefit from the same scientific and technological advances that are helping people who suffer from depression, alcoholism and other disorders" (Christine Reilly, executive director of the NCRG, quoted in NCRG 1998: 11).

Reconfiguring pathological gambling as a mind/brain problem

In reconceptualizing (some) pathological gambling in terms of pathological desires that are fundamentally similar to drug cravings, neuroscientists have brought (non-drug) desire within the realm of the treatable. To a significant extent, as the nature of desire has been successfully remade as an objective, neurobiological phenomenon, debates about the actual, physiological – not just metaphorical – existence of non-substance addictions have begun to be put to rest. But the settling of these neurobiological facts have themselves unsettled, and begun to reconfigure, spheres of practice and experience that are not strictly matters of the brain. If the re-making of pathological gambling as a matter of neuroscience truth is a social and political process, the new, neurochemical truths of pathological gambling (and the technologies they are linked with) also provide a means for re-making aspects of society, politics, and individuality that relate to problem gambling (and other problematic forms of compulsive behaviour). The reconceptualization of pathological gambling has brought about permutations in how pathological gambling itself is

represented as a problem, how individual pathological gamblers are classified, and how gambling problems and problem gamblers are governed.

Classifying neurobiological kinds

The essence of the problem of pathological gambling used to be considered in behavioural and social terms. It was the particular type of behaviour, i.e. gambling, that provided a basis for knowing and treating pathological gamblers. The more or less monolithic classification of 'pathological gambling' provided a means for grouping together individuals who (1) could be thought of as a similar kind of subject and could be dealt with in similar ways, and (2) could be distinguished from other kinds of problems, for example eating disorders and drug addictions.

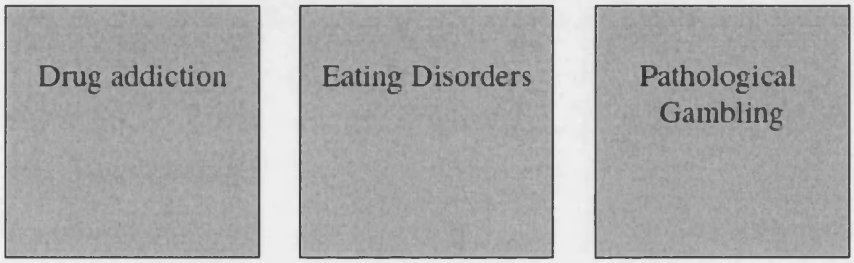

Figure 2. Pathological gambling, eating disorders, and drug addiction as distinct kinds of behavioural problems

Any differences among individual pathological gamblers were considered less important than the similarities in their behaviours; and this was the case for other behavioural compulsions such as binge eating and kleptomania.

Within contemporary neurochemical styles of scientific thought, however, the essence of the problem of pathological gambling is no longer to be found in a behaviour; it is instead to be found in a state of brain functioning. Kim arrived at a novel way of thinking about the problem of pathological gambling – or more precisely, of disaggregating the general problem of pathological gambling into more specific

classes of brain problems – as he was conducting the research described above. He notes:

> That's when I really came out with my novel idea. I really began to dissect individual parts of the illness and said, "I'm not interested in the whole gambling disorder. I'm only interested in craving gambling rather than somebody who gambles because of depression or loneliness" (Kim 2002).

In essence, Kim broke the behavioural problem of pathological gambling down into different types of mind/brain problems. As Kim's focus became the subset of problems about gambling *desire*, a new sorting scheme emerged in which some – but not all – pathological gambling could be known and treated as neurobiological problems of pleasure and reward. Kim makes it clear that he is not concerned with gambling behaviour, explicitly noting that he is "not trying to reduce gambling frequency or gambling intensity," but is instead interested in "reducing gambling urges" (Kim 2002). For Kim, the problem to be managed is not the desired behaviour (pathological gambling), but is instead newly formulated as (pathological) desire itself.

Because pathological gambling has come to be understood as a behavioural symptom of underlying primary brain disorders, pathological gamblers increasingly must be known in terms of different neurobiological kinds. Kim explains that he came to realize that he was not dealing with all individuals who experienced gambling problems but "was really dealing with only the subset of gamblers who had intense cravings, uncontrollable impulses" (Kim 2002). His studies and treatments had little to do, for example, with pathological gamblers suffering from depression or feelings of loneliness. Such individuals, understood to have an affective disorder associated with serotonin imbalances in the brain, had very little in common with gamblers

whose behaviour stemmed from cravings. For the latter group, pathological desire and a dysregulated reward system were understood to be the main problem.

Thus, neurochemical thought styles developed a system for reclassifying pathological gamblers in terms of neurobiological kinds, or what we might refer to as 'neurotypes,' that are based upon their brain chemistry. As it became clear that sub-populations of distinct neurotypes existed among pathological gamblers, no longer could all individuals who experienced problems as a result of gambling be considered in like terms. In an important sense, the pathological gambler has ceased to be a 'kind' of individual and pathological gambling has ceased to be a kind of problem. In order to know the truth about an individual, and to devise a fitting treatment, new subdivisions within too-broad behavioural classifications became necessary – that is, it became necessary to sort individuals into different groups of neurobiological kinds. Among pathological gamblers and other behavioural compulsives, subjects of neurochemical desire need to be distinguished from subjects of other brain conditions; and each class of subjects needs to be studied and treated in particular ways.

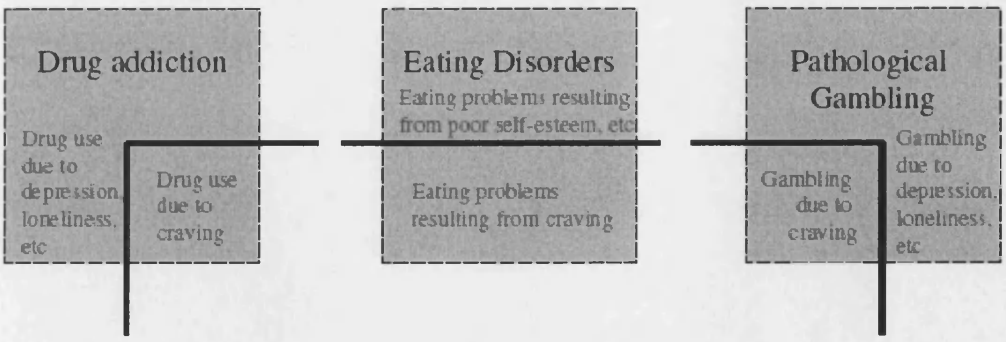

Figure 3. Behavioural kinds of problems disaggregated into different kinds of mind/brain problems

Increasingly, it is only by re-sorting individuals with behavioural compulsions into other categories – categories that are based upon an underlying neurochemical problem – that one can reliably know about the individual, and about what to do with

the therapeutic subject. Pathological desire is one of such underlying problems; and individuals classified in terms of their pathological desires end up being considered as very different kinds of subjects than those who are classified on the basis of other mental disorders. And for researchers investigating naltrexone and addiction-related disorders, these were the only subjects that mattered. Other kinds of subjects – depressed, unstable, lonely, etc. – were another problem (or series of problems) altogether, to be dealt with by other sorts of experts and interventions.

Multiplying pathologies of desire

It should be clear that the discovery of a sub-species of pathological gambler identifiable in terms of its desires has broken down a formerly coherent grouping of therapeutic subjects, and that a more complex classification system is now needed for sorting pathological gamblers. And if we distance ourselves from the single case of pathological gambling, and consider these developments in relation to similar shifts going on in the study and management of other behavioural compulsions, it might appear that we are witnessing a sort of 'balkanization' of diagnostic categories, in which broad classifications are divided into a number of smaller, essentially unlike, and mutually exclusive populations. After all, just as the specification of subjects of pathological desire has occurred within a population of problem gamblers, similar subdivisions have been made in relation to other classifications of behavioural pathology including (as listed earlier in this chapter), compulsive buying, kleptomania, compulsive sexual behaviour, and eating disorders.

But research into anti-craving therapies for the treatment of some individuals who suffer from these problems does not *only* suggest the need to disaggregate an entire range of behavioural problems; it also suggests a basis for new overarching

classifications that re-aggregate subpopulations of different behavioural problems in terms of like neurotypes. In this new biomedical taxonomy, it is not behaviour, but mental states (and associated neurological conditions) that draw together groups of individuals into coherent formations. The most fundamental truths of the problem, and the problematic subject, are not to be found in the facts of what the subject does, but what goes on in her mind and brain. Problematic conduct is not an adequate basis for sorting subjects, because problematic conduct is only a symptom of an underlying neuropathology. As John Grant, a collaborator with Kim explains: "We don't have a gambling center (in the brain) or a kleptomania center, but we do have an urge center" (Chin 2001). Subjects of pathological desire are classified as such on the basis of a shared dysregulation of a particular part of their brains that produces urges and cravings. Pathological gambling, one expression of a neurobiological pathology of desire, shares a common etiology with a range of other problematic behaviours.

And so, in the place of like behavioural kinds – kleptomaniacs, sexual compulsives, and so on – new classes of problems and corresponding sorts of subjects are emerging that are based on pathological mind/brain states. These new divisions cut across old categories, and group individuals together as new, brain-ordered kinds. The biomedical problematization of desire in general, and the study of the effects on desire made possible by naltrexone in particular, have provided the basis for a general classification of pathologies of desire. It has allowed researchers and clinicians to see problems of desire where they may previously have not, and to classify (and treat) problematic individuals as subjects of pathological desire.

Figure 4. Pathological desire provides a basis for reclassifying subpopulations

Pathological desire is used to classify subjects according to a neurochemical problem that underlies a range of problems of conduct. And of course, while it is beyond the scope of this thesis, other pathological mental states / brain disorders such as depression may also provide slots into which individuals suffering from behavioural compulsions may be sorted; the present discussion focuses on only one.

PSYCHOPHARMACOLOGICAL REASONING

It should be clear that pharmaceutical interventions played a central role in the creation of a classification system that is based on neurobiological kinds rather than behavioural types. The need for this new classification system became apparent when, after naltrexone was given to an entire cross-section of an (undifferentiated) population of pathological gamblers, it was noted that only a subset of individuals within this group benefited from the treatment. Some pathological gamblers reduced or stopped their gambling behaviour when they were administered the anti-craving drug naltrexone. But other pathological gamblers seemed unaffected by naltrexone – their behavioural problems did not respond to anti-craving therapies. The difference in treatment results suggested to researchers a difference in the etiology of gambling

problems in the two groups. Changes in the gambling behaviour of these naltrexone responders was explained in terms of naltrexone's effects on the neurological systems upon which naltrexone acted – i.e., the endorphin and dopamine systems implicated in urges and craving.

This specific sort of looping effect, in which neuroscience representations of a condition depend upon feedback from brain-targeting pharmacological interventions, might be thought of as a form of *psychopharmacological reasoning*. As this chapter has shown, pathologies of desire are inextricably linked to the sort of power that is brought upon the body and the brain. The pathologically desiring kind, understood in neurochemical terms, has essentially depended upon the effects of naltrexone on the brain, and the effectiveness of naltrexone on clinical subjects. So treatment has remade addiction, producing its own subdivisions and rearrangements: new slots were created into which subjects were to be fit. On the one hand, these new constellations cut across and bring into question previously unified and coherent classifications; on the other hand, they make possible a new way of unifying some of these subdivided parts.

Thus, naltrexone can be usefully thought of as a sort of transformative governing technology, which reorganizes subjects, and indeed produces new sorts of (newly governable) subjects at the very same time that it works on them. Within neurobiological styles of thought, any behaviour that responds to anti-craving treatments can be classified as an addiction – or better, as a 'pathology of desire,' since the term 'addiction' remains confusing and inadequate. As naltrexone is deployed to manage an increasing range of problems, naltrexone provides a means of

multiplying forms of problematic desire: because pathologies of desire transcend behavioural classification, they are able to proliferate within all sorts of behavioural problems. When Kim is asked by an interviewer whether he and his colleagues think that naltrexone therapy "would ultimately work for a very broad range of behaviours", Kim's response is unequivocal: "We really believe that" (Kim 2002).

A recent article co-authored by several members of Harvard Medical School's Division on Addiction, which proposes a 'syndrome model of addiction' (Shaffer, et al. 2004), illustrates this form of reasoning. A syndrome is defined as an assortment of symptoms and signs that relate to an underlying condition, only some of which are present in any particular case. According to this model, addiction is best understood as an underlying pathology that manifests itself in diverse ways that may not appear, on the surface, to be related. Thus, these researchers suggest that, while up to the present, addictions to drugs, alcohol, gambling and other substances have been treated as separate and distinct disorders, contemporary research suggests that "the current view of separate addictions is similar to the view espoused during the early days of AIDS diagnosis, when rare diseases were not yet recognized as opportunistic infections associated with an underlying immune deficiency syndrome" (Shaffer, et al. 2004: 365).

While these authors do not focus exclusively on brain-based theories of addiction, they do note that research into neurobiological reward activity provides the best-known evidence in support of an addiction syndrome. And indeed, as the NCRG notes, this evidence suggest that since psychopharmacological treatments which regulate the reward system and modulate cravings for a wide range of drug addiction

also work on non-drug compulsions, "perhaps our existing treatments are actually more advanced than our addiction philosophy," and that "all that is required is a rethinking of addiction" (NCRG n.d.-b). This suggestion, that treatments for addiction may require a rethinking of addiction, and that therapies may thus precede models of disease, supports the idea that there exists a looping effect between addiction interventions and representations. A range of disorders have come to be represented, described, defined, and reclassified in terms of the physiological changes that effective medications bring about.

By leading to a reconceptualization of pathological subjects as neurobiological kinds, brain-targeting interventions have also played an important role in bringing about new logics and strategies for governing social problems. With the identification of a class of subjects whose problem gambling could be thought of as pathologies of desire, it became not only possible, but apparently rational, to make a shift from governing conduct (e.g., compulsive gambling behaviour) towards governing affective mind/brain states (craving). The problems of compulsive conduct are thus coming to be formulated not as behaviours, but as the states that precede those behaviours. These states still form part of a social problem, because they lead to behaviours that have negative consequences for individuals, families, and communities. But the ontology of the social problem has changed: it has less to do with the legal and environmental spheres within which the individual is situated, and more to do with the space *inside* the individual – the human brain. The space in which the problem was thought to be most fruitfully worked upon correspondingly shifted from the interface between the individual and the social / environmental (e.g., regulating the gambling industry) to the interface between the mental and neurobiological – in this chapter's

example, the mind/brain states of desire. This does not seem to indicate a 'death of the social,' but rather indicates an emergence of a new sort of sociality in which the distance between social problems and brain problems is significantly diminished. Within contemporary societies, the neurochemical subject is becoming an increasingly important conduit of political power and social government.

Just as neurochemistry is providing a basis for understanding the individual, psychopharmacology is providing a means of governing social problems by acting on neurochemical subjects. And naltrexone governs in a very particular way: by acting on pathologies of desire, not diseases of the will. It is not weak will that becomes the subject of pharmacotherapeutic treatment, but strong desire. The condition being treated is not caused by insufficient self-control, but by excessive drive intensities that arise from dysregulated neurons in the pleasure / reward system. As Kim notes in the first published 'systematic investigation' of naltrexone in the treatment of pathologic gambling that was sponsored by the NCRG:

> Naltrexone inhibits dopamine neurons within the ventral tegmental area and diminishes dopamine function within the nucleus accumbens and its juxtaposed basal brain region [...]. Dopamine function within these regions has been implicated in the subjective experience of pleasure and urges [...]. One of the core symptoms of pathologic gambling is an uncontrolled urge triggered by a potential reward. We hypothesized that naltrexone would dampen the urges and pleasures associated with gambling (Kim, et al. 2001: 914).

Kim's rationale for using naltrexone was that if it "could knock out the urges, everything else would crumble," and notes that "that's exactly what we have seen" (quoted in Marcotty and Lerner 2001).

In the formulations of Kim and his associates, and those of the vast majority of neuroscientists investigating how opiate antagonists are effective in treating addiction

or similar disorders, naltrexone is deployed with the intention of reducing the magnitude of pathological desires and urges to the point where they are manageable or unproblematic, and problematic conduct disappears. What is being fixed or treated, then, is not the (in)ability to exert self-control over these drives or behaviours themselves, but impulses, urges, and desire itself. Control over one's desire is not achieved through an exertion of the will, or a force of self-control. Instead, it is achieved by a neurochemical-targeting substance that regulates the activity of molecules of the reward system. Naltrexone provides a means of governing the neurochemical subject at the precise origin of problematic desire – in the brain.

The importance of this distinction may, in certain respects, seem rather minor: does it matter whether desire is reduced or will-power is increased, since the end result (in ideal circumstances) is that the individual once again gains control over herself? Perhaps it does not, if one's concern is treatment outcomes. But it becomes more significant when considering naltrexone in relation to the subject, and in relation to what, precisely, in the subject, is being targeted. "I am taking this pill to bring my desires under control" is a statement quite different from "I am taking this pill to increase my will-power." In the latter case, medication enhances the ability of the subject to engage in conscious, purposive action: the pill is not the mechanism of control, it does not make decisions about *what* to do; it only helps the subject realize his or her own goals. An enhancement of will-power would allow the subject to resist desires if s/he chose. In the former case, however, the pill becomes the active agent: upon entering the body, it autonomously seeks out desire, neutralizes or incapacitates it to a degree that it is no longer a threat to well-being. The individual does not have to decide what to do about desire, because it is automatically neutralized.

A medication given and/or taken as an adjunct of the will would not involve a rationale that relates to the specific, neurochemical modulation of desire. It would be enough to leave desire as it is, and overwhelm it by the sheer, opposing force of the will. Desire would not be what must be scientifically understood, but what must be overcome: ruled instead of administered, dominated rather than organized. But naltrexone as an anti-craving therapy, as a regulator of desire, is administered as a logical, rational therapy that is understood to work at the molecular level to regulate the neurochemical processes involved in pleasure and craving. Desire is not some unknown force that must be overpowered, but is instead a neurochemical configuration that can be precisely manipulated with the proper tools and understandings.

Of course, these two logics of treatment (enhancing the will and regulating desire) might perhaps be combined within the understandings of an individual (whether doctor, patient, or other) who equates a decrease in desire with an increase in will-power, or vice-versa. But such is not really the case in neuroscience texts – the objects of my investigation here – where mention of the will is all but absent, and even the notion of self-control is infrequently mentioned. These absences are especially conspicuous when neuroscience discourses are compared with those of psychoanalysis and self-help: notions of self-control and will-power proliferate in the latter. This should not be entirely surprising, given the humanist essentialism with which these concepts are so closely associated, and to which contemporary psychopharmacology is opposed (Healy 2002).

Like the 'rational' pharmacologics of contemporary drug addiction treatment, today's psychopharmacological treatments for behavioural addictions no longer focus on teaching subjects methods of self-control, or exhorting them to muster their ethical resources to fight a moral battle. Instead, intense cravings are understood as a logical, predictable outcome of prior learning and conditioning processes which have dysregulated the brain's delicate neurochemical balance; and research and treatment are oriented towards correcting these dysfunctional processes, with the expectation that once pathological desires have been eliminated or sufficiently attenuated, the individual will no longer feel compelled to engage in activities that were formerly experienced as compulsive. With the use of opioid antagonists or other anti-craving pharmacotherapies, the problematic conduct of the behavioural addict becomes literally undesired.

CONCLUSION

This chapter has investigated how it has become possible to think of behavioural compulsions as physiologically 'real' addictions, and to treat them with the same anti-craving medications that are used to treat drug addictions. The origins of a broad category of (neuro)pathologies of desire can be traced back to the developments that led, in the early 1990s, to the dopamine hypothesis of addiction. By this point, all drug addictions had come to be thought of, to a significant extent, in terms of the brain's own neurochemical system of pleasure and reward. And it became possible to suggest that since the root of the neurobiological problem of addiction was conceptualized as a matter of endogenous brain chemistry, all behaviours that were motivated by that endogenous chemistry could potentially be addictive. Today, as representations of addiction that focus on the role of dopamine and the endorphins have come to occupy a central position in neurobiological styles of thought, the

concept of behavioural or non-substance addictions is becoming much less controversial than it once was. The underlying neurochemistry of addiction makes it difficult, perhaps even impossible, to distinguish between drug and non-drug addictions.

But this chapter has suggested that while neurochemical models that explain drug addiction primarily in terms of the brain's own rewards (rather than in terms of the properties of the drugs themselves) may provide an intuitive, or even logical, link between drug addiction and behavioural addictions, the move toward knowing behavioural addictions as neurobiological facts (and treating them as such) has not occurred simply within the realm of ideas, as a result of shifting ideas and representations. It has suggested that the technology of naltrexone (which includes not just the very pharmacological substance, but understandings about what the substance does) has played a key role in bringing about a shift in the truth status of behavioural addictions as neurological conditions. The observation that naltrexone can be 'put to work' to reduce desires for gambling, sex, etc., presumably by modulating neurochemical activity, has been used to justify classifying some behavioural compulsions as – like other addictions – diseases of the brain. Although it had been possible to think about behavioural problems as addictions for decades, it was only as an addiction medication was found to produce effects on these problems that behavioural addictions began to appear as a matter of fact. Naltrexone, within a matrix of understandings and practices, is a technology that has been deployed in the very construction a neurochemical phenomenon of behavioural addiction. Behavioural addictions have been actualized as neurological conditions by becoming the object of a neurology-targeting drug: they exist in the brain where naltrexone acts.

And thus scientists using naltrexone to control desires have been able to produce in neurochemical reality behavioural addictions which had hitherto only existed as a matter of thought.

Of course, behavioural compulsions became a very specific sort of addiction – an addiction within neuroscience styles of thought, understood in terms of a disorder of the brain's neurochemical systems of pleasure, desire, and motivation. Behavioural problems studied and treated within a framework of this particular understanding of addiction have been referred to here as pathologies of desire. This term has been necessary not only to specify the particularity of brain-centred discourses and practices, but also to highlight the ways that the reconceptualization of the problems also involved a fundamental remaking of the categories of problems and the making up of new human kinds.

This chapter has suggested that all of this could not have occurred independently of the political, material, and technological contingencies that have been involved in the production of these new neuroscientific 'facts' and new human kinds. The political economy of scientific research agendas helps determine what propositions, theories, and hunches become candidates for truth, and the fact that clinical experiments were actually done to test the efficacy of treating behavioural compulsions with naltrexone was dependent on the creation and (selective) allocations of resources of space, time, personnel, and of course money. The indirect funding of research on pathological gambling and other behavioural compulsions by parties that could potentially gain from scientific evidence that behavioural addictions exist – most notably, the gambling industry – was not suggested to determine facts or coerce scientists (there is

little evidence to suggest, for example, that such economically interested parties condone academic fraud), but the support of researchers and research projects is obviously selective, and this has implications for what sorts of truths end up being investigated, published in journals, reported in media, and is influential in shaping therapeutic and clinical standards.

The chapter began by examining some of the media reports on cutting-edge addiction neuroscience in the early 1990s, and it is thus fitting to conclude by briefly reconsidering reports on behavioural addiction that have appeared in the media in the wake of the naltrexone research that has been discussed in this chapter. In the beginning of the 1990s, we will recall that it was just becoming possible to enunciate, even if only half-seriously, the possibility of being a 'dopamine head' – a sort of person whose identity and compulsive behaviours were aptly described as a matter of brain chemistry. At this point, the desiring self had come to be, at least to a limited extent, understood as rooted in the functioning of the brain: newspapers, magazines, television programs suggested a link between the brain, pleasure, and addiction that allowed individuals to think about their pleasures as biological processes (e.g., the 'endorphin high' experienced by runners and chocolate eaters). The appearance of these sorts of scripts and descriptions has made it possible for individuals to form understandings of themselves, at least in part, as *neurochemical* subjects.

But over the last decade, as a result of developments described in this chapter, a significant transformation has occurred: whereas in the early 1990s it had become possible to *think* in terms of a neurochemically desiring self, today, with naltrexone, it has become possible to *act* upon oneself as such a subject. This shift has been

especially apparent in media reports, in which naltrexone has been widely described as a pharmacological intervention that can help with all sorts of troublesome conducts and passions under control by working on the brain. Characteristic of these reports are suggestions that naltrexone "blocks the interaction between cells and chemicals in the brain that create feelings of pleasure" (Cox 2001), and that when individuals who take such a pharmacotherapy, known to dampen the activity of the brain's dopamine system, they may "also find that the number of cigarettes they smoke decreases. Also chocolate, gambling, pot – anything with craving decreases" (Symons 1999).

Thus today, the notion of a 'dopamine head' seems a bit more substantial than a joke or suggestion. The intervention of anti-craving pharmacotherapies have made the representation of individuals as dopamine heads seem more concrete, and has contributed to the making up – and governing – of a new kind of person. Thus, naltrexone may be thought of as a *technology of the neurochemical self.* As all of our urges and desires come to be opened up to the possibility of more or less direct, neurochemical control, through an intervention on the dopamine system, thinking of our selves and our desires as rooted biomolecular processes increasingly makes sense. And a technological intervention such as naltrexone seems to be particularly influential at 'converting' subjects to a neurochemical style of thought precisely because of their material effects – their reality. Indeed, in print media, reports about naltrexone frequently include stories in which individuals who are unconvinced by expert explanations of their behavioural compulsions become convinced by the effects of naltrexone. For example, one of the newspaper stories on Kim's gambling study interviewed one of the experimental subjects who reported that naltrexone, as described to him, sounded "too good to be true." Indeed, the subject was so

unconvinced of Kim's explanations that he almost withdrew from the trial. But after deciding to remain in the study, and despite his doubts, within a week of beginning the medication, "the urges [for gambling] went away. They just stopped" (Marcotty and Lerner 2001).

Information appearing in the media about a naltrexone allows us to not only re*think* the nature of our desires, our problems, and our selves. It also presents us with new possibilities for *acting* as a neurochemical subject by telling us what we can do:

> Do you shop till you drop? Are you in financial straits because you are addicted - yes, addicted - to that rush of endorphins that comes from buying things willy-nilly, even if they only nearly fit?
>
> [...] now, help is nigh. For a drug that is used to treat alcoholics and heroin and cocaine addicts has been proven to help compulsive shoppers, too (Fitterman 2003).

Naltrexone makes treatable, as brain disorders, problems that only a couple of decades ago were not taken for granted *as* legitimate disorders. As our cravings become increasingly amenable to control through interventions on the brain, it becomes increasingly possible to think about addiction and strong desires in brain terms, and to deal with our problematic desires as matters of neurobiological fact. The following chapter examines some of the aspects of how the pharmacological regulation of desire is influencing strategies for governing problematic drinking.

CHAPTER 7: LOGICS OF NEUROCHEMICAL CONTROL

INTRODUCTION

Until the mid-1990s, naltrexone had had a relatively isolated life: somewhat useful for the treatment of opiate addictions – but not all that useful, since only the most motivated individuals seemed to obtain substantial benefits from the drug – it was of interest to basic scientists, clinical researchers, and a small number of patients; and of course, to governments who sought an addiction treatment without pleasure-producing effects. In the public sphere, however, naltrexone received limited media attention. Unpopular with most individuals in treatment, it failed to revolutionize clinical therapeutics, and was unsuccessful in replacing methadone clinics. However, when it was approved by the FDA for the treatment of alcoholism on December 30, 1994, and became the first new pharmacotherapy to be approved for the treatment of alcoholism in almost 50 years, it was celebrated not only by public health officials and treatment professionals, but also by the media. Naltrexone got a new lease on life in early 1995, rebranded (and re-patented) as ReVia by DuPont Merck, and appeared to offer the promise of improving the recovery chances of millions of alcoholics.

The chapter focuses on what new ways of knowing and governing problems related to the consumption of and desire for alcohol have been made possible in contemporary "psychopharmacological societies" (Rose 2003b) in which pharmacological preparations are regularly prescribed to patients in order to remedy a wide range of mental, behavioural, and personal problems. After a brief consideration of how it became possible to deploy a treatment, once thought to be a magic bullet for heroin addiction because of its direct and specific actions on the opiate receptors, in the treatment of alcoholism and the control of problem drinking, the chapter focuses on

elucidating some of the social and legal implications of this new means of treatment by describing the emerging logics of control and forms of governance within which naltrexone therapies are situated.

A new era of alcoholism treatment

As was discussed in Chapter 5, it was in the late 1970s and 1980s that neuroscientists began to map the neural pathways of a wide variety of commonly 'abused' drugs. As a result of these new neuroscientific endeavours, addiction came to be explained more as a result of a drug's effects on dopamine reward pathways and pleasure circuits than of any actions on a set of drug-specific receptors. As the mesolimbic dopamine system has been identified as a crucial neural pathway that induces craving and the likeliness of relapse, it has become subject to increasing direct attempts at intervention and manipulation. This falls in line with the belief, expressed in neurological and pharmacological literatures, that "understanding what neuropharmacological processes contribute to the development of substance dependence will provide the key to the development of pharmacotherapies to treat addiction" (Koob 2000: 171). Here, the assumption is that objectively gathered facts, by showing what addiction actually is (on a biomolecular level) will direct the development of addiction treatment drugs according to neurochemical principles. Current neurobiological models focus on the 'dopamine hypothesis' of addiction – that is, the hypothesis that compulsive forms of desire are due to imbalances in the brain's neurochemical reward mechanisms which operates primarily in the dopaminergic systems of the nucleus accumbens.

Proceeding from the rationalist progressionism that provides the foundation of medical education and research today – the premise that therapies should be

developed which directly target the mechanisms of disease that have been explained through scientific research – one arrives at the prediction that the most effective, and most promising, pharmacotherapies for addiction would be those which intervene directly upon dopamine activity in the brain. Since dopamine has been identified as the primary rewarder of behaviour, and over-rewards drug-taking behaviour, scientists have been investigating the possibility of treating addiction by bringing about 'functional blockade' of the 'brain-reward substrates' – that is, by raising the level of neuronal activity required for dopamine receptors to fire (Gardner and David 1999). The pharmacological means of achieving such a blockade is to introduce into the brain chemicals which, having stronger affinity with receptors than dopamine, would attach to dopamine receptors before the endogenous neurotransmitter could. A pure dopamine receptor antagonist would, upon entering the brain, selectively attach itself to dopamine receptors (thereby blocking the action of dopamine) without causing any neuronal activity whatsoever.

However, to date such pharmacological attempts to directly the drug-sensitive dopaminergic reward circuitry of the brain directly have not had a great deal of success: "Attempts to treat human drug addiction with dopamine antagonists (i.e., neuroleptics) have proven to be failures, although one should keep in mind that this class of dopamine antagonists is not highly specific for dopamine receptors and introduces significant unintended effects on brain neurotransmitters other than dopamine (e.g., epinephrine, norepinephrine, serotonin, acetylcholine, histamine)" (Gardner and David 1999: 122). The possibility, therefore, may remain that more specific antagonists, if developed, could prove effective.

Interestingly, however, it seems that at the present, pharmacotherapies are effective in inverse proportion to what might theoretically be expected: those which act upon the DA system *indirectly* are the most efficient at reducing clinical manifestations of addiction. One explanation of these circumstances (namely, the progressionist one) is that they are due mainly to a lack of sufficient theoretical and technological resources; and that increases and improvements in research will eventually lead to better understandings of addiction that will in turn result in better addiction treatments. When we can target the dopamine receptors more precisely, we will be able to treat addiction more effectively. However, there is another possible explanation: that the dopamine system is not, in and of itself, the essential biological substrate of addiction: that it has only come to be targeted by scientists because researchers happened to notice that the administration of clinically effective anti-craving treatments – treatments that produce desirable changes in affect and behaviour in patients – correlate with changes in the dopamine system.

What this suggests is a reversal of the logic of research-based pharmacotherapeutic strategies. Perhaps it is not simply that the biological truth of the disease itself dictates or directs treatment, but that treatment can help determine what truths of the disease are found. In a discussion of alcohol dependence, Littleton observes that while there is not enough information available to scientists to offer a definitive explanation of how or why some drugs appear to reduce craving, "the examination of anti-craving drugs such as acamprosate and naltrexone in models of conditioned reinforcement may be useful in understanding the neurochemical and molecular substrates of craving" (1995: 57). In neurological models, craving is being described in terms of what neurochemical alterations are brought about by drugs that reduce

either subjective reports of desire in clinical settings, or behavioural changes in laboratory experiments. The neurobiological facts of craving are those brain events that are associated with non-brain reports or observations that are supposed to represent states of desire.

This does not suggest that biological processes are unimportant in addiction neuroscience; it only qualifies the truth-status of those biological processes that have become linked to addiction by asking whether the selection of such processes can occur independently of material, historical, and other factors that are not strictly 'natural.' To think of alcoholism in terms of treatable pathologies of desire of course only became possible recently. The discovery of naltrexone's efficacy in treating alcoholism, after all, has been attributed to the 'opportunism' that has played a significant part in identifying pharmacotherapies for addiction (Altman 1996). Rather than rely on the principles of molecularly-targeted therapy development, researchers took advantage of an opportunity to pursue an end illegitimately: here was a drug that was effective in reducing craving for opiates by working on opiate receptors, why not see what happens when it is administered to alcoholics? Important to note here is that addictions that had been conceptualized as quite distinct from one another were becoming increasingly similar as the 'final reward pathway' in the dopamine system was identified. Indeed, to speak of 'different' addictions seemed more difficult, as they began to blur together: could *any* hyperactivity in the dopaminergic reward system be an addiction? Could any addiction, so defined, be treated with opiate antagonists? If so, naltrexone might provide a novel, pharmacological means of producing molecular truths about addiction. And indeed, by the early 1980s, it had

begun to be noticed that naltrexone had some clinical effectiveness in preventing relapse in alcoholics.

This was initially a surprising finding: naltrexone was, after all, known to interfere with the activation of the opiate receptors. So why would it influence addiction to ethanol? Experimentation found that the opiate system could be stimulated by ethanol, the chemical structure of which did not resemble the opiates or endorphins. So much, then, for the central role of the highly-specific receptor in understanding and treating addiction! And so much for then-current understandings of the biochemical nature of alcoholism: naltrexone, by making possible a relationship between ethanol and the endorphin system, made the disease newly intelligible, newly amenable to pharmacological intervention.

In January 1995, after naltrexone had received approval by the FDA for use in the treatment of alcoholism, both scientists and DuPont Merck representatives who were interviewed (cf. Associated Press 1995; Leary 1995) were careful to specify that the pharmacological intervention was not a complete solution to problem drinking in itself. They specified that ReVia should not be considered a 'magic bullet,' but instead should be used as an adjunct to traditional alcoholism counselling, such as group therapy or Alcoholics Anonymous. However, the same reports that contained such statements frequently also made it clear that individuals could obtain and use ReVia without committing to psychosocial therapy. For example, in a CNN interview, after he confirmed that an analogy could "absolutely" be made between a nicotine patch for smokers and naltrexone for alcoholics, Richard Fuller, of the US National Institute for Alcohol Abuse and Alcoholism was asked whether a patient

could go to a general practitioner to be prescribed ReVia. "Well, a person could do that", Fuller replied, "They could go to a doctor. It is available. However, I would discourage that at the moment [...] because, you know, the studies have all been done in conjunction with treatment programs, and we don't have the clinical experience of where a person goes to a physician" (CNN 1995).

And while pharmaceutical executives and some treatment professionals claimed that there was not enough evidence to warrant the use of ReVia as an alcoholism therapy in and of itself, such statements were balanced with – and, to an extent, contradicted by – reports of personal experiences of ReVia users. For example, a Washington Post article appearing in March 1995 opened by stating: "While others may be hesitant to dub naltrexone – a drug recently approved by the Food and Drug Administration – as a 'magic bullet' for alcoholics, Marge Catranbone [a ReVia user] has no such reluctance" (Maier 1995: Z07). The article describes the experiences of Catranbone and another ReVia user, both of whom attribute reductions in their cravings and desires for alcohol to naltrexone, rather than to therapeutic programs.

Moreover, even within reports specifying that ReVia was approved only as an adjunct to other therapies, the nature of alcoholism and addiction was primarily described in terms of brain activity, rather than as a psychological, social, or moral problem. And when the president and chief executive officer of the pharmaceutical arm of DuPont industries, Kurt M. Landgraf, announced the launch of ReVia, he emphasized the biological aspects of the condition: "The DuPont Merck Pharmaceutical Company is committed to developing solutions to critical health and social issues, and we're pleased to offer physicians and those suffering from alcohol dependence this new tool

in the fight against the disease" (cited in PR Newswire 1995). Underlying Langraf's statement and the rationale for the approval of naltrexone was the assumption that 'alcohol dependence' is, like other addictions, a disease of the brain's reward system that should be rationally treated with medications.

This new era in alcoholism treatment, as made clear by the scientists and media reports which proclaimed it (e.g., Maugh 1995), was based on neurochemical styles of thinking about addiction. Reports in the popular press typically explained how a treatment for opiate addiction could be used in treating alcoholism by describing the neurobiological underpinnings of both conditions, for example:

> In certain patients who have a deficiency of beta endorphins (opioids that have analgesic and behavioral effects), drinking alcohol seems to increase beta-endorphin activity. This increase may be responsible, in part, for heightened positive responses to alcohol and thus may contribute to a drinker's increased drinking and eventual loss of control over drinking (PR Newswire 1995).

ReVia was described as addressing a previously unmet need in alcoholism treatment, by blocking the endorphin (and, indirectly, dopamine) response to ethanol in the alcoholic's brain.

Within this context, as was made clear in media reports, providers of medication become moral pioneers of a sort, having the foresight and fairness of mind to move beyond tropes of moral weakness and sin to provision of basic biomedical care for individuals suffering from a genuine biological disorder. And Landgraf announced, in fact, that patients who could not afford the cost of ReVia (estimated to retail for about US$5 a day) would receive their pills from DuPont Merck for free (Associated Press 1995). Certainly, DuPont Merck's moral entrepreneurship may have had something to do with their corporate entrepreneurship. When the company rebranded

naltrexone as ReVia and the alcohol treatment indication was added, it expected US sales to rise to $15-25 million annually. This amount would represent sales to approximately 45-80,000 patients per year, or 1 percent of alcohol addicts and opioid addicts in the US; higher revenues were not expected because of significant market barriers, such as the reluctance of insurance providers to pay for naltrexone, and the hesitation of physicians to prescribe a drug – even a non-addiction drug – to treat addiction, and the lack of certainty that naltrexone could itself control problem drinking without psychosocial therapeutic interventions.

But what if, through a carefully planned introduction of its product, DuPont Merck could overcome some of the market barriers naltrexone was facing? What if, by specifically marketing ReVia as an 'adjunct therapy,' patient and physician expectations could be lowered, so that they were happy with even moderate levels of effectiveness? What if free supplies of ReVia could win over individuals who would otherwise not try naltrexone, and could ultimately create demands for the treatment by patients, physicians, insurers? What if neurochemical styles of thinking about alcoholism and opiate addiction could gain widespread acceptance, and it became a rather mundane idea to treat the estimated 5 to 10 million problem drinkers in the US with ReVia? What if the US government was willing to help ReVia's chances, by making economic concessions and regulatory changes that would encourage DuPont Merck to stay interested in the treatment? Couldn't sales expectations rise from $15-25 million into the hundreds of millions?

There is, in fact, considerable grounds for such suspicions that financial, rather than only moral, entrepreneurship was a motivating force behind DuPont's positioning, as

a report prepared for the US's Office of Health Policy and Department of Health and Human Services reveals in 1997: DuPont sales executives did attempt to manage provider and patient expectations by reminding the public that naltrexone was not a cure for addiction, and chose to market ReVia as a complementary, rather than stand-alone, therapy; DuPont did think strategically about how to convince healthcare providers that it was appropriate to treat alcohol and drug addiction with a pharmacotherapy; the DuPont sales force did attempt to work out ways of explaining the neurochemical mechanism of naltrexone and its benefits to a lay audience (Lewin Group 1997).

But all of this does not mean that we can explain the increasing use of naltrexone in the governance of alcoholics and alcohol-related problems in the reductive terms of commercial strategy and economic interest. DuPont did not have much interest at all in developing naltrexone, either as a treatment for opiate addiction or even for alcoholism. The company was in fact *not* developing naltrexone for either of these conditions, until it was approached by the US government – in the 1970s and again in the 1990s – and persuaded to do so. Moreover, DuPont had been provided with financial incentives by the US government to encourage the company to develop ReVia. When ReVia was approved, DuPont was granted three years of market exclusivity for the alcoholism indication by the FDA, protecting DuPont from competition from generic versions of naltrexone; and the FDA also added a novel, relaxed regulatory system for phase IV clinical trials for ReVia, allowing DuPont to make significantly fewer commitments to studying the long-term safety of naltrexone in humans than normally required for a psychotropic medication (Lewin Group 1997). And without the biopolitics of the US War on Drugs, and the formation of a

neurochemical thought style, it would be hard to imagine naltrexone emerging as an anti-craving medication at all.

Even though sales of ReVia did begin to rise from 1995 onwards, it is difficult to attribute these increases to DuPont's market strategy. In 1998, Charles O'Brien observed that:

> ReVia, used for the treatment of alcoholism, has been steadily gaining in sales over the past two years. Although the manufacturer is no longer actively promoting it (because the drug becomes generic in 1998), ReVia appears to be selling itself because of its efficacy when prescribed and the accumulation of confirmatory clinical data (O'Brien 1998: 160).

Shortly after it was approved for alcoholism treatment, ReVia was more or less abandoned by DuPont; of course, the company retained an interest in ReVia's share of the naltrexone market, but this market was dwarfed by the billion-dollar markets for top-selling pharmaceuticals. And by 2001, when DuPont Pharmaceuticals was acquired by Bristol Meyers Squibb (BMS), the total revenue for both ReVia and generic versions was approximately $22 million, with only about $6.5 million in sales of the branded product. In the same year, BMS signed over all rights to ReVia to Barr Laboratories (a firm specializing in generic versions of pharmaceuticals), and withdrew from the field of alcohol and opiate addiction pharmacotherapies (PR Newswire 2002).

If economic explanations do not appear sufficient to explain how naltrexone has come to be used in the management of alcoholism, might we be better off understanding it as O'Brien, an eminent pharmacologist at the University of Pennsylvania, does? In the quote above, O'Brien argues that naltrexone is 'selling itself' simply as a technology that has been proven effective in both clinical research and consumer

experience. But this argument suggests that 'efficacy' is a more simple matter than it in fact is: after all, as we just learned, part of DuPont's early marketing strategy was to lower expectations about what ReVia could do for addicted individuals. As Woolgar notes, "it is highly problematic to nominate one or other effect as arising from technology per se rather than from other social 'factors'" (Woolgar 1991: 34). Judgments of, and satisfaction with, naltrexone are not determined by what naltrexone 'itself' is or does; its effects and efficiency arise as a result of a variety of factors including, importantly, social ones.

While the use of naltrexone in the governance of drinking can be understood as resulting from a range of technological and epistemological factors, as well as political and economic interests, there is not much to be gained from attempting to single out any one of these as a determining cause. The chapter therefore remains deliberately ambivalent about attributing the developments described to any causal social or material forces. Rather than attempt to explain the development and use of naltrexone in terms of its sheer capacity to produce effects – either economic or therapeutic – the remainder of this chapter seeks to investigate how naltrexone, as an anti-craving therapy, is situated within – and transforming – assemblages of governance. The following sections examine how different assemblages of governance deploy naltrexone, and suggest that naltrexone is not deployed according to logics of discipline and normalization, but rather, according to a new logic of intervention and correction that might be referred to as one of 'neurochemical control.' It is further argued that these logics do not necessarily produce the same repressive and dominating effects associated with 'social control.'

SOCIETIES OF (NEUROCHEMICAL) CONTROL

The term "social control" was originally coined in 1901 by the American sociologist Edward Ross to describe a democratic and liberal alternative to forms of coercion and compulsion that depend on the use of force. Ross felt that social organization and coordination was best achieved when individuals made choices about their behaviour on the basis of belief systems and norms rather than specific, codified laws. He believed that such an internalization of belief systems would guide behaviour and produce in individuals mechanisms of self-regulation that could assure order more effectively than legal regulation (Ross 1969 [1901]). Since Ross' time, of course, sociologists have tended to take a less enthusiastic view towards social control, and have questioned the extent to which such liberal approaches to government create more free, or less intensely governed, subjects. Often, of course, social control supplements, rather than replaces, legal and punitive means of maintaining social order (cf. Cohen 1984). But even in the absence of repressive laws and formal systems of punishment, the freedom of 'socially controlled' citizens becomes entangled with institutional and managerial efforts to produce individuals capable of acting responsibly and autonomously (Rose 1999b).

Although it was not framed in such terms, Foucault's (1979) conceptualization of discipline has become foundational to thinking about forms of social control and regulation that extend beyond traditional legal rule. In 'disciplinary societies,' Foucault notes, power is no longer based primarily on the rules, rights, and laws posited by a juridical model of sovereign power; it is based on the deployment of authoritative and 'enlightened' understandings and practices that govern citizens in their own, and their society's, interests. Thus, subjects must not only be compelled to act according to particular rules, but must also be taught to understand and internalize

those rules. Institutions such as schools, hospitals, prisons, and psychiatric wards achieve their ends not through direct coercion, but rather by moulding individuals into particular types of subjects through training, instruction, and monitoring. Projects of social regulation thus become a matter of governing through the production and enforcement of particular forms of individuality and selfhood.

While Foucault's historical studies have had a tremendous impact on sociological analyses of contemporary societies, it has been suggested that Foucault's work in the 1960s and 1970s revealed the inner logics of disciplinary societies just as such societies were becoming obsolete. In a brief essay on 'societies of control,' Gilles Deleuze (1992), for example, suggests that contemporary forms of government are becoming less disciplinary – that is, less reliant on institutional training and supervision, and on the ordering of time, space, and conduct within isolated enclosures. Deleuze suggests that in emerging societies of control, government is achieved through increasingly constant surveillance and precise forms of intervention that are accomplished with technological modes of control. These technological modes do not merely oversee and instruct (except in the vestiges of disciplinary forms of power), but rather intervene on the very essence of subjects: their everyday social networks, the geographies of their personal lives, and the composition of their bodies.

What we are seeing, Deleuze suggests, is in part a shift from 'moulding individuals' to 'modulating dividuals.' Moulding individuals involves attempts to govern the subject as an indivisible whole – e.g., by shaping the unified soul, psyche, body, or person into requisite or desired forms. This was a fundamental logic of power in disciplinary societies, relying heavily on 'the social' – on institutions and

bureaucracies, on socialization and the transmission of knowledge, on welfare policies and social services. In contrast, in societies of control, modulating dividuals involves governing by constantly monitoring and correcting only specific *parts* of (fragmented) subjects that have been associated with deviance or risk. Such interventions rely on technologies that can precisely target a problematic site or process with little or no collateral effects on the subject as a whole.

In his brief speculations on the general contours of societies of control, Deleuze provides a picture of the immanent future which is not only threatening, but also largely debatable – painted as it is with the broad brushstrokes of generalization and categorical assertion. But leaving aside Deleuze's predictions about the future, his essay provides insight into changing forms of governance, and offers a novel perspective for thinking about the "socio-technological study of the mechanisms of control" which are beginning to restructure fields of power in contemporary societies.[14] Deleuze's notion of societies of control can provide particularly valuable insights on novel political and social aspects of developments in psychopharmacological technoscience, which may help us to understand the place of naltrexone in contemporary society.

Deleuze only makes passing reference to the "extraordinary pharmaceutical productions" of control societies in his essay, but pharmaceuticals – and particularly psychopharmacological technologies such as naltrexone – deserve more careful

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[14] Although he sets up a dichotomy between control societies and (Foucault's notion of) disciplinary societies, this distinction is best thought of in terms of an ideal typology rather than an empirical description. Disciplinary logics of social regulation remain, of course, powerful and significant factors in the shaping of individuality and the government of contemporary societies; and indeed, it might be the case that 'pure' forms of technological control are still the exception rather than the rule.

consideration than they receive. Indeed, products of contemporary biological psychiatry such as naltrexone might be considered as technologies of control par excellence. For example, used as a neurochemical intervention which can regulate neurological mechanisms in order to prevent mind/brain states that increase the risk of problematic conduct, naltrexone allows for a form of control that extends into and incorporates the very material of the human body. Its ability to regulate the brain allows naltrexone to target and bring under control problematic desires in such a way that reduces the chance of dangerous, illegal, or unwanted actions.

In the sections that follow, Deleuze's sweeping philosophical (and arguably paranoid) description of societies of control will be used to analyze the deployment of naltrexone as a technology of control within two dramatically different regulatory apparatuses: a probation program for individuals convicted of drunkenness-related offences, and a brain-based alcoholism treatment program. The more concrete analysis of technologies of control that these case studies offer reveal that the same technology of control (naltrexone) can produce very different effects in different assemblages of governance.

Drug courts and penal psychopharmacology

Following the FDA approval of ReVia – the branded version of naltrexone for use in the treatment of alcoholism – DuPont became a major corporate sponsor of the National Association of Drug Court Professionals (NADCP), a non-profit organization involved in promoting 'drug courts' in the US. A drug court is a special court that handles legal cases that involve substance-using offenders, and diverts individuals away from the prison system to probation and 'community care.' Such courts are increasingly popular, in part because of the crises facing the American

penal system. As the NADCP explains: "With Three-Strikes-You're-Out statutes proliferating and long-term incarceration for serious offenders increasing, drug court programs are needed to free up limited jail space for serious criminals" (NADCP n.d.-b). Drug courts are promoted on the basis of their ability to reduce criminal recidivism – but also on the basis of their cost-effectiveness.

Drug courts are an interesting development in contemporary legal practices for a number of reasons, including, as a result of attempts to reduce costs, the reconfiguration of the boundaries between the market and the state in the provision of justice. The NADCP actively solicits sponsorship from corporations, promoting drug courts as a means for a range of industries to obtain access to an emerging market for products and services:

> As a corporate member, your company gains visibility within the drug court community and access to a broad range of professionals who can help make your goals a reality. This exposure clearly establishes your company's role in the drug court arena. And while corporate membership is a powerful tool for getting your message to the right people, it is also a means of gathering pertinent information for your own use. Corporate membership offers access to a nationwide network of professionals ready to explore your products and services that meet their need.
>
> No other organization can provide you with such direct access to the drug court field. With nearly 1100 drug courts in operation and over 400 more in the implementation phase, the time is right to position your company to be seen by professionals in one of the fastest growing fields in criminal justice (NADCP n.d.-a).

Corporate members receive a number of benefits and considerations from the NADCP, including exclusive meetings with the organization's Board of Directors. Thus, members obtain access to the professionals in the legal system – predominantly lawyers and judges – who make decisions about what treatments are sentenced to offenders, and what technologies and services are declared useful or necessary in the

provision of criminal justice. And while the market to which the NADCP provides access is already considerable – over 400 000 cases have been processed by US drug courts – this number is expected to expand dramatically as the number of jurisdictions establishing such courts grows, and as the drug court model is increasingly coming to be used for dealing with less serious (and more common) offences such as drunk-driving.

ReVia's (and DuPont Pharmaceutical's) entry into the drug court arena provides an interesting case study of the new sort of regulatory assemblage that is emerging in advanced capitalist societies of control, which recombines and mutates the formerly distinct elements of corporate, medical, and legal power. The goal here, however, is not to examine the political economy of what might be referred to as a 'penal-bioindustrial complex' – that is, a coalition of judicial authorities and pharmaceutical industrialists who both demand and supply medical technologies of control. Instead, it is to examine the strategies and logics of control that are emerging as drug courts begin to use anti-craving medications in the management of problem drinkers.

Drug courts provide a potential for, and are indeed posited upon, liberal approaches to dealing with crime. As the NADCP president indicates in a letter on the organization's website, the NADCP advocates a "therapeutic approach to criminal justice where offenders are required to undergo drug treatment". The NADCP philosophy is that "coerced treatment works" (Freeman-Wilson n.d.) – but this coercion does not occur within a total institution which prevents the offender from moving about relatively freely in society. Instead, coercion often involves a commitment on behalf of the convicted offender to submit to relatively mundane, but

technically precise interventions that target only particular, dividual elements of the subject. For example, many drug courts use urinalysis tests to obtain information about drug-using behaviour. Through urinalysis, such conduct – which would violate the terms of probation and possibly lead to carceration – can be monitored not only at a distance, but at a momentary glance. Information about drug use over several days can be collected in the time it takes to collect a sample; thus, surveillance becomes intermittent in practice, but continuous in its effects. But our focus is not on the monitoring of urine; it is on the modulation of brain chemistry with the use of brain-targeting medication, within the subspecies of drug courts alluded to above: courts which focus specifically on processing cases of individuals who have been convicted of multiple offences of 'driving under the influence' (DUI) of alcohol. We will consider one of the very first so-called 'DUI courts' to have been established in the US, which is today promoted as a model for other courts.

The Butte County ReVia® Project[15]

Butte County, California, is situated about 70 miles north of the state capitol, Sacramento. With a population of approximately 200,000, and situated in a predominantly rural area, it is in many ways a peaceful, law-abiding region. However, the county is also home to the Chico campus of California State University, which "has struggled for years with the reputation of being one of the top 10 party schools in the nation. Alcohol use plays a prominent role in the local culture", and both arrests and alcohol related fatalities are deemed by authorities to be unacceptably

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[15] All information about Butte County and the Butte County ReVia Project has been taken from "Butte County ReVia Project", a report by Butte County officials which has been made available online by the American Council on Alcoholism to serve as a resource for information about DUI courts. See **Stevens, D., Harberts, H., Pfeifer, J. E. and Redmond, I.** n.d. 'Butte County ReVia® Project', Accessed 2004: American Council on Alcoholism. Available at http://www.aca-usa.org/reviaproject.htm.

high. While the California Highway Patrol and local police have coordinated efforts to reduce alcohol-related problems, and have met some success, "there remains a core group of addicted drivers who continue to pose a grave danger to the public, and occupy a significant portion of the community resources through health care, emergency services, police, court, jail and probation criminal justice costs" (Stevens, et al. n.d.).

In 1996, acknowledging the "generous assistance" of DuPont Pharmaceuticals, Butte County created the ReVia Project for such offenders. As a report on the Project's first years of operation explains:

> Butte County was one of three courts in the United States who directed the use of ReVia (generic name: naltrexone) as part of a court ordered treatment model. The model was designed as an expedited case processing system, where identified alcoholics would be moved quickly into the treatment process. Key to this treatment process was ingestion of ReVia (naltrexone) (Stevens, et al. n.d.).

Based on the understanding that neurobiological factors often prevent traditional probation programs[16] oriented toward behavioural modification from being effective, and with the assumption that ReVia acts on the brain pathways affected by alcohol, the Butte County DUI Court created a 'ReVia Track' that allows repeat offenders to avoid imprisonment on the condition that they take naltrexone daily (initially, for a minimum of 90 days).

As part of a program of juridically-administered therapy which might be referred to as a form of 'penal psychopharmacology,' the ReVia Project subtly alters traditional relationships between legal authority, medical care, and the doctor-patient relationship.

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[16] 'Program,' rather than 'programme,' is used here not to single out (North) American projects, but merely reflects the standard Canadian spellings which are used throughout this thesis.

Butte County has developed a "ReVia Project Protocol" establishing the steps involved in the naltrexone-based program of therapeutic justice, which are to be applied to "[e]ach defendant assigned to ingest ReVia through the Court's DuPont ReVia Project". Following the initial stipulation that the individual is placed on formal, supervised probation, the protocol requires that s/he be ordered by the Court to "[i]mmediately report to a physician for an examination and issuance of a prescription for ReVia (sometimes referred to as Naltrexone)" (Stevens, et al. n.d.).

It should be noted that the protocol does not require a *medical* judgment of the utility and/or advisability of the use of naltrexone in the treatment of the patient. The individual does not appear to have recourse to a medical opinion at all (e.g., a physician's objection to a judge's verdict). The physician is not called upon by the Protocol to consider the subject as an individual, but only to provide a prescription on the basis of a legal code (from the judge: *this offender is on the ReVia track, and therefore requires a prescription*). There is no medical subject to be known (or diagnosed) under a scrutinizing gaze, because the relevant information and path of action has already been pronounced by verdict. Indeed, it seems that the only reason that medical authorities are enlisted in the ReVia project is because the administration of naltrexone exceeds the authority of the courts: "Because of the unique requirements of the medical protocol, we turned to the local community" of pharmacists and physicians. This was necessary because (despite the fact that the Court's Protocol requires the administration of naltrexone in each and every case) "the Court cannot accept and distribute a prescription drug" (Stevens, et al. n.d.).

In this alliance, we witness something like the "new medicine 'without doctor or patient'" suggested by Deleuze, which "singles out potential sick people and subjects at risk" (Deleuze 1992). Naltrexone is essentially administered to a juridical, rather than medical, subject – i.e., an offender, rather than a patient. Of course, there has long been a close relationship between legal and medical authority, and between punishment and therapy. However, within this new formation, there are important novelties. Naltrexone is administered on the basis of a risk of future offence (to those who are perceived to pose a danger to social or legal order), rather than on the basis of cure (to those who are endangered by disease or illness). Moreover, this subject is also treated as a consumer, who must be sold on their own punishment and treatment. As the Butte County report notes:

> ReVia can *seem* expensive: up to $535.00 per month. However, compared to the cost of alcohol, or a jail bed, it is quite inexpensive. Some defendants have had assistance with the costs from their families. Also, purchasing a supply one-week at a time assists with "sticker shock". The cost of a one-month supply *will* frighten many clients. It is easier to have them obtain a smaller amount (Stevens, et al. n.d.).

The ReVia Project authorities explain that naltrexone is a relative bargain, and provide strategies for making the subject a more willing consumer.

A review by the Butte County court system found that the statistics obtained after three years of the Project's operation demonstrated "one simple and sterling clear fact: ReVia *works* on this group of offenders far better than any other supervision model" (Stevens, et al. n.d.). Within the penal system of Butte Country, naltrexone demonstrates how the move towards a 'therapeutic approach to criminal justice' that the NADCP are focused on bringing about also relates to technoscientific strategies of control: the modulation of the offender's reward system with naltrexone allows for

prison time to be substituted with penalties that do not rely on the structuring of the daily routines, the panoptic supervision of conduct, or the confinement of bodies within tight enclosures. The offender is not subjected to a program of strict discipline that moulds a law-abiding citizen; instead, she enters into a program of somatic regulation, in which neurochemical modulation reduces the likelihood of uncitizenly conduct. These new juridical strategies, which appear to be prototypical technologies of neoliberal governance, achieve legal order in a way that maximizes the liberty of offenders at the same time as they reduce costs.

The Butte County ReVia Project is of interest – and is being discussed here in Deleuzian terms – not because such drug courts are the 'new monsters' of control (cf. Deleuze 1992) that threaten to enslave individuals, but because they embody changes in the way that addiction, desire, behaviour, and justice are being reformulated along neurochemical lines, are being pursued in new technological and practical forms, and are transforming the ways that we see ourselves – as well as the selves of criminal, poor, and deviant 'Others.' The judgment made when offenders are assigned to the ReVia Track is in many ways a neurochemical one, classifying an offender as a particular neurobiological kind of subject, the essence of which can be brought under control with targeted intervention, marking out the material to be controlled. And it is precisely because the ReVia Project targets particular neurotypes, rather than specific forms of behaviour, that it appears possible to extend the ReVia protocol to a range of applications. As the Butte County Court reports:

> It is believed that the utilization of ReVia (naltrexone) will be useful in a number of other contexts [other than impaired driving offences]. The Court also orders ReVia treatment in domestic violence cases. It has obvious applications in the public inebriate,

> self-medicating mentally ill and homeless populations which plague most American cities (Stevens, et al. n.d.).

For a range of conditions, it seems, there arises the possibility of using the new strategies of penal psychopharmacology in which a verdict of neurological dysregulation leads to sentencing the ReVia Track and the enforcement of the naltrexone Protocol. As naltrexone exerts its control – which is both neurochemical and legal – the brain is regulated, behaviour modified, and risk of reoffence diminished.

TECHNOLOGIES OF SELF/CONTROL

Deleuze provides a useful point of departure for thinking about how naltrexone may be considered as part of a shift from disciplinary societies to societies of control. As the Butte Country ReVia Project demonstrates, logics of control – in this case, neurochemical control – do appear to be increasingly ordering lives and governing problems not by enveloping individuals in institutions, but by targeting freely-moving dividualized subjects. However, an analysis of naltrexone as a technology of control would be extremely simplistic if one relied solely on Deleuze's account of societies of control. For, despite the fact that he develops his analysis of control in relation to Foucault's concept of discipline – and despite the fact that Foucault describes disciplinary power as essentially related to the knowledges that it relies upon and the subjects it produces – Deleuze focuses on the power of technological control in abstraction from the conditions of its development and the spheres of its application.

Because preceding material in this chapter (and indeed much of this thesis) has examined how the development of naltrexone has a complex epistemological (and indeed, ontological) history that is bound inextricably with social and political factors,

there is little need to address these issues here. The remainder of this chapter thus engages with the problem of the subject of power in societies of control – not abstractly, but in relation to the use of naltrexone within strategies for managing problem drinkers. Since Deleuze constructs his ideas about control in relation to the work of Foucault, it is useful to draw upon the latter for this analysis.

A fundamental shortcoming of Deleuze's analysis is that it only considers technologies of control as acting upon, rather than affecting – and interacting with – subjects. Indeed, his 'dividuals' appear as more or less abstract *objects* – things at which specific actions and technologies are directed – that have no relation to reflexive, reactive subjects. It is perhaps true that the notion of individuality is peculiar to particular times, places, and societies (Strathern 1988); but individuality exists all the same within the contemporary societies of the west about which Deleuze writes. Technologies of control may be directed at a dividual object, but the strategies of government within which they are deployed are ultimately concerned with the actions, choices, and beliefs of subjects, more than with bits of material or information.

Deleuze's lack of discussion about the subject of power contrasts sharply with the work of Foucault, who considered an account of the subject essential to analyses of disciplinary societies. For Foucault, disciplinary societies are also, essentially, *liberal* societies, in which citizens have a say in how they are governed and indeed play a role in their own governance. It is precisely because disciplinary power operates not through overt coercion, but through systems of liberal management that seek to 'help' (normalize / heal / teach / protect) individuals, that it is able to enlist individuals in

systems of self-rule. Discourses of health, welfare, and other benevolent programs intersect with individuals at the level of everyday, local practice, and, by inserting their ordering capacities into independently operating sites, draw individuals into power situations of which they themselves are frequently bearers. Disciplinary power, then, creates a regulatory complex in which individuals, who are in part the effects of a power that constitutes them as free subjects, also participate in the constitution of their own subjectivity.

In contrast, Deleuze's political conception of control seems to be characterized by the development of radically more efficient means of power which 'enslaves' individuals and unilaterally brings their conduct in line with the programmatic aspirations of some (unidentified) agent. But the crushing, or destruction, of subjectivity that is implied by Deleuze's account is at odds with the politics of 'advanced liberal' societies (cf. Rose 1999b), in which mechanisms of power are largely conditioned upon administering and enhancing freedom rather than repressing it, and which function by discovering, understanding, and managing the hidden and mysterious aspects of human existence in order to bring about personal and social well-being. While Deleuze does seem to suggest that there are anti-liberal tendencies in technologies of control, it does not seem the case that in the present day the logics of control are sheer external domination.

The issue of subjectivity thus remains important to understanding the general logics of control as well as the particularities of different technologies of control. Although technologies of control might most easily be identified within strategies of domination, we also need to investigate how these same technologies, and the

understandings and perceptions that are associated with them, may relate to ambitions and projects of self-government, and constitute what Foucault refers to as 'technologies of the self.' Foucault uses the latter term to specify the means through which subjects constitute and fashion themselves as subjects within particular systems of power. Technologies of the self "permit individuals to effect by their own means, or with the help of others, a certain number of operations on their own bodies and souls, thoughts, conduct, and way of being, so as to transform themselves in order to attain a certain state of happiness, purity, wisdom, perfection, or immortality" (Foucault 1997b: 225). We can thus understand technologies of the self as assemblages of understandings and practices that allow individuals to delimit those parts of themselves that form the object of a moral or ethical practice that operates even in the absence of (external) control.

Just as disciplinary power and knowledge are involved in the production of subjectivities, we need to investigate the relationship between the knowledges and powers of control on the one hand, and their effects of the subjective experiences of selfhood and identity on the other. But because the logics of control are different from the logics of discipline, we need to investigate the extent to which pharmaceutical technologies such as naltrexone allow individuals to extend the principles of self-care and self-improvement in ways that are qualitatively different from those associated with the sorts of traditional / spiritual / holistic techniques with which Foucault's studies concern themselves. Foucault identifies two forms of self care, or ethics: in ancient times, the care of the self was a matter of self-mastery (strengthening those parts of the self that could thwart desire), but through the Christian era it increasingly became a matter of learning about and shaping one's own

inner character (Foucault 1990a). Today, we might ask whether the care of the self is still best described in these terms, or whether, to an unprecedented extent, it has become a matter of mastering the matter / material of the body (though perhaps, still, the ultimate end would be to shape the self – it may merely be that the relation between the 'inner' self and the somatic self virtually disappears). If so, our new means of acting upon the body might constitute something like a third epoch of ethics (a corporeal ethics, an ethics of vitality, a biological morality...). Within this new formation of ethics, the new models of the biomolecular brain sciences, along with pharmaceutical interventions that take the brain as the object of intervention, seem to provide ways for individuals to think of themselves and to regulate their thought and conduct in terms of neurochemical selfhood.

Naltrexone may target the brain as a dividual object, but what of its effects on individual subjects? The interventions into the brain are not made in order to change the actual state of the brain, but rather to influence the thoughts of behaviours of that subject. Thus, the brain may be an object that is targeted by naltrexone, but is also still a part of the self – and part of the subject that comes under control. So the subject of government does not disappear in societies of control; rather, the subject, once known almost exclusively in terms of wholes (e.g., the soul, the spirit, the psyche, the body) becomes reformulated, and knowable and governable as a new hybrid of object (dividual) and subject (individual).

Thus, it is important to ask: how do technologies of control also function as technologies of the self – or as technologies of self-control? What is the role of naltrexone – the pharmaceutical technology, of course, but also the decades of drug

politics and research into addiction science that have made naltrexone possible and intelligible in particular ways – in the care of the self? As we will now turn to discuss, at the same time that naltrexone (certainly, one of the extraordinary pharmaceutical preparations of control) has been deployed within the penal-therapeutic formations of the criminal justice system, it has also formed the basis of a range of other strategies for managing behaviour and desire which do not primarily involve punishment or the crushing of subjectivity. This discussion suggests that naltrexone, as it makes possible new experiences and new means of governing conduct that are engaged in on a profoundly ethical and personal basis, allows certain types of subjectivity (which are perhaps novel) to flourish. Thus, the logics of control may be a logic of *self*-control: intense, constant technological regulation initiated by an individual him- or herself, in the pursuit of his or her own goals and values. One of the most clear illustrations of naltrexone's ability to be used as a technology of self/control is its deployment within treatments for excessive drinking that are sought by patients themselves.

Alcohol, naltrexone, and ContrAl[17]

In the first double-blind, placebo-controlled trials of naltrexone for the treatment of alcoholism (O'Malley, et al. 1992; Volpicelli, et al. 1992), in the FDA specifications for naltrexone's approval, and in the ReVia literature produced by DuPont, the therapeutic potential of naltrexone was described as limited – or rather, as uncertain – since naltrexone had only been studied as an 'adjunct' to social and psychological therapies (such as psychoanalysis and self-help programs). In such a context, naltrexone might have resembled a technology of pharmacotherapeutic discipline – working with other disciplinary apparatuses in order to remould the alcoholic into an

[17] All unattributed quotations about the ContrAl program in this and following sections are taken from (the English version of) the ContrAl website. See www.contral.com/english.html.

abstinent individual living a new way of life – rather than as a technology of neurochemical control that operated according to logics of dividual control and constant, targeted regulation.

However, as indicated at the outset of this chapter, at the time of ReVia's introduction there was considerable debate about what naltrexone could do for alcoholics. Even before ReVia was approved by the FDA, the proper role of naltrexone was disputed within the community of medical experts; initial formulations began to be challenged, reinterpreted, and reworked almost from the very moment they were enunciated. Although naltrexone was approved for use within an older, disciplinary logic of alcoholism treatment, it has nevertheless come to be deployed within a newer logic of (self) control.[18] This section investigates a number of shifts that are taking place that constitute the basis of this redeployment by examining in detail one of the treatment modalities where such changes are most apparent: namely, the 'Sinclair method.' This program, developed by the Finnish alcoholism researcher in the 1990s, was one of the first systematic naltrexone-based treatments to be clinically implemented. Although the Sinclair method may be used (and adapted) by clinicians the world over, the focus here will be on the deployment of the Sinclair method within the proprietary chain of "ContrAl Clinics" that Sinclair developed, and which now operate not only in Sinclair's native Finland, but also in the UK and the rest of Europe, as well as North America, Russia, and the middle east.

[18] That naltrexone could be mobilized both as a technology of discipline *and* a technology of control should not be entirely surprising – since of course, the very substance of the technology itself does not absolutely determine its effects or actions.

From the outset, it must be noted that the shifts under investigation can only be described as partial and incomplete. As we will see, even within ContrAl's own literature, there are contradictions and tensions between the logics of discipline and the logics of control. And while Sinclair is one of the most renowned experts on the use of naltrexone in alcoholism, and the Sinclair method is one of the famous and widely accepted modalities for such use, there remain important disputes and differences of opinion amongst alcohol researchers and clinicians. However, in keeping with the rest of this thesis, the goal here is to highlight important shifts in addiction thought and treatment that relate to contemporary molecular neuroscience and psychopharmacology, rather than to offer a comprehensive overview of the field of addiction (or alcohol) studies. More specifically, the goal here is to highlight first, how the deployment of naltrexone has shifted towards logics of control; and second, the models for understanding and acting upon the self that such logics of control are bringing about.

Changing logics of treatment

Naltrexone was approved by the FDA to prevent 'slips' in abstinence from leading to a full-blown relapse. As naltrexone prevents alcohol from exciting the neurological reward system, drinking becomes less pleasurable, less rewarding, and thus easier to resist. Since naltrexone blocked the euphoric effects of alcohol, it was reasoned that an individual who took an initial drink while on naltrexone would receive no pleasure from the alcohol consumed, and would therefore have no reason to take a second drink. This was, of course, the same logic initially used to explain the utility of naltrexone in the treatment of opiate addiction: the blocking of pleasure and protection of the addict. But just as naltrexone came to be reconceptualized as an

anti-craving therapy for the opiates, a similar process occurred in re-interpreting the actions and uses of naltrexone in relation to alcohol.

John Sinclair was one of the first researchers to argue that naltrexone was most useful not merely to assist in the maintenance of abstinence (i.e., prevent intoxication and slippage in detoxified alcoholics), but as therapy that extinguishes the conditioned responses to alcohol in individuals while they continued to drink (Sinclair 1990). Sinclair's approach, and the theoretical foundation of ContrAl's program, draws upon Wikler's (e.g., 1976) theory of addiction and relapse, which was based on the concept of extinction of learned responses. The ContrAl program explicitly frames the problem of drinking as a biochemical one that relates to how behaviours are reinforced:

> The craving and the continual thinking about alcohol are both gradually learned. This learning involves the opiate receptors where morphine acts. Alcohol causes some neurons to release a substance like morphine that fits into opiate receptors, reinforcing the alcohol drinking. This tends to cause more drinking, causing more reinforcement, more drinking, etc., in a vicious circle.
>
> Some people, due to genetics and experience, have alcohol drinking grow so strong that it dominates their lives. They cannot control their drinking, nor can anyone else.

And the solution that ContrAl provides is, unsurprisingly, likewise framed in biochemical extinction of learned responses. As the website explains, "pharmacological extinction with naltrexone reverses the process":

> Learned responses can be removed: they are extinguished if made while reinforcement is blocked. Extinction of alcohol drinking progressively weakens the craving for alcohol, decreases incessant thinking about alcohol, and reduces the chances of relapse. Used correctly, pharmacological extinction, in effect, reaches into the nervous system and safely removes the unwanted, life-threatening behaviour that has grown beyond control.

With naltrexone, cravings for, constant thoughts about, and the actual consumption of alcohol are all brought *under control.* As the neurochemically reinforced responses to alcohol are extinguished, craving diminishes, and problematic behaviour becomes manageable.

This novel logic of control has brought about a de-emphasis on the role of will-power and motivation in the treatment of alcoholism and problem drinking. Initially, the therapeutic success of naltrexone was thought to depend heavily on goal-orientation and motivation. A willing therapeutic subject was required, because patients could easily stop taking naltrexone if they were intent on experiencing intoxication. Naltrexone was in fact thought to be of use primarily in only the most highly motivated patients, because it was presumed that naltrexone merely blocked pleasure. Since desires for drinking persisted (but could simply not be gratified) while on naltrexone, individuals were thought to need a constant motivation to take a medication that would prevent them from achieving intoxication *despite* their desires. There had to be a constant will to take naltrexone and to resist the desire to drink.

Today, however, naltrexone is understood to act on desire itself, and to mitigate what ContrAl literature refers to as the 'internal cause' of pathological drinking. Once craving is brought under control with naltrexone, the desire to drink is regulated, and motivation becomes less important factor in therapeutic success. One does not have to fight one's desires with one's will, for desires can be been tamed by naltrexone. The decision to take naltrexone can hence be made with relative ease, since there is little or no desire against which a patient must contend.

The shift from using naltrexone as a means of maintaining abstinence to a means of modulating desire was accompanied by a shift from a logics of exclusion or interdiction of alcohol to a logic of incorporation and governance. Originally approved as a means of helping establish long-lasting sobriety, naltrexone was understood to be of use in helping banishing alcohol from the life of an individual within all-or-nothing regimes of abstinence. At a particular point, one had to move from drinking to not-drinking – the only two choices, and classifications of conduct, available. In such a context, naltrexone was deployed as a means of protecting the individual from alcohol, and keeping her alcohol-free.

But within ContrAl's program, drinking is not banished; instead, it is included within the sphere of thought and conduct that needs to be managed. Ethanol need not be forbidden, since desire for it can be brought under precise control. Because naltrexone prevents reinforcement of drinking by blocking the opiate receptors, "the urge to drink gradually is weakened, and alcohol becomes an insignificant matter." As alcohol becomes an 'insignificant matter,' a single drink, or even more than one drink, is not an alarming event that signals the failure of treatment. Unlike strict regimes of abstinence that do not deal with the internal, biological causes of drinking and simply impose external barriers to alcohol consumption, the ContrAl program claims to treat the craving for alcohol. As craving diminishes, so too does the consumption of alcohol; thus, drinking is not avoided, but brought under pharmacological control. And indeed, Sinclair (somewhat controversially) suggests that for some patients, controlled drinking – rather than abstinence – is a valid objective of treatment (Sinclair 2001).

Because naltrexone is understood as a means of controlling drinking, rather than eliminating it, one does not have to make the decision that one is an alcoholic who need to give up drinking completely to be a candidate for naltrexone therapy. While naltrexone was approved with the treatment of alcoholism in mind, it has increasingly come to be used not only to treat particular types of pathological individuals (i.e., alcoholics), but to manage particular problems that even non-alcoholics may encounter. The ContrAl program describes its program as a treatment for "excessive drinking, alcohol abuse and alcoholism". One's whole life and soul do not have to be facing ruin before one seeks out treatment; one only needs to identify a problem in one's life, and associate it with a pathological brain function, in order to benefit from naltrexone's logic of targeted control. Naltrexone can be put to work in governing the desire for alcohol and the harmful and compulsive behaviours that result from seeking to fulfil such desires not only in alcoholics but also in problem drinkers, drinkers with alcohol-related problems, and indeed, drinkers who merely want to *prevent* alcohol-related problems or inconveniences from arising.

Thus, while originally it may have been true that the target of naltrexone could be described as "that venerable social and psychic problem associated with excess, immoderate, uncivilized drinking: alcoholism" (Valverde 2003: 440), today naltrexone does not just target an individual dominated by or absorbed in a classification of a pathological kind, but targets specific problems of affect and conduct. This suggests, perhaps, that we are moving away from thinking and acting in terms of pathological kinds – treating only those who have been judged or diagnosed as alcoholics – and toward the treatment of pathological desires (which does not require judgments to be made about the person experiencing them). But

while individuals may increasingly be able to escape the stigmatizing subjectivity involved in a diagnosis of alcoholism that binds them, in more or less irreversible ways, to lifelong projects of self-discipline, we are not necessarily seeing an end to the governance of problem drinkers. Indeed, interventions into one's neurochemistry seem to provide a basis for *expanding* the possibilities for governing individual drinkers. Naltrexone is not just for managing the kinds of subjects identified as alcoholics, but for managing an increasingly broad range of desiring subjects that includes more flexible and less stigmatized categories of problems than alcoholism. Thus, a waning of pathological kinds is accompanied by a multiplication of degrees of pathological desire amendable to intervention.

The expansion of eligibility for naltrexone treatment is also accompanied by an extension of the duration of treatment. Naltrexone was originally formulated as an intervention of limited duration – a medication that would help individuals maintain abstinence for a few months as they learned to live their lives in a new way. In this formulation, naltrexone's role was to prevent relapses during the initial and most difficult period of abstinence until the individual was more capable of remaining substance-free without the (continued) use of pharmacotherapies. Today, however, it is increasingly suggested that, rather than being used within a limited time-frame as a means of retraining an addict for an abstinent future, naltrexone is perhaps best used within an indefinite program of 'controlled drinking.' Sinclair argues that "There should be no end to naltrexone treatment [...] Patients are advised to always carry a pill with them – for the rest of their lives – but to take it only on those occasions when they are likely to drink" (Sinclair 2001: 7). The idea here is not that individuals

should take naltrexone constantly, for example on a regular daily or weekly basis; rather, it is that they should always be ready to take naltrexone.

And here we come to a contradiction in the ContrAl literature and Sinclair's work. Sinclair claims that the ContrAl treatment is in fact "cure, rather than a treatment" (cited in Agence France Presse 1996) on the grounds that naltrexone gradually extinguishes the pathological neurochemical rewards that compel individuals to drink excessively. Since the brain is no longer affected by alcohol consumption, it no longer provides the incentive to drink: neurochemical rewards to alcohol-related cues that cause craving. Whereas naltrexone was previously used to ensure abstinence by protecting the vulnerable brain from external triggers (i.e., the actions of alcohol) that lead to relapse, Sinclair explains that within his treatment modality, naltrexone is deployed in such a way that it neutralizes the fundamental, 'internal cause' of alcoholism, which is located in the brain. And by changing the way that the brain works, naltrexone, as administered in Sinclair's method, "does indeed change the person, unlike other treatments" (Sinclair, cited in Agence France Presse 1996). And yet, within regimes of controlled drinking, there is no 'starting over' (as, for example, there is in Alcoholics Anonymous, when after a relapse one begins once again at zero to calculate one's period of sobriety), precisely because one is never finished with drinking – or with naltrexone therapy. Thus, if targeted medication forms part of a 'cure, rather than a treatment,' it seems to be a cure of indefinite length, with limitless postponements.

Regimes of self-care

When naltrexone was first approved for use in the treatment of alcoholism, ReVia promotional tracts emphasised the fact that the drug would do no good on its own:

"After all, quitting drinking is more than just putting down a drink. It's learning a new way to live your life" (cited in Continelli 1997: 2). Initially, naltrexone's place in treatment did not appear very far removed from the approaches of Alcoholics Anonymous and similar groups, which insist that alcoholics must undergo a profound change in their approaches to life in order to abstain from alcohol permanently. In contrast, the ContrAl program promotes itself on the basis that it "does not interfere with everyday life but allows people to live normally while undergoing the program."

In this and other ways, the suggestion seems to be that naltrexone and ContrAl provide a less intensive form of governance over drinking. Within the context of Sinclair's program, for example, internal, pharmacological mechanisms of control replace, or at least minimize, the need for the intense forms of introspection and self-monitoring that constitute the basis of disciplinary power – which Sinclair and his ContrAl clinics suggest are degrading. With ContrAl treatment,

> Client dignity is assured; there is no need for self-effacement since the motivation for alcohol is reduced by extinction. Excessive drinking is simply a case of a behaviour having been learned too well – somewhat like fingernails that have grown too long. The ContrAl Method provides a means for trimming.

Thus, the control of desire becomes a routine matter of self-care, based on simple decisions about taking medication, rather than complex confessional and revelational practices (associated with, for example, Alcoholics Anonymous). Ostensibly, one does not need to delve into the depths of one's soul in order to control oneself; one needs only to know oneself and one's desire in neurochemical terms.

And yet it turns out that ContrAl patient practices are not, in fact, quite as simple as trimming fingernails. Fingernails are clipped when they get too long, but other, non-

fingernail growth / functions are not affected by clipping. Naltrexone does not appear to have an analogous effect on desire. Clinical trials demonstrate that patients taking naltrexone exhibited reductions in interest in eating sweets and having sex: a result explained theoretically by the understanding that these behaviours are motivated by the endorphin system, and that "any opioidergically reinforced behaviour produced while naltrexone blocked such reinforcement should be extinguished" (Sinclair 2001: 7). Unlike fingernail clippers, naltrexone does not merely trim away the unwanted excess; instead of reducing desire for alcohol, it reduces desire itself on a general, rather than specific, basis.

This presented a practical problem for those developing naltrexone therapies: how could one extinguish the pathological urge to drink without extinguishing healthy urges to engage in all sorts of valued behaviours? The solution devised by Sinclair was a regime of 'targeted medication,' in which patients are instructed to take their naltrexone only on days they expect to drink. On these days, when naltrexone impedes the brain from being excited from behaviours (and from providing incentives for such behaviours in the future), individuals are advised to avoid engaging in activities in which they do not want to lose interest. Optimally, "patients avoid making other responses that are probably reinforced through the opioid system (e.g. eating highly palatable foods, having sex, jogging) while they are on the antagonist. Then when the craving for alcohol is manageable, they have days when no antagonist is taken, no alcohol is drunk, but these other behaviours are now enjoyed" (Sinclair 2001: 7). In this way, the brain's rewarding of drinking can be 'selectively extinguished,' while other behaviours continue to be reinforced.

Thus, whereas the decision to trim one's fingernails involves a consideration of only the nails themselves, the decision to take naltrexone involves calculations about how to control one's desire for alcohol while at the same time not unduly interfering with other desires. In fact, using naltrexone within a regime of targeted medication requires a whole complex of decisions to be made about how to conduct oneself, according to certain rules and principles of how to care for the brain, but also in relation to one's settings and plans. Every day, an individual must make calculations: 'What is the likeliness that I will drink today? If there is not much likeliness, maybe I should not take naltrexone; but what if I find myself in a situation where I unexpectedly end up drinking? Shouldn't I take one just in case?' This decision-making process could involve financial considerations as well: naltrexone provides a sort of insurance against drinking uncontrollably, but at a cost of between US$5-10 a day, some individuals might forgo coverage in an attempt to reduce their medication costs.

One could imagine a fairly complex decision-making process arising, involving evaluations of one's current states of desire (I don't feel like I want a drink that bad today, do I?), the likeliness of those desires to change (but will I later on?), one's embeddedness in geographical and social configurations of risk (after all, I am going to be going to a bar tonight; and although I don't plan on drinking, I may well run into my old drinking buddy and feel obligated), and the relative costs and benefits of taking naltrexone (if I do take naltrexone, I don't have to worry about my cravings, and I'll have peace of mind; but on the other hand, that'll make four naltrexone days this week, and I haven't even had a drink; if I don't drink again today, that's $30 this week down the drain). Moreover, once the decision has been made to take naltrexone,

one might need to consider making adjustments to one's entire daily schedule, evaluating plans and activities in light of one's modulated endorphin system. Given the fact that patients have been advised that, on naltrexone days, they should avoid pleasurable activities, they may have to give a new, intensified consideration to seemingly mundane affairs, such as exercise (can I still go for a jog this afternoon?), eating palatable food (should I avoid having cake after dinner?), and having sex (should I tell my partner that I have a headache tonight?).

Sinclair explains that his treatment regime enables naltrexone to change the person, and suggests that this is done through naltrexone's modulating effects on the brain's reward system. This is perhaps partly the case, but certainly the Sinclair method requires more than the modulation of neurochemical desire. The technology of targeted medication in fact requires individuals to make decisions about their conduct actively, their environment, and their brains. Individuals situate themselves within a continuous network of undulatory neurochemical control: naltrexone allows one to regulate one's brain chemistry according to personal, social, financial, and geographical contingencies; but while circumstances change, thought about the brain and brain-modulating medication remains a constant.

Sinclair's formulations (as cited above) inform the practices of clinicians and the experiences of patients in ContrAl clinics. Within these discursive, practical, and experiential spheres, the desiring subject has to a significant extent become neurochemical; and as a result, desires have become newly calculable, and newly targetable by technical interventions. This regulation does not require the summoning

of will-power, but rather calls upon a complex set of practices that are based upon, but not limited to, the control of one's neurochemistry.

CONCLUSION

> Mastery and awareness of one's own body can be acquired only through the effect of an investment of power in the body: gymnastics, exercises, muscle-building, nudism, glorification of the body as beautiful. [...] But once power produces this effect, there inevitably emerge the responding claims and affirmations, those of one's own body [...].
>
> Foucault, "Body/Power" (1980: 56)

This chapter has investigated one particular form of biomolecular mastery – the neurochemical control of desire. This is a form of control which, as has been discussed, may be initiated either by others or by the self; but in both cases, it should be clear that the molecular changes brought about by naltrexone is only one element within larger assemblages and investments of power. The alteration of one's neurochemistry is only done to achieve particular goals. Naltrexone is never prescribed or used merely to balance levels of brain chemicals, but is done to achieve certain ends – less drinking, better behaviour, fewer desires – and these ends arise out of socially produced values and beliefs. In a more historical sense, vast political and material investments had to be made over the course of several decades for naltrexone to emerge as a technology that could be used to control desire.

But if the possibilities for what we can and want to do with our own bodies and brains depend upon various investments of power, such investments do not strictly determine the 'responding claims and affirmations' we make in relation to the products of neuroscience and psychopharmacology. As they come to appear self-evident, obvious, and even natural, the molecular truths of the brain may fold into the ways we see and

experiences ourselves. And at this point, the molecular truths of the brain cease to be scientific constructs and instead become subjective truths which reconstitute our bodies and provide the basis for self-understandings that are not necessarily those of neuroscience, and practices that are not necessarily sanctioned by biomedical experts.

Evidence of the power of the received facts and technologies of addiction neuroscience to convince individuals to reassess and redefine themselves and their desires in neurochemical terms is found easily, and in depth, on the many addiction-recovery websites and listserv archives that are to be found on the internet. In such forums, individuals can post their own narrations of their experiences with naltrexone, allowing us to get a sense of how individuals begin to see themselves and conduct their lives under the descriptions of their neurochemicals selves that their culture provides. As a conclusion to this chapter, we will now turn to consider some of the personal narratives that appear on a forum for patients that use naltrexone as an 'aid to controlled drinking' (Deluca 2001a).

One of the striking features of many of the patients' narratives is the relationship they learn to make between themselves, their brains, and their conduct. For example, one woman, Sophia, reportedly learned from her physician that her excessive desires for drinking and her inability to control her drinking were a result of endorphin activity in her brain:

> It appears that on occasions when I would take that first glass of wine, the surge in my endorphins was so swift and high that the effects of the wine would kick in and I would lose all control as I wanted to maintain that state.....and of course ending up in a disaster (quoted in Deluca 2001c).

With the information she received from her doctor, Sophia, like many of the other patients on the forum, is able to develop a new form of self-awareness and self-understanding that allows her to reinterpret events and experiences in her life. Indeed, out of her experiences with naltrexone, her relationship with her physician, and her interaction with others online, she forges a new self identity. Having read a posting made by Serena, another naltrexone user who had coined the neurochemically-centred term 'endorphin challenged' in order to avoid the moralistic connotations of the term 'alcoholic.' Sophia, adopts the neologism and explains: "I do love the phrase 'Endorphin Challenged' because that is exactly what I am" (quoted in Deluca 2001c).

As Sophia, Serena, and others have been provided with a new understanding of their behaviours and themselves, they have become able to see themselves in a new light, and indeed, to remake themselves as different kinds of persons. An interesting point here, which reminds us of Ian Hacking's (2002c) description of dynamic nominalism or 'making up people,' is that Sophia's conception of her self is not merely shaped by her physician's description of her drinking problem, but it allows her to run with that description, and to make up her own, new category of personhood. 'Endorphin challenged' is of course not the same categorization as 'alcoholic,' even if the latter is understood to have a neurochemical component. Sophia's self, at least as far as it relates to drinking, is almost wholly neurochemical.

While Serena and Sophia are the only two individuals on the forum who refer to themselves as endorphin challenged, many other individuals describe their conduct and experiences with alcohol in neurochemical terms. But it is important to note that frequently, these novel identifications do not appear to have resulted only from

discursive interactions (with doctors or other patients) alone – experiencing the effects of naltrexone also appears as an essential component of self-reconceptualization. Accounts often describe the initial doubt felt by individuals who are told that naltrexone could solve their problems, and how such doubts were eliminated as a result of using the medication. And for many individuals, naltrexone is the pivotal element in their narratives, and indeed becomes an agent in its own right:

> This prescription [of naltrexone] is really something. . .it controls the cravings, controls the urges, it blocks some receptors or something in the brain and blocks the high from the alcohol. It blocks the pleasurable effects of alcohol, and the desire to drink, and if I do drink one or two it controls the compelling urge for more more more (Sandra, quoted in Deluca 2001b).

What the above quotation makes clear, along with many other descriptions of naltrexone on the website, is that complex assemblages of technologies, discourses, and individuals are involved in the formation of neurochemical selfhood. Sandra's experience of reduced craving is only possible because of naltrexone, and her understanding of her brain's role in determining her conduct depends upon an understanding of drinking, pleasure, and opiate antagonists that is contingent upon her association with a neurobiological thought community.

Such complex assemblages not only reconstitute identity along neurochemical lines, they also produce new forms of ethical subjectivity and experience. Naltrexone allows individuals who are plagued with compulsive urges to drink to eschew the moral precepts of vice and virtue, and to develop practical methods of thinking about and acting upon themselves, their desires, and their conduct. Dealing with problematic desires becomes primarily a technical, rather mundane – but not simple! – process. In their narratives, and in discussions with one another, individuals discuss a wide range of experiences and problems; but they formulate, interpret, and respond

to these primarily as neurochemical agents, who deploy naltrexone within a regime of self care that focuses on the molecular elements of the brain.

CHAPTER 8: KNOWING AND CARING FOR THE NEUROCHEMICAL SELF

INTRODUCTION

The preceding chapter, and indeed much of this thesis, has been concerned with two dimensions of the 'neuropolitics' of addiction and desire. On the one hand, the forms of knowledge and expertise that have made it possible to render addiction into a treatable brain disorder are themselves political. This study has been based in part upon the assumption that social, political, and material issues are not merely *relevant* to an understanding of the development of contemporary addiction neuroscience and psychopharmacology, but that these issues are essential for understanding how we have come to know what we do about addiction as a disease of the brain. Taking the most obvious example of this political and historical ontology of the desiring brain, we saw that national concerns and political objectives, such as those epitomized by the US government's War on Drugs, have led to unprecedented investment into and support for addiction science research. It is only as a result of these and other cultural conditions that it is possible, today, to understand that in addiction, processes and structures of the brain itself are responsible for the mind/brain states of craving, and these strong urges or impulses increase the likeliness of relapsing to drug-using behaviour.

On the other hand, the actual intervention into patients' brains with pharmaceutical drugs has also been described within what we might refer to as a politics of neurochemical subjectivity, because these interventions are involved in the production of new sorts of ways of identifying and governing individuals. The brain of an individual with certain types of behavioural problems (e.g., repeated drunk driving,

compulsive gambling) has come to be managed, more or less literally, as a *risky matter* that can be controlled and optimized with anti-craving pharmacotherapies that 'down-regulate' the activity of the endorphin and dopamine systems. This technoscientific management prevents the pleasure system from over-reacting to drug-associated cues; and as a result, the individual does not receive a surge of neurochemical rewards, and does not experience the cravings that lead to relapsing behaviour. This new form of power is significant not only because it produces new ways to know and rule subjects, but also because it potentially transforms the 'appropriate' means of dealing with what previously were thought of as 'social' (as distinct from biological) problems.

This thesis has not, however, examined the influence of neuroscience discourses on large-scale projects of governance that go beyond treating pathological subjects with direct interventions, to the prevention and minimization of the incidence of addiction among wider populations. The present chapter, as the penultimate of this study, moves towards a conclusion by opening out the scope of investigation to a consideration of how contemporary addiction science is being deployed within something like a 'biopolitics of the population' (Foucault 1985) – that is, attempts to foster the health and biological well-being of societies by *preventing* the onset of addiction. Such operations focus not on managing specific problematic individuals, or the treatment of particular cases of pathology; they instead are geared towards targeting 'the public' or some element thereof in order to pre-empt thoughts and behaviours (e.g., opinions that there is nothing wrong with experimenting with drug use) that might lead to drug use or addiction.

This chapter provides evidence that today, biopolitical strategies of addiction prevention govern, at least in part, through the production of a form of neurochemical subjectivity that is similar to those which provide an essential conduit of power in the regimes of naltrexone control described in Chapters 6 and 7. However, in addition to the absence of direct pharmaceutical intervention, strategies of preventative governance do not involve the deployment of scientific discourses in unmediated form (i.e., as enunciated by scientists or other biomedical experts themselves). Instead, they rely on translations of technical science into ostensibly value-neutral forms of advice and information that can be used in training and educational programs for a wide range of non-experts. Thus, as the focus of this study shifts from regulatory strategies involving direct therapeutic intervention into the lives of risky individuals, toward those strategies that place an emphasis on the preventative administrative management of populations 'at risk,' so too must the sources of empirical data change. The texts that do the work of preventative governance are not those which are produced by experts for other specialists, but are instead those which are oriented towards the population at large – i.e., popular science.

This chapter develops a case study of popular neuroscience accounts of addiction provided by the American National Institute for Drug Abuse – the world's foremost source of information on drug use and addiction – in order to examine some of the ways that 'at risk' individuals are provided with particular ways of knowing themselves and thinking about their lives that are based on incitements to care for their brains. This material is analyzed in terms of its relation to a cultivation of neurochemical subjectivity among its target audiences. The material, it is suggested, provides a model for living a good and healthy life which is based upon knowledge

about one's brain and about how one's actions and choices about substance usage may affect one's neurological functioning. Because the NIDA material represents the brain as an object of individual (and social) care, and offers neuroscience-informed techniques for living one's life on a day-to-day basis as a responsible, autonomous neurochemical subject, the material is considered as a technology of neurochemical selfhood which encourages individuals to effect, by themselves or in alliance with others, programs of self care aimed at bringing about certain neurological states that are associated with happiness, well-being, purity, and functionality.

Popular science in action

Ludwig Fleck was one of the first sociologists of science to contemplate the significance of popular science in Western societies. Towards the end of his most significant work, *Genesis and Development of a Scientific Fact* (Fleck 1977), he notes that "an epistemological investigation of popular science has yet to occur", largely because philosophers of science of his time had "never investigated actual knowledge but only a fanciful picture of it" (Fleck 1977: 112). Fleck's own comments on the matter are relatively sparse, and he acknowledges that he offers only a few "hints" on how to move beyond the role of "speculative epistemologist" (Fleck 1977: 112); but they provide a useful starting point for our analysis, since they relate to the concepts of thought styles and thought communities which have been used so far in this study.

We will recall that for Fleck, no absolute boundaries can be drawn between experts and non-experts; a thought community is a construction that is analytically useful, but what we actually find in society is a continuum of expertise. Fleck is of course mainly concerned with the 'esoteric circles' in which scientists and researchers are clustered; but he notes that these consist of only one (more specialized) pole on a

continuum of actors sharing a common thought style – which, in its most general form, does not appear as science, but as something more like common sense. Fleck notes that the style and content of most academic science is more or less incomprehensible to the uninitiated, and reminds us that if we want to find the sources that furnish the major part of every person's knowledge, we need to consider popular science.

Fleck calls attention to the ways in which popular presentations of scientific knowledge characteristically omit detail and controversy. He describes popular science as "an artistically attractive, lively, and readable exposition with last, but not least, the apodictic valuation simply to accept or reject a certain point of view" (Fleck 1977: 112). We might say that popular science involves both *simplification of detail* and *valuation of content*, because what are presented in popular science as the objective 'conclusions' of science are determined by subjective judgment and the conscious selection of particular facts. Simplification and valuation are not, however, either specific to popular science, or lamentable in general; for Fleck, every utterance, even in the most specialized scientific text, depends upon these processes. Without them, "each word would require a footnote to assign limitations and provide explanations. Each word of the footnote would need in turn a second word pyramid. If continued, this would produce a structure that could be presented only in multidimensional space." And expert knowledge, rendered impotent by a complete lack of clarity, would be "unsuitable in any practical sense" (Fleck 1977: 114).

So we might take from Fleck the insight that popular science is not produced in order to create new scientific knowledge, but rather to select and purify the contents of expert science to provide a basis for thinking and decision-making. Fleck does not

consider the matter of who does the simplifying, and towards which purposes and values they orient their translations; his mention of popular science is only made in relation to a general discussion of the range of materials with which epistemological studies of science should concern themselves. But we can push Fleck's characterization of popular science as 'purposively simplified' science a fair bit further; if we consider popular science in relation to particular fields and substantive topics, we might be able to make the case that popular science can be understood as *teleologically* performative. (While all science is perhaps performative, in the sense that it is oriented towards making action – and only certain kinds of action – possible, popular science is perhaps distinguishable insofar as it is designed with specific ends in mind, and oriented towards closing down further scientific inquiry and debate.) This does not suggest that authors of popular science wilfully and arbitrary misrepresent expert science; but that popular science can only be made by presuming and inducing particular goals, values, and futures on the basis of more or less final answers to questions about the nature of reality.

In this light, the texts, images, and representations of popular science, as points of translation in the network of scientists and society, might be thought of as the products of 'spokespeople' for scientists. Latour uses the term 'spokesperson' (or 'mouthpiece') to describe the role of the scientist in relation to the data produced by laboratory equipment, which is ostensibly one in which the scientist merely interprets the signs produced by technology. However, as Latour points out, "scientists do not say anything more than what is inscribed [by their instruments], but without their commentaries the inscriptions say considerably less!" (1987: 71). Similarly, popular science accounts of addiction do not claim to say anything more than what is

demonstrated by scientists (such accounts frequently include references to original source materials, in case there is any doubt); but without these simplified versions of expert knowledge, the conclusions scientists make about addiction would be considerably weaker.

But popular science is not *just* a mouthpiece for scientists. It also provides a service to scientists, insofar as it helps them demonstrate that their work is useful, and thus helps them recruit allies that value and support their work. Moreover, popular science is used by non-science actors (for example individuals, communities, and families) in order to achieve their own goals in the most efficient, rational, and legitimate ways possible. Thus, popular science provides a *point of brokerage* for a mutual, interdependent relationship between experts and non-experts. Popular science emerges as the product of a broker, or go-between, that is able to convince each party that while it needs the other's services, it is unable to transact with the other directly; and the success of the broker relies on its ability to appear as a more or less disinterested party that will not unduly interfere with the transaction.

NIDA's brokerage of addiction science is especially salient in relation to the governance of drugs, pleasure, and desire in contemporary advanced liberal societies, which can be characterized by a rejection, or at least suspicion, of highly visible political and social projects undertaken by governments in the name of society. Despite some shifts toward a form of 'enabling' government which increasingly uses private enterprise and market-oriented approaches to the delivery of social provisions (cf. Gilbert and Gilbert 1989), the evidence is unclear as to whether the scale of government, in terms of size and spending, is shrinking dramatically. It seems more

reasonable that the forms of governmental intervention are undergoing a transformation. Rose notes that what we are seeing is not a simple 'downsizing' of the state, but the rise of "a new kind of activist government to create freedom and those capable of inhabiting it" (Rose 1999a: xxiii).

To describe a government as "activist" seems somewhat incongruous with the crisis of authority that a state faces when it comes to acting too directly in its efforts to influence society. It seems that increasingly governance is undertaken in ways that in some senses are less direct, less activist. In matters of health and well-being, at least, it may be that one of the ways that governments, whose legitimacy is in question, take action is by engaging in *governance as brokerage*. In relation to drug use, there are scientific experts, on the one hand, that have authority over defining acceptable and unacceptable behaviours (on the basis of biological consequences); and individuals, on the other hand, that are responsible, autonomous agents that make their decisions for themselves.[19] In between, governments work to build relationships between these two parties, convincing each that they need the other; and in so doing, shape the form and content of the exchange.

Of course, not all popular science is produced and / or disseminated by governments.

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[19] In the case of choices about drug use, individuals are not entirely autonomous, because there are of course formal restrictions on their freedom, since certain laws prohibit or regulate the legality of certain types of substances; but it is increasingly acknowledged that these legal imperatives no longer create moral imperatives (or fears of punishment) strong enough to regulate drug-using conduct in the absence of direct surveillance. Individuals will, and often do, chose to take illegal drugs; in part because the authority of the state to regulate what one ingests or experiences is no longer recognized. Governments still have the legal power to control substances, but to an extent, they no longer have the authority to do so. This devolution of authority over and responsibility for personal choices does not mean the end of government, but instead gives rise to its reinvention. If people will no longer listen to the state when it tells them to 'just say no,' they perhaps will listen when it tells them 'this is what scientists say about the implications of your choices about drug use for your body and brain.'

Rose (2003b) has explored the ways in which- popular science is involved in the marketing of psychopharmacological products by the pharmaceutical industry. Rose suggests that psychopharmaceuticals, and the discourses and practices in which they are embedded, increasingly,

> oblige the individual to engage in constant risk management, and to act continually on him or herself to minimize risks by reshaping diet, lifestyle and now, by means of pharmaceuticals, the body itself. The new neurochemical self is flexible and can be reconfigured in a way that blurs the boundaries between cure, normalization, and the enhancement of capacities (Rose 2003b: 59).

Rose's analysis makes it clear that the reshaping of our self-understandings does not only involve pharmaceuticals themselves, but also the strategic positioning of the drugs within particular discursive and representational contexts – particularly in advertising and promotional campaigns.

However, the popular science of advertisements for pharmaceuticals and the educational strategies embedded in marketing techniques, which are "conceived, designed, and disseminated in the search for bio-value" (Rose 2003b: 59), are less relevant to public health initiatives than to commercial ones. In the case of drug use and addiction, neuroscience research and pharmaceutical technologies have for the most part developed in an environment dominated by biopolitical, rather than bioeconomic, concerns: supported by governments (in the interests of their constituents) and not by industries that depend on increasing demands for their health-related products. This may be changing, as desire-regulating treatments have begun to be commercially developed which offer the prospect of reaching a larger market than the relatively small (and not very affluent) population of illicit drug users – for example, brain-targeting products that help individuals regulate their cravings for nicotine. But the main sources of information about the neurochemical basis of

addiction today remain the educational and public health campaigns of government and charitable sectors, as well as reports and representations found in popular media.

This is *not* to suggest that the hand of the state is invisibly manipulating scientists and individuals; only that through its position as a broker (literally, a "broacher"), in its role of bringing together scientists and non-scientists, and in its contribution to the expansion of particular styles of thought, it exerts a certain influence over what self-understandings inform and constitute individuals, and what obligations and expectations replace – or at least supplement – those of law and decree. The educational materials produced by governments in collaboration with scientists are far from neutral; instead, they have embedded in them implicit judgments about the meanings of pleasure, and conceptions of what humans are and should be. Thus, these accounts can be thought of in terms of the production of neurochemical subjectivity, and a corresponding set of obligations and expectations that embody definite norms of conduct and citizenship.

MAKING NEUROBIOLOGICAL CITIZENS

Former NIDA director Alan Leshner, one of the most outspoken proponents of the argument that addiction is a disease of the brain, argues that in light of modern neuroscience, the "notion of mind-body dualism is a thing of the past" (quoted in Leavitt 2003: 3). For Leshner, addiction is not a characteristic of a person as much as it is a characteristic of the brain; and to the extent that the term 'addict' is still sometimes used to describe a person, it indicates only that person's state of brain functioning. As Leshner explained during an interview on the American Public Broadcasting Service while he was still the Director of NIDA: "People are able to understand this now when they think about schizophrenic people who act in very

bizarre ways. Why are they acting so weirdly? Because there's something wrong with their brain. That's exactly what's happening in addiction." The individual whose personality, conduct, and affective states drastically change with the onset of addiction cannot be understood apart from neurochemistry: "You are an addict because your brain has been changed by drugs" (Leshner 1998).

Unfortunately, Leshner notes, there is "terrible disconnect" between the public's understandings of drug use and addiction, and "what scientific findings are actually showing us;" too often, individuals form opinions and make decisions about these matters that are "intuition-based or ideology-based". Leshner argues that in order to help address this problem, the task of scientists should be "not only to generate interesting scientific data, but to make it both useful and used"; the power and insight of laboratory science must be brought to bear upon individual lives and personal choices. And it is only when contemporary scientific knowledge is put to use by lay individuals as well as governments that real progress be will be made in dealing with drug abuse and addiction, which constitute "the most complex phenomenon that's facing our society" (Leshner 1998).

It is in the gap between scientists and society that NIDA comes in – to broach the gulf between scientific fact and public perception. NIDA works toward teaching individuals that in order to deal with drug- and addiction-related problems in day-to-day life, they should "seek the scientific solutions that have been so successful for helping us deal with heart disease, cancer, schizophrenia, other kinds of disorders where science has been hope, science has provided the answers" (Leshner 1998). And at the same time, NIDA helps scientists achieve the goal of being "the source of

hope about addiction for this country" (Leshner 1998). To the extent that the power of science can be brought to bear on addiction, the need for science, the support of scientists, the importance of scientific research also increases.[20]

NIDA is at the forefront of the many groups and agencies involved in public health and education initiatives on addiction and drug use. The organization is notable both because it is by far the world's largest and most active organization in this field, but also because for decades it has been one of the world's most vigorous proponents, governmental or otherwise, of neuroscience and psychopharmacological understandings of addiction. Although the vast majority of its efforts are oriented toward treatment initiatives and the funding of research in a wide range of areas (e.g., basic, clinical, and epidemiological studies), NIDA has also played a pioneering role in developing science-based prevention initiatives which provide the lay public with popular, scientific information about the effects of drug abuse on the brain.

Although drug abuse prevention efforts have a long history, most of which involves legal regulation (e.g., the formulation of laws and policies, the policing of populations, surveillance of individuals), such prevention efforts have, over the last two decades, increasingly begun to be supplemented with more liberal forms of governance. Individuals are increasingly considered not simply as subjects of rule, but as citizens who must be convinced that they should be responsible for themselves and others. In a 1997 book entitled *The Selfish Brain*, NIDA's first Director, Robert DuPont, asserts

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[20] This brokerage also allows NIDA to set the terms of the exchange within certain boundaries, specifying to scientists what sorts of brain research are of value to individuals – and to individuals, which scientific facts are relevant to persons and communities.

that "[p]revention of addiction is everyone's responsibility, one that is never finished" (DuPont 1997: 283) because drug use and abuse not only harm the American nation, but also because they directly impact all individuals and families. DuPont describes the $67 billion that the US loses each year as a result of illegal drug use in private terms, as an "involuntary addiction tax paid by everyone, which comes to about $270 each year for every man, woman, and child in the United States" (DuPont 1997: 4).

The National Institute on Drug Abuse's mission is to "lead the Nation in bringing the power of science to bear on drug abuse and addiction" (NIDA n.d.-h). And since the most critical time for prevention is thought to be before the age of twelve, when non-medical drug use is understood to begin to increase markedly among students, NIDA has developed a science-based curriculum specifically for students at the early stages of their elementary schooling.

> The curriculum is titled *Brain Power! The NIDA Junior Scientist Program* and consists of six modules. The goal of the curriculum is to lay the foundation for future scientific learning and substance abuse prevention efforts by providing an early elementary school-age audience with a basis of knowledge and critical thinking skills (NIDA n.d.-i).

For each of the six *Brain Power!* modules, NIDA provides a package for educators (second- and third-grade teachers) of about twenty-five to thirty pages of material, each of which includes instructions and background information for the teacher, activities ("missions") and printouts for students, and an informational newsletter to be copied and sent to students' parents.

The NIDA program is remarkable for a number of reasons. For instance, this program, geared towards students between about 8 and 10 years old, predictably contains silly rhymes, elementary humour, and simple stories; but these appear alongside, and in

relation to, quite sophisticated representations and explanations of the molecular structures of the brain, patterns of neural communication, and the biochemical interactions between drugs and neurochemistry. Indeed, much of program's content could easily surpass the average knowledge of children's parents, and it is interesting to note that the parental newsletters contain virtually the same content as the childrens' lessons – as well as a list of 'additional resources' to which parents can refer. Parents are not simply enjoindered to help educate their kids, but also seem to be the somewhat indirect targets of NIDA's training.

With this program, children – and to an extent, their families – are recruited as part of a national, science-based strategy effort of drug prevention. This recruitment is most literally apparent in NIDA's invitation to children to complete the six modules and become 'junior scientists' of the *Brain Power!* Club. And in fact, as the following examination of the content and educational strategy of the six modules indicates, this program provides a fascinating example of the induction of very young individuals into neurochemical styles of thinking about drug use and addiction. In so doing, the program provides the basis for the development of notions and norms about the self, the brain, and behaviour that relate closely to ideas about citizenship, individual responsibility, and social obligation.

NIDA's *Brain Power!* Program

The first module ("Ooey Gooey: Making sense of scientific inquiry") of the *Brain Power!* program does not begin with a discussion of drugs, addiction, or the brain; rather, it focuses on introducing students to the processes of scientific inquiry – observation, prediction, experimentation, and conclusion-making – and emphasizes the value of using scientific inquiry as a tool for approaching and solving problems.

Children themselves are inducted into the fringes of the scientific thought community, because throughout the program "they, too, will be working as scientists" (NIDA n.d.-q). Children are encouraged to see themselves as champions of the objective pursuit of knowledge of the brain, dedicated to fostering health and well-being – just like the NIDA scientists they see portrayed in their *Brain Power!* trading cards:

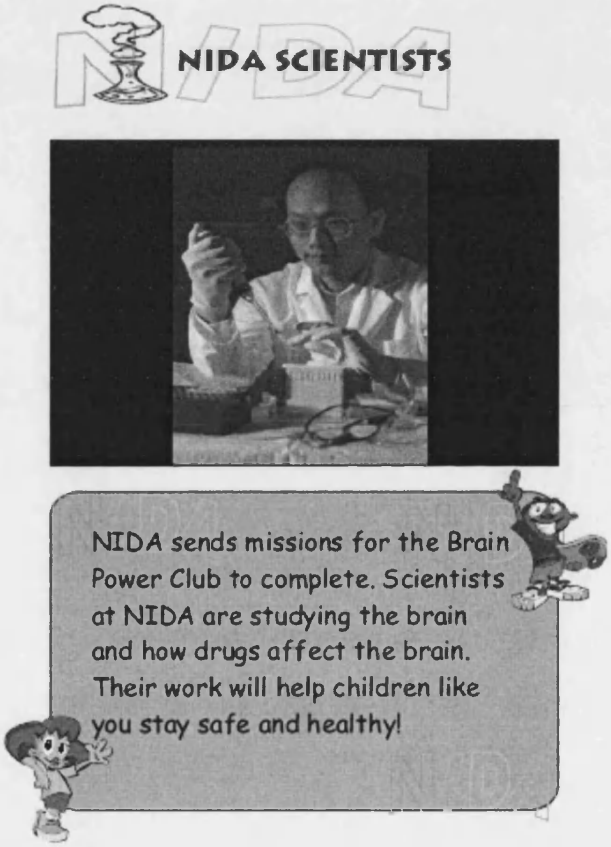

Figure 5. Trading card from the first module of the Brain Power! Program (NIDA n.d.-s)

In the first parental newsletter, family members of the 'NIDA junior scientists' are encouraged to reinforce at home what their children have been learning at school. Parents are brought into an alliance with NIDA to keep children of the nation safe and healthy: "By teaching young children about how drugs affect the body, we can lay a foundation for students to make better decisions about their own health and future" (NIDA n.d.-j).

During the second module ("Brains in a box: what your brain can do"), students are provided with activities that allow them to learn about and visualize their brains, and to understand that the brain, as the control centre for their bodies, is a treasure worth caring for. Children are provided with modelling clay and instructed on how to make a three-dimensional model of the brain, including the two hemispheres of the cerebral cortex, the cerebellum, and the brain stem. Fictional characters of an introductory story invite children to "Make something wonderful with the Play-Doh – something that represents an amazing gift" (NIDA n.d.-f). Teachers then guide the children through the activity, providing information about the functions of each part of the brain.

One of the trading cards for this module dramatically illustrates how the brain has an effect on personality, and suggests the serious repercussions that can follow from intervening into the brain. It features Phineas Gage, a man who suffered a brain injury in the mid-19th century, and who has become a figure of considerable interest in the neurosciences:

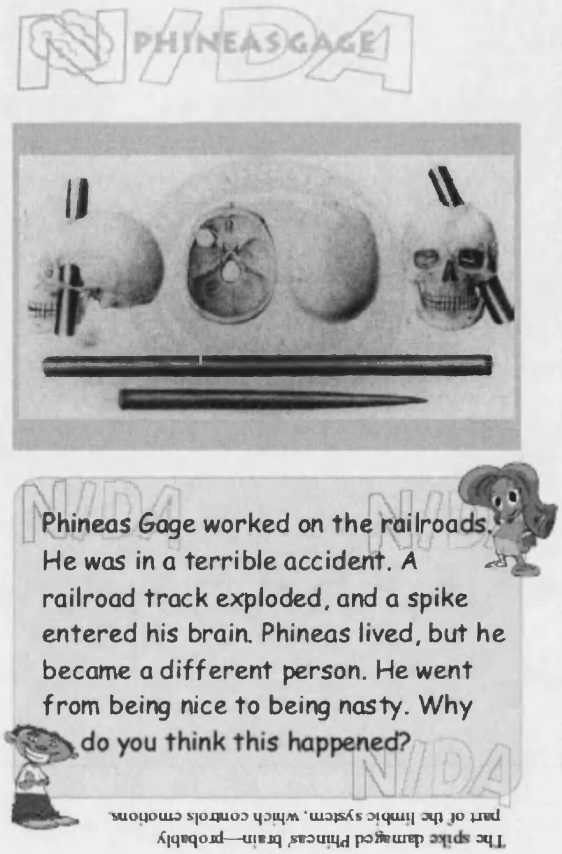

Figure 6. Trading card for NIDA Brain Power! Program, module 2 (NIDA n.d.-t)

From this card, children learn that changes to the brain can create (negative) changes to the person. Gage went from "nice to nasty" because of damage to the limbic system – one of the main areas affected by addictive drugs, the children will later learn.

Gage's case is not instructive only for NIDA junior scientists, but also for professional neuroscientists. In his book, *Descartes' Error: Emotion, Reason, and the Human Brain*, Antonio Damasio – the Head of the department of Neurology at the University of Iowa – uses Gage as an example of the neural basis of personality, as well as ethical behaviour:

> Gage had once known all he needed to know about making choices conducive to his betterment. He had a sense of personal and social

> responsibility. He was well adapted in terms of social convention and appears to have been ethical in his dealings. After the accident, he no longer showed respect for social convention, ethics were violated, the decisions he made did not take into account his best interests" (Damasio, cited in Jonsen 2003: 12).

Before its trauma, Gage's brain was apparently healthy and properly functioning, and Gage was, to use NIDA's term, 'nice.' He made the right sort of choices about his own well-being, his responsibilities to others, and his observance of social standards of conduct. After his frontal lobe was pierced, Gage maintained control over his physical capacities and cognitive functions, but he became a nasty person and a bad citizen: untrustworthy, unconcerned with his own welfare, and unwilling to adhere to social norms.

The moral of Gage's story fits in well with the goals of *Brain Power!*'s second module, which is, as parents are informed in their newsletter,

> to show children how amazing the brain really is. Most children this age know that the brain helps them think, but they don't realize that the brain is also responsible for just about everything else, too—from regulating heartbeat and breathing to controlling emotions and artistic expression. By teaching them about "*Brain Power!*," we hope that they will think twice about doing anything that might harm their brains (NIDA n.d.-k).

Gage's transformation demonstrates to neuroscientists that neuroanatomy and personality cannot be separated from one another, and to children enrolled in NIDA's *Brain Power!* program that care for the brain amounts to care for the self, as well as care for others.

In the third module ("Sending and Receiving Messages"), students learn in more detail about how the brain works, especially how it communicates with the rest of the body through the process of neurotransmission. This module reiterates the value of

the brain and its importance in controlling the body and the mind, and emphasizes the extreme delicacy of the brain's cellular structures. NIDA acknowledges to teachers that "Neurotransmission is a very difficult subject and may be a challenge for some second- and third-grade students" (NIDA n.d.-a), and in order to make the principles of neurochemical communication easier to grasp for students, the teacher's *Brain Power!* package for this module contains detailed instructions and props that will enable groups of children to act out the process of neurotransmission, with each child taking a part related to a neurological structure.

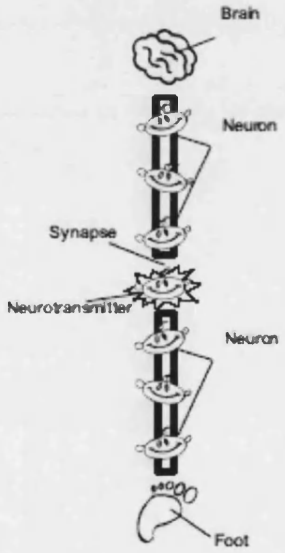

Figure 7. Teacher instructions from NIDA's Brain Power! Program, module 3 (NIDA n.d.-n)

For example, students in a 'neuron team,' enclosed in a masking-tape outline of a nerve cell, receive an impulse (i.e., a message on a piece of paper) from a 'sore foot' student and relay the message to the 'neurotransmitter student,' who passes the impulse on to the next 'neuron team.'

The reasons for introducing students to the principles of neurotransmitters are explained to parents in this module's newsletter:

> For one, scientific information about the brain and the nervous system is growing at a rapid rate. By the time your child is an adult, we may understand the mechanisms behind many diseases of the nervous system, such as Alzheimer's disease and multiple sclerosis. People will need to understand how the brain works in order to make informed decisions about their health and the health of their families (NIDA n.d.-l).

In relation to the brain disease of addiction, however, the neurological mechanisms are already well-enough understood that these lessons will allow students to make responsible, informed decisions about their conduct in relation to their neurochemistry. Thus another key reason for introducing neurotransmission to students is that the *Brain Power!* program is "paving the way for explaining what happens if people interfere with this process by taking drugs" (NIDA n.d.-l). This is important because, as children learn in the following modules, drugs have a major impact on neurotransmission.

The fourth *Brain Power!* module ("Medicines and Drugs: What's Helpful, What's Harmful") focuses on how drugs – both licit and illicit – effect the body and the brain. In order to meet one of the modules key learning objectives – i.e., that students become able to "classify drugs and their effects on the body into two groups: helpful medicines and harmful drugs" (NIDA n.d.-g) – the students are taught about different types of drugs, from aspirin and immunizations to alcohol and cocaine, in terms of their uses, how they work, and their effects on the body. Perhaps predictably, information about the effects of drugs is simplified, and while the "medical" drugs are described solely in terms of the intended therapeutic effects (and not their possibly undesirable side-effects), the effects of "harmful drugs" are described only in terms of the negative consequences (with no mention made of recreational uses or pleasant affective enhancements).

Significantly, the effects of the harmful drugs are always related to negative brain changes. For example, in a 'who am I?' riddle, illegal drugs describe themselves to children:

> Each kind of me changes the brain;
> Once the brain is changed it's never quite the same.
> Marijuana is one kind to smoke,
> And the white powder, cocaine, is also called coke.
> Cocaine can make someone feel quite high;
> But watch out, that feeling does die.
> It's not hard to go over the top;
> Then someone will find it too hard to stop (NIDA n.d.-r).

Once again, the basis for decision-making about using substances is their effects on the brain, which are here presented as irreversible and, as the information included in the teacher's discussion guide makes more explicit, addiction-producing: "Both cocaine and marijuana turn on the pleasure center, part of the limbic system, making the body crave the substance" (NIDA n.d.-r). In the take-home newsletter, parents are encouraged to work with their children to clarify points that remain unclear, "so that when the time comes, he or she will make a solid, science-based decision not to take drugs" (NIDA n.d.-m).

The fifth module of the *Brain Power!* program ("The Science Behind Smoking") investigates the neurobiological basis of addiction through a discussion of smoking and the effects of nicotine on the brain. Children are reminded of the principles of neurotransmission that they reviewed (and enacted) in the third module, and are presented with posters and explanations which demonstrate how nicotine, one of the

"harmful drugs" of module 4, interferes with the brain's natural communication process.

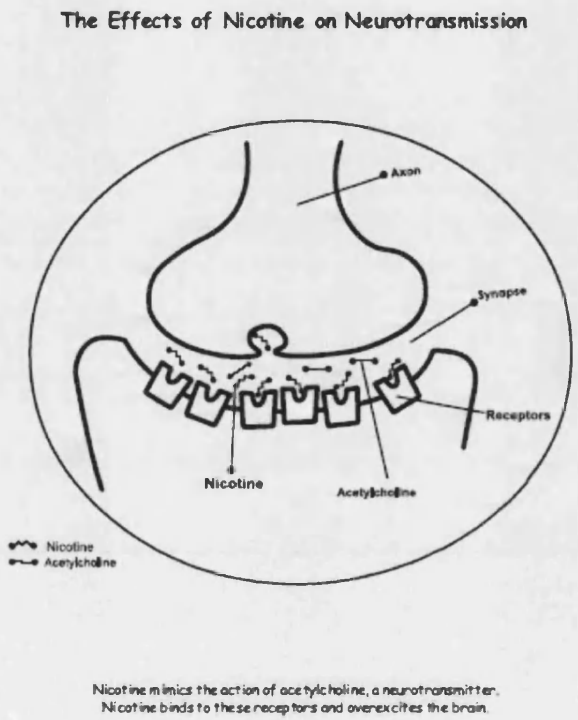

Figure 8. A poster from NIDA's Brain Power! Program, module 5 (NIDA n.d.-e)

As the *Brain Power!* material explains, the shape of nicotine molecules is very similar to that of the neurotransmitter acetylcholine, which is involved in many biological functions, including muscle movement, learning, appetite, and mood. Teachers are instructed to show their class a poster representing the effect of nicotine on neurotransmission (shown above), and to "[a]sk students what they think it means if nicotine takes over the functions of a neurotransmitter. Explain that when nicotine takes over the functions of a neurotransmitter, it is the beginning of addiction" (NIDA n.d.-p).

It is in this module that the neurobiology addiction is finally directly addressed, as children are introduced to what is referred to in scientific literature as the 'dopamine hypothesis of addiction.' The *Brain Power!* material explains that nicotine also affects areas of the brain involved in regulating levels of the neurotransmitter dopamine, which produces feelings of pleasure and reward. "Increased levels of dopamine produce the strong, pleasurable feelings that lead to nicotine addiction. In fact, nicotine is so addictive that it is usually very hard for people to quit using tobacco products" (NIDA n.d.-b). Surpluses of dopamine – levels above and beyond the amounts needed for normal functioning – result in surpluses of pleasure, and deficits of self-control.

Brain Power! explains the causes of addiction in terms of the structural and functional changes in the brain, but these brain changes are of concern not in and of themselves. Students learn to associate the neurobiology of addiction with mental, personal, and social effects, such as:

> - A strong compulsion or need to use drugs despite negative consequences (someone keeps using drugs even though he or she is having problems);
> - Loss of control over the amount of the drug used (someone uses more than he or she plans) and other drug-related behavior (someone does or says things he or she would not ordinarily say or do);
> - Intense craving for the drug when it is not available. This craving is due to changes in the brain. Once a person is addicted, he or she must have the drug just to keep from feeling bad. This is because drugs can cause changes in the functioning of neurotransmitters in the brain (NIDA n.d.-b).

Thus, changes in the brain involved with the onset of addiction are used to explain changes to personality, mood, and behaviour. Students are taught that compulsions,

desires, and losses of ability to control one's conduct stem directly from brain states; that mental experiences and self-control are matters of brain chemistry.

Children learn that addiction-causing drugs change people by changing their limbic systems – the same system, of course, that one of the *Brain Power!* trading cards suggested was changed in Phineas Gage's brain when a railroad spike entered his brain and changed him from 'nice' to 'naughty.'

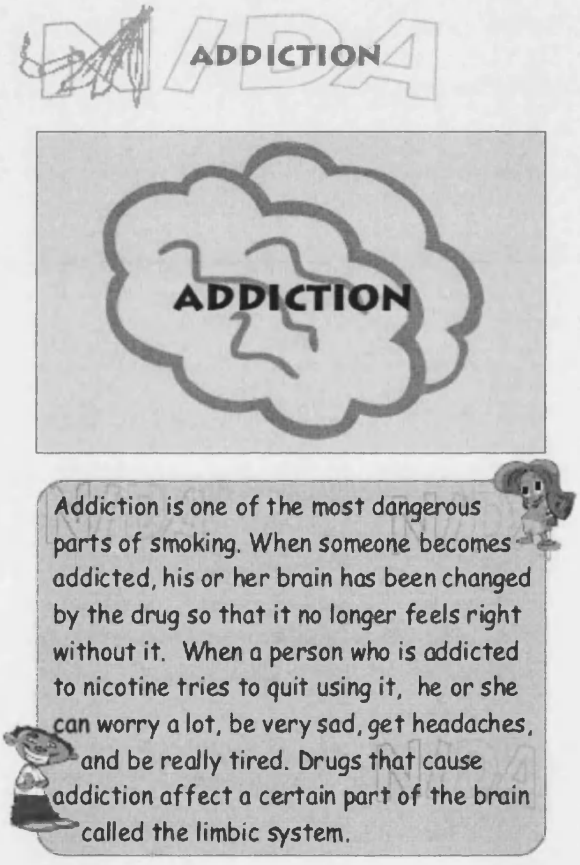

Figure 9. Trading card from NIDA's Brain Power! Program, module 5 (NIDA n.d.-c)

In the trading card above, the brain is described as not only the source of feelings, but of self: *it*, not the addicted person, is what no longer feels right without the drug.

The final module of *Brain Power!* ("How Drugs Affect the Brain") is organized around the objective of enabling students to apply what they have learned in previous

modules about the brain, its functioning, and the negative changes to brain functioning that result from the use nicotine to other harmful drugs. Students are placed into groups and instructed to draw conclusions about what they have learned during the program. An example of a desirable conclusion to be drawn is provided to teachers: "It is important to take care of your brain by not putting unnecessary drugs into your body" (NIDA n.d.-o). While the intended learning outcome for students is, unsurprisingly, that drugs should be avoided, what is notable is that these reasons are based around attempts to maintain neurobiological health: "Each group's conclusion should emphasize the importance of not taking any substance that could harm the way the brain and the nervous system work" (NIDA n.d.-o). With the completion of a final assignment – to create a poster that conveys an important message of the *Brain Power!* Program – students become official members of the NIDA Junior Scientists club, and are able to consider themselves in possession of the tools of science which are necessary to make proper decisions about their brains, their bodies, and their lives.

CONCLUSION

The content of NIDA's *Brain Power!* program is representative of contemporary changes in the ways that selfhood is being reconstituted in an era in which the precepts and principles of molecular biology are increasingly coming to be deployed in understanding and governing human life. *Brain Power!*, as well as other NIDA initiatives, recruit individuals to neuroscience styles of thought, and as they encourage children (as well as adults) to think of themselves, their actions, and their experiences in terms of their brains, they are involved in the production of neurochemical forms of subjectivity that are governable in new sorts of ways. Gone are the days of 'just say no,' when children were expected to avoid drugs simply because they were so instructed. The *Brain Power!* program, sharply contrasting with educational material

from as little as two decades ago, is not oriented towards a simple binary distinction between legal and illegal drugs (nicotine and alcohol, for example, are categorized as harmful). Instead, it involves a more subtle strategic orientation based on an incitement to care about and for the neurochemical self, which is only achievable when individuals are inducted into neurobiological styles of thought.

As Fleck notes, styles of thought exert a 'gentle constraint' on the individual, steering her attention to some things and not others. What interests Fleck is not that thought styles exist (he takes them to be necessary, since without their constraints, we would not be able to distinguish, among the endlessly varied information our senses receive, the differences that make a difference), but that different styles co-exist, that we can move between them, and that as we come to take on a particular thought style, we reshape our understandings of ourselves and the world around us. Fleck points out that every induction into a style of thinking creates new possibilities for thought and action. The assimilation of scientific thought styles, for example, "is epistemologically analogous to the initiations we know from ethnology and the history of civilization. Their effect is not merely formal. The Holy Ghost as it were descends upon the novice, who will now be able to see what has hitherto been invisible to him." (1977: 104). And altered 'sight' not only makes new demands on individuals in regards to how they express themselves, but also changes how our bodies themselves contribute to "setting limits and providing capacities for people to act" (Bury 1997: 198).

This chapter has examined how a form of popular science – addiction education materials for school children provides a means for introducing children (and perhaps

their parents) to new styles of thought and to 'tools of science' that allow them to 'see' new things. The *Brain Power!* Program enables children to bring the power of science to bear on themselves and their brains, to imagine new possibilities for their bodies, to see new things, and to perceive the self in new ways. For an indication of these changes, we need go no further than the representations produced by students themselves in an educational poster competition sponsored by NIDA.

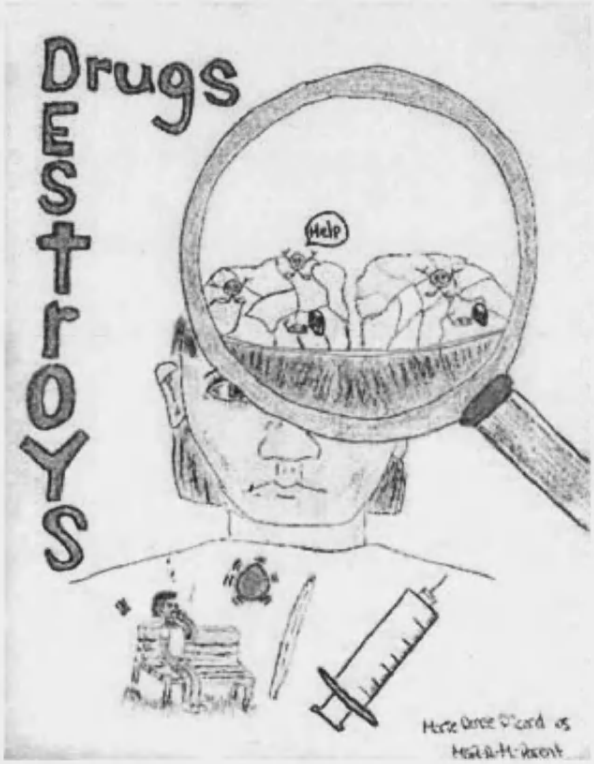

Figure 10. A winner of Scholastic's NIDA-sponsored 'Heads Up' poster competition (Scholastic n.d.)

In the poster above, we see how the tools of science allow for a reinterpretation of the reality of individual actions. In the lower left corner, an individual is portrayed from a non-expert, common sense perspective: a young man sits on a bench, smoking some sort of substance, with no negative consequences apparent. But in the perspective that dominates, the tools of science (represented by the magnifying glass) allow us to observe the truth of the drug use by revealing the neurobiological reality. The truth of

the brain belies the common-sense interpretation of appearances, offering evidence of serious harm.

The poster illustrates a significant point that Fleck does not consider when he focuses on constraints to cognitive perception; namely, that the constraints of a thought style are not *only* cognitive; when a style of thought becomes part of a person's general world view and is used in his or her day-to-day life, it does not just set limits to what can be seen or what can be asked; in reconfiguring the way we see and judge things, it also changes our interpretations of our bodies and our actions, and the expectations and objectives we develop for ourselves. The neurobiological thought style, represented in the illustration with a magnifying glass, allows the brain to call attention to itself, and to make demands upon the individual.

NIDA imparts to children an understanding of themselves and their futures in terms of a sort of neurological potential: each child possesses latent 'brain power' capable of development, with proper care, into actual capacities. Brains appear as resources that should be used, developed – and certainly protected from harm.

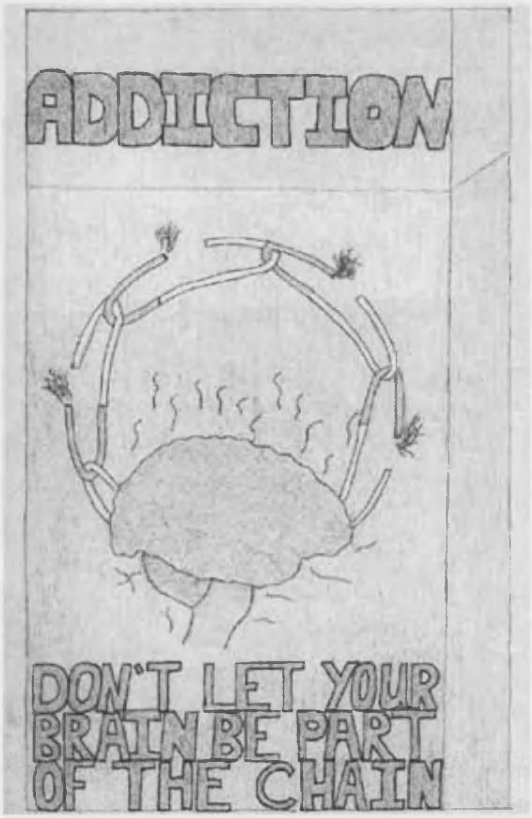

Figure 11. A winner of Scholastic's NIDA-sponsored 'Heads Up' poster competition (Scholastic n.d.)

Self-knowledge in terms of neurochemistry, and knowledge of drug use in terms of neurochemical damage, provides the basis for neurobiological imperatives to conduct onself in certain ways. One must consider one's brain when making decisions about how to act, and keep his or her brain free from the shackles of addiction.

Thus, the neurological model of selfhood produces understandings of the most essential part of oneself, the brain, as prone to pathology; and corresponding obligations to participate actively in the monitoring and prevention of risky conduct. Children in the NIDA program are not merely provided with 'techniques for the leading of a life in the face of illness' (Rose and Novas 2003: 14), but are provided with resources to avoid perhaps ever coming face-to-face with illness. Individuals are incited to optimize neurological health even without the specter of specific susceptibilities or tendencies towards mental illness.

All of this suggests that NIDA's program can be thought of as providing the basis for devising technologies of neurochemical selfhood. Newly constituted self-understandings form the basis for a new regime of self-governance. Students are trained to be able to use neurobiological facts in order to interpret and evaluate conduct, re-orienting their behaviours to a consideration of their brains. Standards of conduct are formulated almost exclusively in terms of a care of the neurochemical self; systems of choice and judgment are established for the sake of one's own body, rather than concern for the social body. And what is at risk is more than one's status as a good legal subject; one's own brain, one's self is at stake.

This is not to suggest that it is *only* the self that is at stake, or that these technologies of self have developed outside of projects of governance. It is not mere coincidence that even when individuals make a choice to abstain from drug use in order to meet their own desires for health rather than to avoid social disapproval or legal repercussions, their decisions still coincide, to a considerable extent, with cultural standards. When, as in *Figure 10*, individuals hear from inside themselves a call to care for their neurobiological selves, to make healthy, brain-friendly choices, it is not only their brains that they hear. For how have they come to know their brains? Who has initiated them into the neurochemical thought style? The biopolitical dimensions here are obvious: NIDA is mandated to create children's education programs (and is well funded to do so) precisely because drug and alcohol use are understood to be the leading preventable cause of death in the US, and to cost taxpayers more than US$300 billion a year (Volkow 2003).

But we should be wary of any temptation to reduce these developments to political or disciplinary aspirations. NIDA is not fabricating facts about the brain, and knowledge of the brain does not translate directly into power. NIDA is not disciplining students by programmatically subjecting them to forms of surveillance that are internalized in order to produce docile subjects. This form of power is conditioned upon the premises of liberal governance – upon administering and enhancing freedom rather than repressing it. Nothing more, and nothing less, is happening than the making, or incorporation, of neurobiological citizens. Students become incorporated into neurological societies – training allows them to join the community of free and responsible individuals who are capable to manage their freedom and their lives through a management of their brains. And as a result of this incorporation, their bodies, which provided them with rights, also end up making demands on their choices and conduct.

CHAPTER 9: CONCLUSION

What sorts of desires should be pursued and gratified? Which should be avoided, or brought under control? How does one know if a desire is becoming too urgent, and what can one do about the matter? This thesis has examined some of the ways that the answers to these questions, which depend upon historically contingent understandings of what desires mean, consist of, and do, are today coming to involve understandings of ourselves as neurochemical subjects.

In order to do this, it traced the neuromolecular style of thinking about addiction back to points at which some of its most fundamental theoretical and epistemological principles emerged, and followed the development of representations of and interventions on addiction as a neurological condition through to the present – always in relation to the discursive, practical, material, technical, and relational conditions which made such developments possible.

This genealogy provided a basis for an examination of some of the changes that are occurring in the ways in which we are coming to think about ourselves in general, and ourselves as subjects of desire in particular. Increasingly, the knowledge and practices of the molecular life sciences – as well as those strategies and rationalities of institutions and authorities which draw upon them for their legitimacy and effectiveness – relate our identities, our experiences, our impulses and our behaviours to our neurological constitutions. The workings of our brain at a molecular level, the precise arrangement and combination of various parts or elements of our biochemistry, are understood to influence, in a fundamental way, who we are and what we do; and

correspondingly, the functions and structures of our brains have increasingly been subject to intervention in efforts to reshape our conduct and our selves.

In a general sense, we can think of these developments as the 'making up' (cf. Hacking 2002c) of neurochemical subjects: today, manifold elements in our culture – our scientists, our media, our health experts, our public officials – use the concepts and terminology of the brain sciences to describe the biological basis of an expanding range of conditions and experiences. As we come not only to understand these explanations, but also to apply them to our own lives, we become, to a significant extent, individuals whose self-conceptions and conceptions of others are bound up in ideas about our biology, and whose repertoires of acting and behaving increasingly include practices which only make sense within a neurobiological style of thought. As we develop views of and beliefs about human beings as, at least in part, neurochemical beings, we develop new possibilities for governing ourselves and others. This study has examined some of the ways that individuals today have come to be provided both with the language that makes it possible to think of and describe themselves as individuals whose pleasures, impulses, drives, and motivations originate in the reward circuitry of their nervous systems, and with technologies and practices for acting on themselves as desiring subjects of a neurobiological kind.

As this thesis has demonstrated, the process through which we have come to know and care for ourselves as neurobiological subjects is a complex one. It involved, in part, the emergence of new rules and strategies for the production of truth: beginning by examining the theoretical and empirical precursors to our current molecular and neurochemical models, neurobiological theories of addiction and dependence were

described in relation to factors which made their origins possible. For example, when applied to drug use, existing theoretical principles (e.g., ideas about the human body as a self-regulating, homeostatic entity) made it possible to formulate new research questions about addiction; networks of researchers, brought into contact through conferences and the development of specialized research centres, made it possible for researchers to form a loose community sharing a common style of thought; legal and social prohibitions of drug use made available for (some) researchers a captive group of subjects which could be observed continuously; public health programs provided material and institutional provisions for not just the treatment or discipline of addicts, but also for the study of the addicted condition; methodological innovations made it possible to collect and analyse data that would reveal findings not apparent to casual observation; technological advances made it possible to discover new things, and to determine new criteria of objectivity. And as these and other conditions made possible the development of the biomolecular thought style to the point at which the brain became an obligatory passage point for truth of addiction to be enunciated, even apparently social and environmental factors came to be thought of as involving a biological process: their relevance to addiction science was formulated in their ability to affect neurological processes.

This new regime of truth was essentially related to a new ontology of compulsive desire. The shift from molar to molecular studies of addiction did not simply involve the development of a more detailed understanding of the same phenomenon; the shift was actually accompanied by a redefinition of the facts and biology of addiction. Thus, we saw that whereas addiction was, before the 1960s, understood to result from the effects of drugs of addiction on the brain (and the body's response to them), only a

few decades later, as laboratory technologies and methods were developed which allowed brains to be studied at the molecular level, the primary mechanism of addiction came to be defined in terms of the brain's own system of chemical rewards. What came to count as evidence that a drug or activity was addictive came to be, to a significant extent, information about the functioning of the endogenous neurotransmitter systems (especially those of dopamine and the endorphins). Addiction came to be understood as a state in which these systems, which reward behaviour through the production of pleasurable sensations, became dysregulated. Moreover, as researchers devised neuroscientific and pharmacological investigations which translated behavioural theories of conditioning into principles of brain functioning, the dysregulated brain of the addicted individual came to be seen as a persistent condition which resulted in pathological cravings that continued even if (and long after) an individual had become abstinent. The subject of neurochemical craving was thought to be at constant risk of relapse – conditioned neurochemical responses to environmental stimuli, for example, could trigger craving impulses that led the individual back to former patterns of pathological behaviour.

The generation of these new neurochemical facts of addiction, which depended upon the assemblage of complex interrelations of social, technical, political, and practical factors, had implications that extended far beyond the laboratory study of the human (and animal) brain. The elucidation of new formations of risk, in which the craving neurobiological subject was embedded, allowed for a reconceptualization of the therapeutic objectives of addiction medicine. Expert care expanded beyond the treatment of the individual experiencing withdrawal symptoms, and began to take part in the management of factors that increased a subject's likeliness to relapse. The

novelty here was not simply the shift from acute treatment to chronic care (this shift had already begun within the behavioural psychologies of the early 1970s), but its relation to the biomedical problematization of relapse. Whereas behaviouralist strategies focused on teaching coping and avoidance strategies to their subjects that would allow individuals to render desires less harmful, the interventions developed by neuroscientists and psychopharmacologists were neither psychological nor environmental: 'rational pharmacotherapies' took as their target craving itself; or rather, the neurochemical processes associated with craving.

Brain-targeting pharmacological technologies were developed by researchers according to new logics of intervention that have little to do with fortifying weak will-power, supplementing inadequate moral fortitude or deficient self-discipline. While psychoanalytic treatments (as well as those of Twelve Step groups and similar programs) might seek to strengthen self-control in order to enable individuals to take control of their lives and realize their fundamental purposes, the object targeted by naltrexone and other similar pharmaceutical interventions was more specific than 'the will,' and these therapies were developed with objectives much less grandiose than governing the faculties of conscious and intentional action. Indeed, tropes of will-power are virtually absent in the biomolecular literatures that have been investigated in this study. The interventions of contemporary addiction pharmacotherapies have been formulated and justified only on the basis of modulating the biochemical mechanisms that produce the intense, pathological cravings that many individuals experience as overwhelming.

Thus, this thesis has argued that the molecular brain sciences of the contemporary era have not taken as their project the governing of wills, but rather the governance of craving and desire. The latter project is less ambitious than an all-encompassing reformation of the subject as a responsible and autonomous individual; but this is not to suggest that it cannot contribute to the ability of individuals to manage their own freedom. Nor is it to suggest that such interventions are not intimately bound up with issues of subjectivity and experiences of selfhood. Terms that individuals have coined and used to describe themselves, such 'endorphin-challenged' and 'dopamine head' may appear humourous and imprecise, but they should not be laughed off by those of us interested in the formation of novel, biologically-informed categorizations of humans. Indeed, such lay terminologies may indicate, in a more persuasive way than the existence of expert classifications, the development of *neurobiological human kinds*, for they offer evidence that such classifications are actually adopted by individuals as elements of their own self-understanding. The autobiographical descriptions of naltrexone users and their own accounts of their experiences on these medications indicate that their understandings of themselves change as a result of taking naltrexone. And it seems clear that this case of making up neurobiological selves is not merely discursive, but technological as well: individuals may have their desires and compulsions explained to them as neurochemical phenomena, but they may also doubt those explanations and resist expert classifications. For many individuals it seems that experiences of naltrexone remove doubts about the neurochemical nature of their desires.

This study has argued that naltrexone and similar pharmacotherapies might best be thought of as *technologies of the neurochemical self*, because they seem to engender a

mode of forming and shaping the self that relies on (at least the perception of) technoscientific control of the brain – the part of the body that, more than any other, is associated with personhood and identity. However, this thesis has not attempted to make the case that such technologies can be understood independent of political and social factors. Indeed, it has made an argument that would be more easily deployed to support the opposite case: the progress of the molecular research into addiction and the development of desire-targeting pharmacotherapies such as naltrexone have been closely related, from their very origins, with political strategies – particularly those of the US government and its waging of the War on Drugs – that sought to maximize the well-being of populations by reducing the social and economic harms associated with drug use and addiction. For example, without the infusion, from the US government, of vast amounts of resources into neuroscience and pharmacological research into addiction, and the creation of special economic and regulatory conditions for pharmaceutical companies, it is doubtful that naltrexone would have developed in the way that it has. This is just one of the many elements of this study that has suggested that our understandings of ourselves as subjects of neurochemical desire must be understood within a biopolitical context.

This study's discussion of the use of naltrexone as a form of penal psychopharmacology within the programs of 'therapeutic justice' of US drug courts is perhaps the most striking example of how technologies of the neurochemical self can be brought in line with the aspirations of institutions most closely associated with disciplinary power and deployed within projects of normalization. But the biopolitics of contemporary neuroscience and psychopharmacology cannot be reduced to such operations. For today, the assumptions of neurobiology extend well beyond the

courtroom, the hospital, and the doctor-patient interaction. The views and values embodied in neuroscience discourses increasingly inform the opinions and advice we receive in articles in the popular press, information on the internet, and discussions on radio and television. Incitements to care for brains, to think of and manage desires in certain ways, and to deploy pharmaceutical technologies towards such ends are not directed only at those who come to be associated with the specific 'disease of the brain' of addiction, but to individuals who could be at risk of developing such conditions – which is to say, anyone who has a brain and who experiences pleasure!

In relation to drug use, the NIDA *Brain Power!* program makes this clear: young schoolchildren are provided with the language and concepts of neuroscience to understand themselves in terms of their brain functioning. Knowledge of the molecular, neurochemical processes provides a basis for making choices about one's conduct and one's use of both licit and illicit substances. We may have trouble thinking of such imperatives of caring for one's brain as moral in the traditional sense – tropes of good and evil are a thing of the past in most educational literature today – but normative standards of health and well-being *are* embedded in the knowledge that students learn about themselves. These forms of judgment, of course, do not require absolute allegiance to an unquestionable set of transcendental principles: they are based on the veridical discourses that are subject to questioning, correction, and change. But the apparent decoupling of the political/moral from the scientific in biomedical principles about health and vitality is exactly what has inspired the present project.

Although much of the present work has focused on the biomedical research on addiction as a specific classification of brain pathology, it has also suggested that these developments have implications that extend beyond our understandings of illness, to more general understandings of ourselves. As addiction has come to be understood in neurochemical terms, all our desire – whether compulsive or not, whether for drugs or for behaviours – have come to be thought of as determined, to a significant extent, by what our brains reward us for. And within neurochemical styles of thought, any behaviour or activity that the dopamine system rewards may become *too* pleasurable. In the most extreme cases, other pleasures become relatively unrewarding, and relatively uninteresting; but in more moderate instances, our desires may merely compel us to make unhealthy or 'impulsive' decisions. At both of these poles, and in between, as desires have become newly intelligible, new possibilities have emerged for the governance of behaviours that are commonly associated with compulsive desire (e.g., sex, gambling, and eating), including regimes of pharmacological intervention. This is not an indication that our desires, even compulsive ones, are antithetical to projects of self-government and freedom, but quite the opposite: that even those aspects of ourselves that we experience as the most intensely personal and profoundly visceral have become embedded in such projects.

Ultimately, then, this thesis has consisted of an investigation of some of the new forms of knowing and caring for the self according to neurologic and pharmacologic rationalities that have begun to change how we think about ourselves and our desires as the subjects of regulation. By providing some insight into the historical contingency of how we have come to think and act as neurobiological human kinds, it is hoped that this work contributes to an expansion of the extent to which we can

think of ourselves, our biologies, and our experiences as spheres for potential contestation, political intervention, and reconceptualization.

POSTSCRIPT: EPIDEMICS OF PATHOLOGICAL DESIRE?

Among other things, my thesis described the ways in which new ways of scientifically studying addicted individuals have continuously produced new understandings of what addiction is, and in turn, have produced new perceptions (and self-perceptions) of the individuals under study. This drew, of course, upon elements of the work of Ian Hacking that focus on how expert classifications produce new slots into which certain individuals are made to fit, and the ways in which the study of individuals in such slots actually produces changes in the understanding of them. As Hacking puts it: "New sorting and theorizing induces changes in self-conception and in behaviour of the people classified" (Hacking 1995: 370). I suggested that biomolecular styles of thought about addiction, and the interventions associated with them, have made possible the formation of a new type of subject of addiction: a neurobiological kind of desiring subject.

And in relation to this, I argued that contemporary neurochemical styles of thinking about addiction have developed around a problematization of desire. This was not to say that desire has been constructed as a problem to be solved, and that an attempt was made to *eliminate* desire. It was to suggest the contrary: desire, in neurochemical models, came to be associated with the biological mechanisms which motivate and reward the most basic behaviours essential for human life and survival. Because desire came to be understood as a persistent, fundamental element of human existence, it was something that required apparatuses of management, not final solutions. What was required was a detailed, intricate knowledge of desire, which would provide the basis for subtle and delicate interventions into pleasures, desires, and motivations that had become dysregulated. (Interventions based on brute force, such as the chemical

destruction of neurons associated with pathological desires, were not an option.) Thus desire, which could never be eliminated, never excluded from the study of addiction, became the very *basis* of these studies. Desire, virtually coterminous with life itself, became the object of study for addiction scientists, and the subject of indefinite regulation.

And attempts to sort, classify, and treat desire led to a sort of looping effect of neurobiological kinds. I argued that naltrexone – which has been shown to enhance the capacity of individuals to manage their desires not just for illicit drugs, but for a range of other compulsions such as eating, gambling, and shopping – seems to be leading to a biological re-classification of behavioural compulsions and persistent desires as neurological conditions (and, in some cases, pathologies). I therefore concluded my thesis by suggesting that we are all starting to think of and act upon ourselves as subjects of neurobiological desire.

It was a fairly sweeping statement, but one which I thought was justifiable, since I had presented evidence that the incredible increases in knowledge about and control of desire as a neurochemical phenomenon has produced new opportunities for self-understanding and self-directed forms of action. But I could not help worrying a bit about the ground I was standing on: If we are starting to become neurobiological kinds of desiring subjects, shouldn't more of us be acting on our desires with receptor-targeting medications that can modulate the pleasure circuitry of our brains? Why aren't there any blockbuster anti-craving drugs on the market? Yes, it was true that trials were in place for using naltrexone to treat pathological gamblers and shoppers, but no scientists or pharmaceutical executives were predicting that such

interventions were going to become commonplace. Why aren't there, I wondered, desire-regulating lifestyle drugs that are used by huge numbers of individuals? I resigned myself to having chosen a topic which was valuable and interesting, but one which would not allow me to formulate claims about the emergence of a desire-treatment equivalent of an 'Antidepressant Era' (cf. Healy 1997) or a 'Prozac Nation' (cf. Wurtzel 1994).

Two days after I had completed my final draft, I received an e-mail from a friend with whom I had been out of contact for almost a year (until a couple days earlier when he had written to see how I was doing and how my thesis was coming along, and I had written him back, including a few details about my project). In the e-mail, the friend informed me he had recently gotten a job with an advertising agency that dealt with the pharmaceutical industry, and that he had been assigned to a project that sounded like it might be related to my research:

> if you are interested in medications, such as ReVia, you have to check out this future drug (it is going to be a mega blockbuster—it is in phase III) Acomplia (Rimonabant)—it is a smoking cessation/weight loss drug (while it may be marketed as something different)...I think you will find it very interesting... It has got such a Buzz around it, before I started working on the drug I saw a news article on the nightly news...and last Sunday it was the topic of the TV show Boston Legal...[21]

Not having heard of the medication, I was intrigued, and turned first to the internet, and then to media reports to find out the story of Acomplia.

The coming Age of Acomplia?

It was unclear to me, at first, how some of the information which turned up from my search for information on Acomplia related to the medication my friend had

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[21] Anonymous, personal communication, 1 February 2005

mentioned. Instead of information about a specific drug, I read about the fact that there had recently been a shake-up in the corporate world of 'Big Pharma.' In April of 2004, a relatively small French drug company, Sanofi-Synthélabo surprised many when it successfully completed a hostile takeover of the considerably larger French-German firm Aventis. The result of the $65 billion merger was the birth of Sanofi-Aventis, now the third-largest pharmaceutical company in the world (behind Pfizer and GlaxoSmithKline).

The motives behind this merger were initially unclear. Before the deal had been completed, most commentators seem to have attributed the action to the goal, shared by both of Sanofi-Synthélabo executives and the French government (who have been accused of 'all but stage-managing' the takeover, which had been delayed by court challenges, and preventing a similar takeover attempt by Switzerland's Novartis), to create a national pharmaceutical industry champion for France. However, a few months later, the new pharma giant released information about a pipeline drug which, analysts have suggested, could become a once-in-a-decade 'blockbuster' drug. Only after this information was released did it become clear that a key motivating factor in the Sanofi-Aventis merger was the fact that Sanofi was not capable of marketing a drug that had such massive sales potential:

> "We needed more muscle to sell such a product," Gerard Le Fur, the company's chief scientist and No.2 executive, said in a telephone interview from Paris. "We knew the first results of the trials back in January. Knowing that, it was more or less linked to the deal" (Landler 2004).

Prior to the merger, Sanofi had relied on Bristol-Myers to market two of its best-selling products in the US – an agreement that significantly reduced Sanofi's US profits. Now, however, should Acomplia be approved, Sanofi-Aventis will be able to

deploy its own American marketing force, keeping all the return on a product analysts predict might generate up to $6 billion a year in sales by the end of the decade, and become the best-selling drug in the world.[22]

Acomplia is predicted to be such a massive seller because it has the potential to be prescribed in the management of two conditions identified as leading causes of death in the industrialized world: obesity and smoking. In human clinical trials involving smoking subjects on Acomplia were reported almost to double an individual's chances of success at stopping smoking. (While the effectiveness of Acomplia in smoking cessation is comparable to other pharmacological therapies, it has the advantage of almost eliminating the weight gain frequently associated with quitting smoking.) In obesity trials, subjects lost an average of more than 8 kilograms. In these subjects, levels of 'good' cholesterol rose and 'bad' varieties declined significantly. Moreover, the weight lost tended to be the deep abdominal fat associated with increased risk of heart disease and type II diabetes. Unlike other anti-obesity drugs on the market in the US, which has such troublesome side effects as 'anal leakage' and elevated blood pressure, Acomplia's side effects appear to be relatively minor.

How does Acomplia achieve what appear to be such remarkable results? (And: how is all of this relevant to my thesis?) The title of a USA Today article gives us a clue: "One pill a day could keep food and nicotine cravings away" (Sternberg 2004).

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[22] The NY Times article reports: "Marc Booty, a pharmaceutical analyst at Commerzbank Securities in London, said that if Sanofi could sell Acomplia to 5 percent of the obese population in 2010, at a price equal to that of standard anticholesterol drugs -- which can range from about $2 to $4 a pill -- it would generate 5 billion euros, or $6 billion, a year."

Acomplia is remarkable because, as an obesity treatment, it is "the first in an entirely new class of drugs that affect a pleasure center in the brain" (Capell and Matlack 2004). Acomplia works not by acting on diseases themselves (e.g., through the reversal of heart or lung disease) – or even the physiological conditions considered risk factors for diseases (e.g., obesity, elevated cholesterol) – but by reducing the cravings that produce the behaviours associated with such risks. It does this by selectively targeting a specific type of receptor associated with the reward circuitry of the brain: the cannabinoid receptor.

Although Acomplia acts on the endocannabinoid system, the neuroscientific and psychopharmacological principles relating brain chemistry, desire, and conduct are essentially similar to those of naltrexone, discussed in depth in my dissertation. Desire for certain things (particularly food, but also nicotine – as well as alcohol, some evidence is suggesting) is caused in part by the activity of cannabinoid receptors, which belong to a system normally kept in check by other parts of the reward system. However, a dysregulated brain reward system can produce too many of the endogenous cannabinoids that, like the psychoactive components of marijuana, create powerful food cravings. Steve Alexander, a Nottingham neuroscientist researching cannabinoid receptors explains: "The brain processes you undergo as you eat nice food are very similar to what you do when you smoke cannabis or inject heroin" (quoted in Adam 2004). Acomplia occupies the cannabinoid receptors and prevents them from producing cravings.

There is little need to revisit the historically contingent factors on which these understandings, and this treatment, depend, for they are analogous to those already

discussed in the preceding work. Nor do I think we need to ponder long over whether we should be thinking about Acomplia as a treatment for a disease or epidemic of the will. As a fairly simple content analysis of media reports suggests, Acomplia is thought to be effective because it targets desire, not will-power: Of 98 articles published between April 2002 and January 2005, the term 'will-power' (or 'will power,' or 'willpower') appears only nine times. In six instances, the term appears within contexts which specifically refute the position that the behaviours targeted by Acomplia are related to will-power. For example, an obesity specialist at Addenbrooke's Hospital, Cambridge, was quoted as saying: "It is not true to say that people who are obese have no will power [...]. My patients have plenty of will power. They have lost 10 times more weight than other people but they cannot keep it off" (quoted in Hall 2004). Two mentions of will-power are made in neutral or unclear contexts (once the suggestion is made that Acomplia is useful as a craving-reducing treatment is useful when will-power alone can not control behaviour; and once the question is asked: 'what ever happened to will-power?'). In only one instance within 98 articles is the suggestion made that Acomplia acts on will-power. In contrast, the term 'craving' (or 'cravings') appears 59 times.[23]

But the case of Acomplia does seem potentially to add another dimension to the broader social, economic, and cultural implications of the constitution of ourselves as subjects of neurochemical desire explored in my thesis. In many ways, what I had investigated was a relatively isolated style of thinking about addiction and desire, which, had only begun to extend into popular culture, public institutions, and private

[23] This analysis relies on data gathered through a methodology that can only be considered rudimentary: A LexisNexis search was made for articles containing either the term 'rimonabant' or 'acomplia' in 'major world publications', with no date parameters. The search returned 98 articles, published between 8 April 2002 and 10 January 2005, all of which were included as data.

practices. The material on Acomplia, which I have only briefly described, seems to suggest that this may soon change – or, rather, that the change has already begun. Acomplia is currently under consideration for approval by the US Food and Drug Administration, as well as by other national regulatory agencies. If it is approved, and if we assume that the market potential of Acomplia – for which Sanofi-Aventis seems to be putting a lot at stake – will result in a new 'awareness' of the extent and incidence of problematic desires in our lives, it seems to raise the question: may we, in the not-too-distant future, come to encounter something like an 'epidemic of pathological desire'?

ADDENDUM

This thesis has developed an analysis of the neuroscience of addiction that begins from the assumption that all of the molecular facts we have about our brains and our desires are to some extent social; that is, that the science of addiction is inextricable from social relationships, cultural conditions, and societal institutions. As an examination of the socially contingent development of both our knowledge of the brain and our capacities for acting on ourselves (and others) in terms of our neurobiology, this study has contributed insights into some of the most fundamental issues that the discipline of sociology has grappled with since its inception. For example, the thesis has examined some of the ways in which politics and culture influence the production of knowledge; the ways in which individuals come to understand themselves as particular kinds of subjects (here, focusing on the emergence of the 'neurochemical self'); and the changing ways in which particular forms of deviance (such as drunk-driving) are explained and controlled. But while this study focuses on many traditional sociological topics as they relate to the subject of addiction, it has attempted to avoid depending upon traditional sociological theorizations of addiction. Although some of the reasons for this were discussed in its introductory material (especially Chapter 2), it might be worthwhile, as a final conclusion, to reflect on some of the theoretical and methodological tools deployed in this thesis in light of the findings presented in the preceding analysis.

In part, this study emerged out of an engagement with social constructionist analyses of addiction treatment, and especially those Foucauldian-inspired strains of constructionism that rely on analyses of expert discourses and scientific texts. Since the 1970s, and in the wake of the anti-psychiatric movement (in which some key

figures, such as Thomas Szasz, dismissed the existence of addiction altogether), sociologists have tended to focus on the arbitrary standards and taken-for-granted ideals of health and functionality that addiction medicine often relies upon. For example, they have noted that while addiction is generally *explained* in terms of biological factors (genetic influences, physiological processes, etc.), it is most always (i) diagnosed in terms of impairments in social functioning (such as problems at work or in interpersonal relationships) or undesirable subjective feelings (e.g., guilt, shame, or loss of control), and (ii) treated with the goal of restoring social functioning and personal capacity.

However, this thesis avoided merely applying a standardized Foucauldian framework to addiction neuroscience – one which might, for example, argue that addiction neuroscience is best analyzed as a form of knowledge/power that is essentially political in nature, and is best understood as the result of efforts to establish new programs of social and medical discipline over individuals' bodies and desires. While Foucauldian analyses of addiction medicine have usefully highlighted the sociopolitical aspects of addiction treatment by demonstrating that addiction treatment may function as a form of social control, they have generally left unexamined the scientific and technical content of addiction science. This reflects, perhaps, the assumption held by many constructionist sociological analyses of addiction medicine that science is not more objective or robust, but only more dominant, than other types of knowledge. Such an assumption tends to imply that addiction lacks a 'real,' biophysical basis – or at least one that warrants careful sociological consideration.

My thesis sought to develop an analytic framework which could provide an account of the development and impact of addiction neuroscience (and the accompanying partial re-definition of ourselves and partial re-configuration our social and political lives): one which did not dismiss questions of biology altogether, or reduce biological explanations to exclusively social or political factors. It sought to take the neuroscience of addiction seriously, and to describe the contingent conditions making the rise of such understandings and instruments (at particular times and locations) possible. In order to do so – to reach a more fertile ground for the sociological investigation of the science and technologies of addiction medicine – the thesis sought to acknowledge that science can produce real facts, and can do so objectively. But of course, the thesis's use of the terms 'facts' and 'objectivity' differs significantly from the ways these terms are used in standard positivist, progressions discourses.

Drawing on the work of Ludwik Fleck and scholars he has inspired, the study considered facts and objectivity as things that exist – but exist as social events that are not independent of historical and cultural contexts. It therefore examined facts and criteria of objectivity not as if they could be expected to stand on their own, in a realm of pure reason and truth, but within the concrete contexts of their actual production and reproduction. These contexts involved local networks in which ideas and practices circulated, material and institutional factors that made research possible, and the scientific and political contests in which rules for objectivity were decided upon.

The methodological choice made for this study – which was to rely exclusively on archival research and textual analyses – should not be taken as evidence that, despite the claims otherwise, the study upheld the view that medical disciplines are simply

discursive or 'ideological edifices', or that the thesis thereby underestimated the broad range of (non-discursive) social, personal, and political issues at stake in contemporary understandings and practices of addiction medicine. This study was not designed as a traditional form of 'discourse analysis' that predominantly focuses on the technical rules, semiotic principles, and rhetorical structures that appear in the official discourses of addiction science. The range of materials examined extended well beyond official scientific texts, to include many forms of popular and less technical science that appear in public education materials, media stories, interviews, legal codes, and online patient discussion groups. Moreover, both expert and lay texts were analyzed not simply in terms of their internal logics, but also in terms of the social considerations, political rationalities, and practical and historical contexts to which they related.

The resulting analytical frame, which might be described as 'post-Foucauldian' or 'neo-Fleckian', was used in this thesis to integrate a careful, detailed investigation of the production of scientific knowledge (taking cues from studies of science and technology that focus on micro-levels and local settings) with a fairly wide-scoped account of the socio-historical antecedents and implications of medical practices (of the nature offered by the best Foucauldian-inspired scholarship). Although the thesis sought to develop and apply this framework only in relation to a concrete field of investigation (rather than to evaluate the framework's potential in abstract or theoretical terms), the resulting analysis demonstrates, I think, that this approach has potential for thinking sociologically about the incredible complexity and novelty of contemporary neuroscience – not only as a form of isolated academic discourse, but as a form of knowledge which has practical implications within clinical settings.

Undoubtedly, further insights would emerge if this analysis was supplemented with research using other methods. Ethnographic studies could certainly contribute valuable insight into the findings presented. Participant observation in scientific laboratories, as well as in the manifold settings in which neuroscience models of and treatments for addiction are deployed, could add another dimension to the present study. However, while an ethnography of a laboratory or treatment program would likely have offered a thicker description of some aspects of scientific activities or patient experiences, it would have failed to offer the social and historical scope that documentary methods make possible.

With unlimited resources, a multi-sited ethnography might render more complex the picture offered by the findings of archival research. However, given the financial and temporal constraints that come with a sole-authored PhD thesis, such a study was not possible. Instead, what was aimed for, and what the thesis offers, is a means of engaging with and rendering intelligible the transformations in systems of thought and practice concerning addiction neuroscience, and a preliminary analysis of some of the multi-faceted ways that neurobiological styles of thought are changing patient experiences, treatments, legal procedures and practices relating to addiction. Certainly, one of the conclusions to be drawn from this thesis is that the social and personal transformations being brought about by the contemporary brain sciences of addiction warrant further investigation with a range of sociological methods – including not only ethnography, but also comparative analyses between different national contexts, and also different patient groups.

To the extent that they could provide rich, descriptive accounts of the lived experiences of social actors as they react to, interpret, and differentially adopt (or, indeed, reject) the theories and treatments of contemporary addiction neuroscience, ethnographic and comparative methods would also likely demonstrate that even when such cutting edge discourses are accepted as legitimate, the result is seldom lead to a complete revolution or paradigm shift in any individual's or group's conceptualization of addiction. But of course, this insight would stand in contradiction to any claims made in this thesis, which did not have as its goal an analysis of 'thought on addiction' as a whole (as if there could ever be a single way of thinking about addiction within a culture). The starting point for this thesis was not to ask, 'has thinking about addiction, within industrialized societies of the North Atlantic, shifted to a neurological style of thought?', because such a question could be answered, quite easily, with a simple 'no'. Over the last decade or so, analyses of addiction within sociology and cultural studies have convincingly demonstrated that addiction has long been (and remains) a hybrid entity, and that different addiction treatments (such as Alcoholics Anonymous, Narcotics Anonymous, and other twelve-step programs) continue to draw upon a range of medical, moral, spiritual, and psychoanalytical precepts and tools.

As already indicated, the original motivations for this thesis in fact developed in relation to concerns about sociological and cultural studies of addiction, which, to the extent that they excluded a consideration of addiction neuroscience from their analyses, seemed to imply that the developments within the brain sciences, as they relate to the understanding and treatment of addiction, were not significant. Such work seemed to beg a number of questions: *What was the state of the molecular brain sciences of addiction? Had these sciences altered the field of inquiry into addiction – that is, had*

they actually changed the subject matter of 'addiction' research? Were new rules about what counts as acceptable experiments, truth claims, theories, and so forth emerging? Preliminary research for this project made it clear that neuroscience has indeed come to be seen, by a range of actors concerned with drug use and addiction (e.g., researchers, clinicians, patients, government agencies), as providing new ways of understanding and dealing with compulsive behaviours and desires. This finding seemed to raise important, hitherto uninvestigated questions about the moral, ethical, and social implications of addiction neuroscience: *Were neuroscience models and treatments changing the ways in which we think about the self as it relates, within programmes and strategies of social regulation, political domination, and ethical judgment, to desire and conduct? Did the 'addict' as a type of person, or form of subjectivity, disappear (or become reconstituted along different lines within these programmes and strategies)? In molecular discourses, to what extent are individuals considered 'at fault' for their condition – or for conduct that is attributed to their condition? In what ways are individuals, as well as other actors or agencies, held responsible for dealing with the condition within such styles of thought? What new forms of treatment and control have arisen on the basis of molecular conceptions of addiction?*

Archival research and textual analysis seemed a more appropriate means of beginning an investigation of these sorts of questions than ethnography; not because local, individual, and heterogenous experiences and understandings are less worthy of sociological investigation than official and public discourses, but because the ways that persons come to understand their selves and their conduct at a particular historical moment are linked to the formation of systems of thought and programmes of intervention that are *not* only local. It would be an interesting and worthwhile

undertaking to document the processes of self-reflection, introspection, and negotiation through which individuals come, to greater and lesser degrees, to know themselves through neuroscience, and to form notions about proper and improper conduct (as well as governing strategies for that conduct) on the basis of that knowledge. But the goal of this thesis was not to offer an account of *how* individuals come to reinterpret their experiences, desires, lives as being based in neurochemical events; it was the more modest goal of understanding how they might become *able* to do so.

Such an undertaking, it seemed to me, required both an examination of the historical roots of contemporary understandings of addiction and the conditions under which these became possible, and also an analysis of how addiction neuroscience becomes relevant to the management of social, political, and personal life in the present day. The latter half of the thesis in particular focused on describing how new ways of thinking about the self and controlling one's conduct which are based on neuroscientific styles of thought have been made available, in a range of settings, to non-experts. The materials of media reports, legal protocols, and public health programs were analyzed in order to gain a sense of what images, symbols, rational concepts, and attitudes have become associated with addiction in non-specialist discussions of neuroscientific discoveries and advances. An implicit assumption of the thesis is that these sources are crucial in providing an informational backdrop to our lives, containing many implicit and explicit messages about the body and health which not only influence policy-making, but also perceptions of self-worth, hopes for the future, and understandings of the very nature of disease. Since the goal was to understand how present-day debates about and experiences of addiction are becoming amenable to transformation as a result of molecular and pharmacological sciences, the

analysis attended to a variety of portrayals of addiction science and treatment in public culture.

In its efforts to highlight the novelty of the changes in scientific styles of thinking about addiction, and in its attempt to portray the neuroscience of addiction as worthy of serious consideration, parts of this thesis may appear in danger of either uncritically accepting some of the claims about addiction made by the contemporary neurosciences, or of overstating the influence and impact of these sciences. However, as was asserted in the opening chapters, the thesis focused on describing addiction *only as it is conceptualized within a particular, emerging style of thought*, and its analysis was written with an intended ontological ambivalence that both endorsed and questioned neuroscience theories: endorsed, insofar as it set up addiction neuroscience as having an objective basis that warrants careful attention; and questioned, insofar as it argued that that objective basis was historically, politically, and culturally contingent.

This ambivalence sometimes resulted in statements about addiction, and the neuroscience style of thought, that make a somewhat ambiguous (and unconscious) use of irony. For example, on page 84 above, a sentence states: "Today, addiction is a disease of the brain". The fact that the sentence was written that *today*, addiction is a disease of the brain, suggests that addiction has not always been, and need not necessarily be, a disease of the brain. While this sentence (and others like it) was intended only as a description of a historically contingent belief, it could be read out of context as an ontological proposition being made by the thesis. This would, however, be an erroneous interpretation, since the study did not make claims about the nature of addiction (but instead, only interrogated how addiction and desire are thought into being within the specific discursive and practical spheres of molecular biomedicine).

Ultimately, the purpose of analyzing the development and implications of addiction neuroscience was not to support a claim that 'we are all now neurochemical selves', in the sense that all human beings, everywhere and at all times, think and act upon themselves in terms of their brain chemistry. It was only to elucidate and reflect upon the new possibilities for thought and action that the neuroscience of addiction has begun to bring about, and to explore some of the consequences these possibilities may have on how individuals understand themselves, their behaviours, and their desires. This study was not intended to suggest that these new forms of knowledge and practice are ubiquitous, or that they are universally subscribed to; it was instead to describe the emergence of what, following Ian Hacking, we might call a new 'human kind' (cf. Hacking 1995). The 'neurobiological human kind', which the theories and models explored in this thesis represent, provides new descriptions of people, of their emotions, and of their behaviours – and also new means of acting on individuals, feelings, and conduct. But it does not supplant all other kinds of understanding, and indeed, it may co-exist with other systems of thought. Thus, the argument upon which this thesis has been based is not that, following the emergence of neuroscience understandings of compulsive desire, addiction is no longer a hybrid entity; it is the more modest proposal that the nature of this hybrid has begun to change, with potentially significant consequences that warrant careful sociological attention.

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