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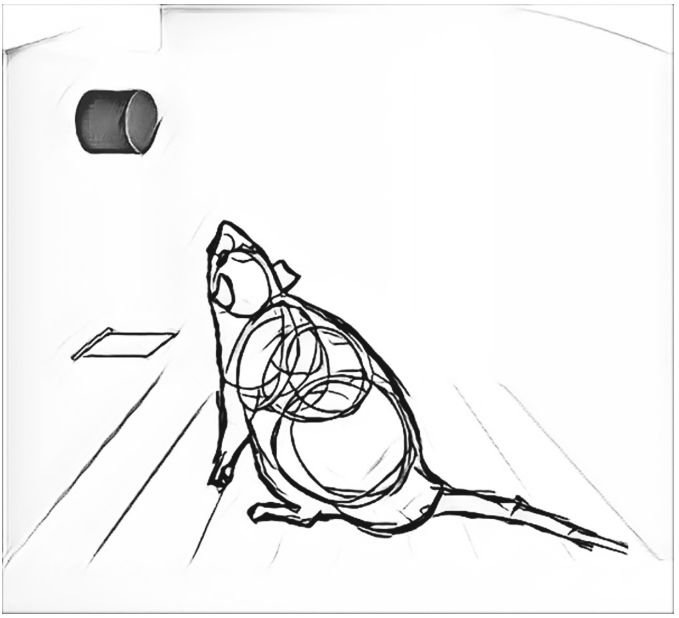
# Introduction

WHAT WOULD YOU DO alone in a cage with nothing but cocaine? In a seminal study published in 1985, the animal experimentalists Michael Bozarth and Roy Wise asked this question of rats.[1] The rats learned to press a lever to get a dose of cocaine, delivered immediately and intravenously. They were then permanently housed in an experimental chamber containing only food, water, and the lever, which they could press for as much cocaine as they wanted. You will not be surprised at the answer to the question. The rats in this experiment took a lot of cocaine. They also stopped eating and drinking. Within a month, 90 percent had died, from exhaustion, starvation, and dehydration. This image of a rat in a cage pressing a lever again and again—abandoning itself to cocaine at the expense of food and water—is strikingly evocative of the nineteenth-century writer Oscar Wilde's chilling description of human addiction: "Men and women at such moments lose the freedom of their will. They move to their terrible end as automatons move."[2]

The rat relentlessly pressing the lever, the human whom drugs have made into a walking zombie—these are the poster children of the currently dominant scientific paradigm of addiction, which sees it as a brain disease causing compulsive drug use. In 1984, the biological psychiatrist Nancy C. Andreasen published her book *The Broken Brain*, capturing in that turn of phrase a way of thinking about mental disorder that had been gaining popular and scientific ground since the nineteenth century, buoyed by progress in brain-imaging techniques and increasing use of animal experimentation.[3] In 1997, Alan L. Leshner, then director of the US National Institute on Drug Abuse (NIDA), published his seminal article "Addiction Is a Brain Disease, and It Matters," cementing a broken brain model of addiction as scientific orthodoxy.[4] Since then, the brain disease model has grabbed the spotlight, claiming by far the lion's share of funding and credibility, and putting neuroscience and animal



models at the center of addiction research. Dissent is heresy. Junior scientists would be foolish to question the paradigm, while senior scientists have built their labs and legacies within it.

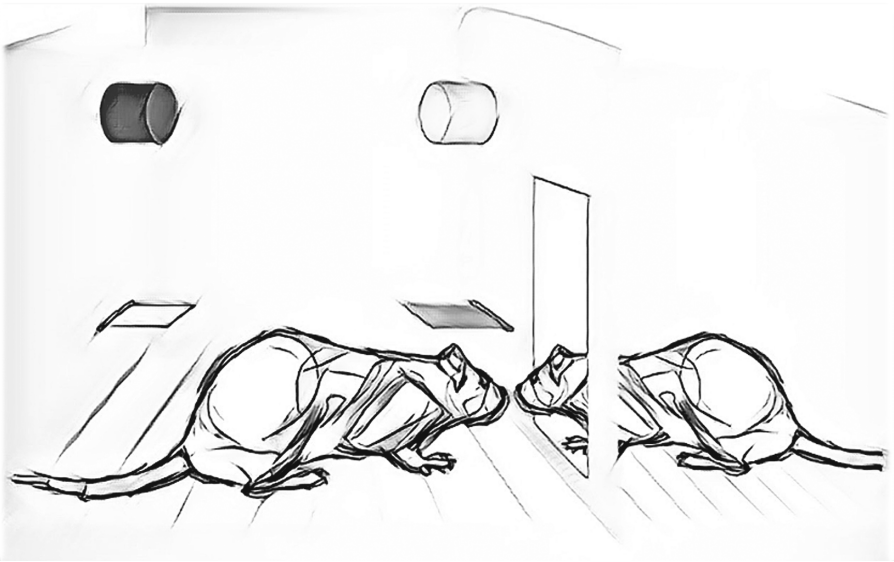
The core idea of the model is as gripping as it is familiar. After prolonged drug use, "a metaphorical switch in the brain" is flicked and the brain is "hijacked" by drugs.[5] In keeping with Wilde, the brain disease model treats addiction as occurring when the kind of freedom of will that we normally take for granted is lost: The ordinary, voluntary behavior of a rat or a person—pressing a lever, downing a drink, swallowing a pill, injecting, inhaling or snorting a substance—becomes compulsive, transformed into a passive, involuntary symptom of a brain disease.[6]

Yet hidden behind the party line, models of addiction within neuroscience—as well as models stemming from adjacent disciplines and those at greater remove—are multiplying and competing, fomenting disagreement about something as basic as what addiction is (see chapters 1 and 2). Meanwhile, translational results issuing from the brain disease model—that is, the discovery of new and effective clinical treatments, which virtually all scientific funding applications claim as their justification and aim—are shockingly meager.[7]

The most effective medications for opioid addiction are methadone and buprenorphine. These are themselves psychoactive opioids, typically taken in liquid form or as tablets or strips that are dissolved in the mouth. Together with naltrexone—an anti-craving drug used for both opioid and alcohol addiction—they were discovered in the 1960s and 1970s, before the ascendency of the brain disease model.[8] The go-to pharmacological treatment for alcohol addiction is disulfiram, marketed under the brand name Antabuse, and discovered in the 1940s.[9] It produces a host of unpleasant symptoms if combined with ethanol and so discourages drinking.

Perhaps even more surprisingly given the dominance of the brain disease model, the most effective treatment for cocaine and some polydrug addictions is not pharmacological but behavioral. Contingency management treatment reliably delivers small rewards, such as prizes, money, and vouchers for meals or movies—or, in the behavioral psychologist Kenneth Silverman's inspired innovation of a *therapeutic workplace*, skills training and employment—on condition of drug abstinence.[10] It is based on operant conditioning principles, a form of reinforcement learning originating in the psychologist Edward Thorndike's discovery of the law of effect at the end of the nineteenth century, and elaborated by B. F. Skinner in the 1930s and 1940s.[11]

These principles are in fact the foundation for a series of animal experiments conducted over the past two decades that require reinterpretation of Bozarth and Wise's 1985 seminal finding. In 2005, the animal experimentalist Serge H. Ahmed published the first results from a forced-choice rodent study paradigm. Ahmed pioneered a simple but ingenious innovation. He introduced a second lever into the experimental chamber, thereby making it possible to give rats an either-or choice between cocaine and an alternative reward. Over a series of studies, he and his colleagues found that, in controlled conditions, when given a choice between pressing a lever for cocaine or pressing a lever for water sweetened with saccharin, 90 percent of the rats—including those who showed every indication of addiction-like behavior and none of whom was water or food deprived—chose the saccharin water, forgoing cocaine.[12] Then, in 2018, the animal experimentalists Marco Venniro, Yavin Shaham, and their colleagues published findings from a series of studies that extended Ahmed's paradigm by switching the alternative from saccharin water to social reward. They found that, when given a choice between pressing a lever for methamphetamine or heroin or pressing a lever to get one minute of playtime with another rat, almost 100 percent of the rats—including those who showed every indication of addiction-like behavior and none of whom



was socially deprived—chose to press for playtime with the other rat, thereby forgoing the drug.[13] Indeed, in a follow-up study, it transpired that rats will even forgo cocaine for the lingering scent of another rat in an empty experimental chamber.[14] So much for the idea that rats are compelled by their cocaine-broken brains to keep pressing the lever for more, when all it takes to lure them away is a drink of saccharin water, a minute of playtime, or the whiff of a buddy (see chapters 6 and 7). Why then did they take so much cocaine in Bozarth and Wise's 1985 experiment, when they were alone in a cage with nothing but cocaine? Presumably, because they were alone in a cage with nothing but cocaine.

The currently dominant scientific paradigm is broken. It is time for heresy.

Although I have collaborated with addiction scientists and animal experimentalists, I am not one. I am a philosopher. But I also spent a decade from 2007 to 2017 working part-time as an assistant team therapist in the United Kingdom, in a National Health Service (NHS) therapeutic community for people with personality disorder and complex needs, many of whom struggled with drug and behavioral addictions. UK therapeutic communities (TCs) are distinctive care environments, different from what often falls under this label in the United States as well as from more conventional medical contexts. They are informal and nonhierarchical, requiring genuine, sustained relationships

between clinicians and patients, as well as between patients themselves. Medication is of course part of treatment if appropriate, but a great deal of time is spent not merely in therapy together but as a community: cooking, eating, playing games, going on outings, running the business of the group. Relationships are considered the crucial mediators of psychological and behavioral change and recovery, and thus integral to therapeutic success. Indeed, at the therapeutic community where I worked, patients typically arrived at our door taking a staggering number of medications that did them little if any good while having many negative side effects, which we then worked to help them come off. We also typically helped those patients whose identity revolved around having a brain disease to reflect on its meaning in their lives—what it did for them and how it limited them. A willingness at least to question, if not indeed abandon, this identity—and, with it, the idea that fundamentally their problems stemmed from a broken brain—was often an essential step toward recovery. So too, unquestionably, was the social support and sense of belonging provided by the group.[15]

I begin this book with the juxtaposition of experiments with rats and my own experience of working in a therapeutic community—an experience that is certainly atypical for a philosopher, but atypical even for clinicians—because where ideas come from matters. I have spent years studying addiction by drawing on a wide range of perspectives, including animal models but also neuroscience, cognitive science, social psychology, cultural anthropology, behavioral economics, public health, psychiatry, law, literature and addiction memoirs—all of which inform this book. But my understanding of addiction is both anchored in philosophy and has its origins in my own experience of working clinically with people with personality disorder in a therapeutic community in the UK. That is, with a patient group whose primary diagnosis is most certainly considered a mental disorder, but is not considered a brain disease, and in a clinical context where the primary orientation to understanding and addressing mental disorder is not neurobiological, but social and psychological. No surprise, then, that I am a broken brain heretic. I believe we need a new paradigm for understanding addiction that jettisons what is wrong with the brain disease model but retains what is right. This book hopes to provide one.

The paradigm I develop in the pages that follow can be distilled into two core and connected characteristics: *humanism* and *heterogeneity*. Although it will take a book to complete the presentation of the paradigm, I introduce these characteristics here. I also begin the task of uprooting some of the

dogma, fear, and moralism that infects so much of our thinking about drug use and addiction—a clearing of the ground, so to speak, so the paradigm I go on to present can be considered with a more open mind.

To begin, then, with humanism. What I mean by this idea can be summed up in a single, foundational principle for explaining human behavior. *Psychology first.*

Psychology is our most basic, powerful tool for understanding ourselves and others.[16] Humans are self-conscious and self-reflective beings. We understand ourselves to act for reasons, both good and bad. We take our actions to be explained by our thoughts and feelings, beliefs and desires, pleasures and pains, hopes and fears, plans and intentions. In other words, we take our actions to be explained by our psychological states. We act in order to express, communicate, and satisfy our psychological states: to show what we think and how we feel, to get what we want, to avoid what we don't, to further what we hope for and make good what we intend. We begin to master the art of psychological explanation in the cradle, and we carry it with us to the grave. But it is neither fixed for all humans for all times, nor simple and easy to codify.

On the one hand, the psychology that we use in our day-to-day lives shifts and deepens as we study it. Philosophical and scientific psychology can come to inform ordinary psychology. This happened in our culture at large with Sigmund Freud and the advent of psychoanalysis, B. F. Skinner and the coming of the behaviorists, and Noam Chomsky and the cognitive revolution; it happens if you read (or write) an article or a book, and rethink or refine your individual understanding of some aspect of our minds that you previously hadn't given much attention to.

On the other hand, to be powerful and generative, the psychology that we use in our day-to-day lives must take not only individual history and personality into account, but also the social, cultural, and economic circumstances within which each person finds themself and fashions a life. Psychology and psychological explanation are inextricably linked with life circumstances. This is why pointing out that a rat is literally alone in a cage with nothing but cocaine (or, as we shall see in some of the chapters that follow, that this is metaphorically true of a person) can be explanatory of why the rat (or the person) might take a lot of drugs. We imagine the psychological impact of being trapped in isolation and emptiness; we imagine the boredom, the loneliness, the misery and the suffering. And we note that, in this cage, literal or metaphorical, there is only one thing that offers any relief. *Cocaine.*

If you pick up any addiction memoir or listen to how people with addiction speak about it, you cannot help but see that, on the whole, they both describe their use of drugs in psychological terms and weave it into the story of their lives. Here is how the writer and journalist Pete Hamill introduces his memoir *A Drinking Life*:

> This is a book about my time in the drinking life. It tells the story of the way one human being became aware of alcohol, embraced it, struggled with it, was hurt by it, and finally left it behind. The story has no hero.
>
> The culture of drink endures because it offers so many rewards: confidence for the shy, clarity for the uncertain, solace to the wounded and lonely, and above all, the elusive promises of friendship and love. From almost the beginning of awareness, drinking was a part of my life; there is no way that I could tell the story of the drinking without telling the story of the life. Much of that story was wonderful. In the snug darkness of the saloons, I learned much about being human and about mastering a craft. I had, as they say, a million laughs. But those grand times also caused great moral, physical, or psychological damage to myself and others. Some of that harm was probably permanent. There is little to be done now but take responsibility.[17]

As Hamill does in his memoir, people with addiction tend to speak of when and why they first started using; where they use; who they use with; what makes them want to use; what drugs do for them; what drugs mean to them; how they crave them, love them, need them, hate them; how they feel when they use; how they feel when they don't use; how they feel about the fact that they use; and even, for some people, how their use is part of their identity—their sense of who they are. Often, although not always, they also express uncertainty about the brain disease model, which sits uneasily with their own experience of addiction.[18] This idea—of a psychological relationship to drugs and how that relationship is linked to a person's life circumstances and identity—is crucial to understanding and treating human addiction.[19] It is explored in many of the chapters that follow (see especially chapter 4, and parts III and IV).

Two clarifications of the *psychology first* principle are in order. First, psychology *first* does not mean psychology *exclusively*. On the one hand, even when psychological explanation is successful, both neuroscience and cognitive science can supplement it, identifying physical states and computational processes that underpin psychological states and psychological processes. But

supplementation is not the same as supplantation. The philosopher and ex-addict (his preferred term) Owen Flanagan puts the point thus in his book *What Is It like to Be an Addict?*: "When we identify genes behind susceptibility or brain circuits that subserve craving we explain susceptibility and craving, we don't explain them away."[20] Nor does an appeal to neuroscience and cognitive science to supplement psychological explanation depend in any way on the presence of pathology—understood in the most basic sense as the idea that something is neurobiologically or cognitively *wrong* (see chapter 8). Studying the brain at either the neurobiological or the cognitive level can illuminate addiction—as it can illuminate all forms of human behavior—whether or not addiction is a brain disease. Put otherwise, the fact that neuroscience and cognitive science can contribute to an understanding of addiction in no way shows that addiction is a brain disease—on pain of the consequence that all human behavior is a brain disease.

On the other hand, when psychological explanation fails—when we simply cannot make sense of ourselves and others in psychological terms—neuroscience and cognitive science may sometimes step in and take over, supplanting rather than supplementing psychology. Indeed, some people with addiction experience a kind of self-opacity—a failure, at least some of the time, to fully understand themselves or to be able to explain their drug use in psychological terms without remainder—inviting us to look beyond psychology and life circumstances for explanations (see chapters 9, 14, and 15). But if you want to understand a self-conscious and self-reflective being, the place to start is to consider how they understand themself. This is as true of a person in the grip of addiction as of anyone.

Second, psychology *first* does not mean treating what people say about themselves as infallible or beyond question. No doubt, there are aspects of our minds—most notably, what our current conscious experience is subjectively like for us—where we have a great deal of authority. But we can all be wrong about why we do what we do—about what explains our behavior—even if we have made serious and sincere efforts at self-understanding.[21] There are many reasons why this is true in general; here are two reasons that bear on addiction in particular. The first is the possibility of denial and self-deception: psychological processes whose function is to block self-knowledge. This possibility can at times provide a reason to question what people with addiction say about their drug use, both to others and to themselves. This fallibility does not, however, require us to abandon psychology for neuroscience or cognitive science. Quite the contrary. Denial and self-deception lie at least partly outside of our

conscious awareness, but they are fundamentally psychological processes that serve to protect us from the pain of facing up to certain truths (see chapter 13). They are psychologically motivated. And it is typically through a conscious, painful process of psychological reflection that they are overcome. The second is that, as the philosophers and qualitative researchers Doug McConnell and Anke Snoek have emphasized in their work on addiction, culture shapes self-understanding by providing us with interpretive tools: ideas, tropes, schemas, archetypes, narratives.[22] If these are misguided—as I believe the brain disease model in many respects to be—then so too is any self-understanding shaped by them.

The paradigm proposed in this book is therefore humanistic in that it aims to counter the brain disease model's persistent sidelining of psychological explanations of addiction in favor of explanations that are both neurobiological and pathological. The paradigm does not thereby reject the scientific study of addiction: The supplementation of psychology by neuroscience and cognitive science can be important and illuminative. Nor does it pretend that self-report is infallible. It does, however, thereby raise a serious concern about the scope of the relevance of animal models to human addiction[23]—one of the experimental methods most central to addiction neuroscience, and from which I, myself, have learned a great deal. For nothing about a person's own understanding of their relationship to drugs and the role of this understanding in forming and maintaining their addiction can be modeled in animals who, like rats and most other nonhuman animal subjects, are not self-conscious or self-reflective beings.

Let us now turn to the second core characteristic of the paradigm: heterogeneity. What I mean by this is that there is no one-size-fits-all explanation or theory of addiction.

Different cultures sanction and condemn different drugs. For example, in our society, alcohol and prescription drugs are generally culturally sanctioned, while MDMA (methylenedioxymethamphetamine—colloquially known as molly or ecstasy) and heroin are generally culturally condemned; but other societies condemn alcohol, while some subcultures within our society sanction MDMA and heroin. The importance of cultural attitudes to how and why people become addicted cannot be exaggerated. Meanwhile, different drugs act differently on the brain and have different psychoactive effects. People, of course, are also different: their histories, their personalities, their life circumstances. And the ways they understand themselves are different too. One person is in denial, another is not. One person's identity is wrapped up in their

addiction, another's is not. One person is obsessed with drugs and constantly craving them, another is not. One person is alone in a metaphorical cage with their grief, anxiety, anger, shame, despair, and self-loathing, while another is defiant, reckless, impulsive—something of a thrill seeker. Actions—including drug use—that appear similar on the surface may have different explanations in different people when we dig deep. As the philosopher Jenann Ismael succinctly puts the general point, "human beings are all specificity."[24] Explaining behavior by appeal to psychology opens the door to heterogeneity, allowing different cases of addiction to be explained by appeal to different psychologies and life circumstances. Heterogeneity is in this way connected to humanism. But, once this door is open, heterogeneity also allows some cases of addiction to be explained less by ordinary psychology than by neuroscience or cognitive science—that is, it allows for supplantation, not just supplementation, in some cases. Indeed, the paradigm put forward in this book allows that some cases of addiction may be explained by brain pathology—even if others are not. Why not, if addiction is heterogeneous?[25]

Recognizing the heterogeneity of addiction therefore holds tremendous promise as a way of moving beyond the deep disagreement currently found between models of addiction, as well as aligning addiction science with good clinical care—care that, to count as good, must be attentive to individual differences and tailored to individual needs (see chapter 3). But this promise is predicated on heterogeneity: on relinquishing the idea of a universal explanation of addiction or underlying "essence" that makes it what it is, and which every case of addiction must have, and no case of addiction can lack. As we shall see, the brain disease model is committed to such an essence, namely brain pathology (chapter 2).

Here, then, are the bare bones of the paradigm to come. I shall argue that addiction is a pattern of drug use that persists despite evident and severe costs such that it counts profoundly against a person's own good—that is, addiction is a pattern of *behavior* (chapter 1). To identify cases of addiction, we need to think about what it means for drug use to be—or not be—*good* for a person (chapter 4). To explain addiction, we need to explain *why* a person would persist in using drugs given that doing so is *not* good for them. The explanations canvassed over the course of this book include the possibility of brain pathology (chapters 8 and 9) but also self-medication and a person's social, cultural, and economic circumstances (chapter 10); the development of a security-based attachment to drugs (chapter 10); self-harm and a desire to die (chapter 11); an "addict" identity (chapter 12); denial and cognitive difficulties

(chapter 13); problems of control (chapter 14); and cravings for drugs (chapter 15). Some of these explanations will apply to some people with addiction, while other explanations will apply to others; and sometimes more than one explanation will apply to one and the same person—potentially interacting in complicated ways—even though some are mutually incompatible. The paradigm is humanistic because most (albeit not all) of these explanations not only render drug use in addiction *psychologically* intelligible but are, in addition, deeply personal—as is the question of what is good for a person. The paradigm is heterogeneous because there is no universal explanation or underlying essence of addiction. Its explanations are varied (chapter 3 and part III). Its causes are diverse (chapter 9).

I am by no means the first to propose that these two characteristics—humanism and heterogeneity—are central to understanding addiction. The history of addiction research contains many heretics.[26] But despite this counter history, my experience is that the paradigm shift I propose in this book is often met with outrage, as if it demonstrated a total failure to understand addiction or to care about those who struggle with it. The paradigm must of course be judged on its intellectual merits, but I am writing this book because I believe the dominance of the brain disease model is not only stifling addiction research but failing people in need of help. I state this here, plainly, not to provoke, but to emphasize the common ground between many of us who think about addiction and want to understand it better: the desire to help those who struggle with it. Given this common ground, why then is critique of the brain disease model so often met with outrage—indeed, so readily characterized as heresy rather than simply as respectful disagreement? I believe part of the answer is an underlying fear on behalf of people with addiction: the fear that only the brain disease model can save us from what has come to be known as *the moral model* of addiction.

The moral model treats drug use in addiction as no different from ordinary drug use apart from addiction. According to the moral model, both kinds of drug use are voluntary and morally wrong. The brain disease model aims to counter the moral model by claiming that drug use in addiction is different from ordinary drug use. According to the brain disease model, only ordinary drug use is voluntary; drug use in addiction is compulsive and caused by brain disease. I know of no actual addiction researcher from any academic discipline who espouses the moral model. It is, rather, a picture of addiction associated with a kind of black-and-white, pulpit-thumping, puritanical morality that has seeped into our shared cultural understanding of drug use over the course of

history—perhaps especially in the US.[27] But with the moral model contaminating the cultural air, it can seem as if the only reliable way to undermine the stigma surrounding addiction and stifle the tendency to blame and punish people who are addicted is to view them as victims of a brain disease that compels them to use drugs. This kind of motivation for the brain disease model is explicit in Leshner's seminal 1997 article and reaffirmed in an article published in 2021 by a group of distinguished neuroscientists led by Markus Heilig, "Addiction as a Brain Disease Revised: Why It Still Matters."[28] In other words, the brain disease model has become the scientific and public health orthodoxy in part out of faith that it, and only it, is capable of changing stigmatizing attitudes toward those with addiction, thereby countering the moral model and helping those in need. This faith in effect creates a dilemma: Either you commit to the brain disease model, or you fail to address stigma and adequately care about people with addiction.

The dilemma is false. The moral model and the brain disease model are not the only options. Although it will take the whole of the book to complete it, the paradigm developed here includes an ethics for addiction: a framework for addressing stigma, thinking about values, needs, differences, and responsibility, and supporting relationships with people with addiction (see part IV).[29] The dilemma also embodies two mistakes, one theoretical and one empirical, that are important to uproot at the outset.

The theoretical mistake is to allow the (real or imagined) ethical consequences of adopting a model to bear on the question of its scientific validity. The validity of any scientific model is established by considering confirming and disconfirming evidence—not by considering whether it does harm or good. A valid scientific model may, in certain circumstances, have terrible consequences. Think of the use of atomic theory in the development of the atomic bomb by the US and its subsequent deployment against Japan. These consequences are morally horrific. But they do not speak against the theoretical validity of atomic theory. Similarly, the dissemination of a scientific model that is not based on solid evidence but on mistake or even fancy may, in certain circumstances, have at least some consequences that are welcome. Think of the theory that the sun, moon, and stars all revolve around the earth, putting us at the center of the universe. Historically, belief in this theory may have bolstered religious conviction and a sense of humanity's importance, offering a kind of reassuring protection against the fear of death and meaninglessness that can overwhelm any of us. Needless to say, this silver lining, such as it is, does not confer theoretical validity. The bottom line is that ethics is one thing,

science another. Ethics needs to govern how science is practiced and how scientific knowledge is used, and scientific models can do harm or good in light of how they are developed and used. But these points have no bearing on whether a model is supported by the evidence. Applying this lesson to the brain disease model: Whether it helps or hinders attempts to combat stigma and counter the moral model of addiction is irrelevant to its scientific validity.

The empirical mistake is to assume, from the armchair, that the ethical consequences of the brain disease model are plainly good: that it is successful in combating stigma and countering the moral model. Although more empirical research is needed, initial findings suggest that things are much more complicated. The effects of labeling addiction a brain disease are both good and bad—a "mixed blessing," as the sociologist Nick Haslam puts it.[30]

A caveat before presenting the relevant studies: It is in truth unclear how much real-world impact the brain disease model has had. Scientific support for it initially coincided with the American War on Drugs in the 1980s and 1990s. This kind of punitive policy was politically advantageous in the US and elsewhere because it appealed to a popular moralism about drugs and drug users, intersecting with discriminatory gender, race, and class stereotypes.[31] Indeed, this intersectional dynamic is arguably still present. The more recent push in the US to frame addiction as a public health rather than criminal justice problem coincides all too conveniently with the impact of the opioid epidemic on suburban and white communities.[32] Meanwhile, increased public acceptance of broken brain models of mental disorder was not associated with decreased stigma in the 1990s and 2000s.[33] Rather than itself shifting punitive and stigmatizing popular attitudes to addiction, the brain disease model may simply have functioned as a useful prop when these shifted for independent reasons.

Nonetheless, large-scale surveys and experimental vignette studies suggest that acceptance of the brain disease model has a variety of effects on people's attitudes. So let us begin with the effects that are good. The brain disease model—like other broken brain models of mental disorder—is associated with public support for research and treatment.[34] It can also have a positive impact on relationships between people with addiction and families, friends, and colleagues (but see chapter 16 for a fuller picture).[35] We are in general inclined to attribute responsibility and to blame people for actions only if they are voluntarily undertaken. Actions that are compelled are not voluntarily undertaken. By characterizing drug use in addiction as compulsive, the

brain disease model undermines attributions of responsibility and the tendency to blame people with addiction for drug use and any associated wrongs or harms.

Turning now to the bad and with the proviso that cultural differences are under-studied yet likely to be significant:[36] Whatever its effects on personal relationships, the brain disease model—like other broken brain models of mental disorder—does not appear to combat addiction stigma, which remains high.[37] Indeed, there is evidence that it encourages social rejection and public perceptions of dangerousness and difference.[38] On reflection, this is hardly surprising. There is no reason to expect that labeling a condition a disease would be an antidote to stigma, for diseases are frequently associated with stigma. Think of cancer, leprosy, syphilis, or HIV/AIDS. People with diseases are often shunned and ostracized. To be pitied, yes, but as the acronym "NIMBY" says, "not in my backyard."

Relatedly, just as the brain disease model does not appear to decrease stigma in others, it does not appear to protect people with addiction from a kind of internalized stigma themselves. Even if they are not responsible or to blame for using drugs, they may yet feel that something is deeply wrong with them—deeply shameful.[39] After all, they have a brain disease. For this reason, the model can also impede people's ability to recognize that they have a problem with drugs and seek help. Broken brain models in effect divide people into two kinds: those whose brain is broken, and those whose brain is not. As the psychologist James Morris has argued, this may motivate denial and self-deception as a way of avoiding the stigma and threat to identity that the brain disease label creates.[40]

Lastly, the brain disease model may contribute to pessimism about recovery[41]—a mark of poor prognosis—and increase the likelihood of relapse.[42] In characterizing drug use as compulsive, the model undermines what is arguably a plank of all successful treatment, namely a sense of one's own agency and ability to do things differently. That is, an ability to change one's relationship to drugs and construct a life, an identity, where they play a less significant role.[43] To preview one of the core ideas of the ethical framework (see part IV): Because of its connection to voluntary agency, responsibility can have an important role to play in recovery if divorced from blame.

As I say, with respect to its ethical consequences, the brain disease model is a mixed blessing.

Three final introductory points of clarification and orientation, to finish clearing the ground for the paradigm to come.

First, I take it as a basic truth that there is nothing intrinsically morally wrong with using drugs. Most of us use drugs. We drink alcohol—beer, wine, whiskey, martinis, negronis. We drink caffeine—in coffee, tea, cola. We smoke cigarettes and vape. We use cannabis, opioids, amphetamines, benzodiazepines, Z-drugs (for example, the sedatives zopiclone, zolpidem, zaleplon)—prescribed or not. We use MDMA, psychedelics, ketamine, kratom. Meanwhile new drugs are always being created—by pharmaceutical companies as much as by drug cartels. There can no doubt be better or worse reasons for using drugs, as there can be better or worse reasons for anything we do. Equally, using drugs carries health risks—again, like other things we do. And in certain circumstances, namely those that straightforwardly involve or significantly risk seriously harming or violating the rights of others, using drugs is morally wrong. To take but a few examples: It is wrong to drink (or to use any drug) and drive, or to leave drug paraphernalia in public spaces, thereby putting others at risk. It is wrong to use drugs produced—as so many consumer goods in our society are—through exploitation, violence, and the destruction of communities in developing countries.[44] It is wrong to use drugs in ways and at times that compromise your ability to care for your children—whether you are addicted to drugs or not. But there is nothing intrinsically morally wrong with using drugs—nothing wrong with drug use *in itself*. Why would there be?

I do not believe there is any good answer to this question. If you disagree, I challenge you to provide it. I can see no moral principle that, for example, sanctions a bottle of beer or a glass of wine but condemns a line of cocaine or a tab of LSD or a joint. I can see no moral principle that permits opioids for pain relief when prescribed by a doctor as part of palliative care but forbids the use of heroin to escape from a life of misery and suffering on the streets. And, to address a common objection, let us be clear: The fact that a religion condemns the use of some drugs, or that a country criminalizes the possession of some drugs for some purposes, does not show that such use is *intrinsically* morally wrong.

The argument for this point originates in Plato's dialogue *Euthyphro*, written over two millennia ago.[45] The argument is beautifully simple. It cannot be that something is morally wrong because the gods proscribe it; rather, any gods worth worshipping as opposed to obeying merely for fear of their wrath—that is, any gods who are themselves morally good—will proscribe something only if it is morally wrong. Just so with the criminalization of conduct, as the philosopher Douglas Husak has in effect argued in his lifelong plea

for drug decriminalization.[46] Although the principles by which conduct is legitimately criminalized are complex and contested,[47] the essence of Husak's argument mirrors Plato's *Euthyphro*. It cannot be that something is morally wrong because it is criminalized; rather, one of the most basic, agreed principles by which conduct is legitimately criminalized is that it is morally wrong—specifically, it involves or significantly risks seriously harming or violating the rights of others.

To help see the force of these arguments, let us take a concrete example: Think of how many religions and criminal codes have prohibited homosexual acts over the course of human history. Such prohibition did not make homosexual acts morally wrong. Quite the contrary. The fact that homosexual acts are in no way morally wrong makes it morally wrong to prohibit let alone punish them—a moral stain on religions and criminal codes that do. There are, inevitably, a host of reasons—religious, legal, political, social, cultural, prudential—to obey religious and criminal laws, even when these laws are bad, and so when there are, equally, moral reasons to resist, to revolt, to refuse to obey. The point here is not about whether, on balance, resistance or obedience is called for in whatever religious or legal circumstances we may find ourselves. The point is that, if conduct is not intrinsically morally wrong, religious and criminal law cannot make it so.

I believe it is imperative to state plainly the basic truth that there is nothing intrinsically morally wrong with using drugs, and to keep it clearly before our minds, if we are to be in any position whatsoever to address addiction stigma and reflectively consider what our attitudes to drugs and relationships with people with addiction ought to be like. I do not diminish the impact addiction can have on families, friends, colleagues, and communities. People with addiction sometimes wrong and hurt others—as well, of course, as harming themselves. (It bears saying immediately that so, too, do many people who are not addicted to drugs.) Thinking about how to reckon with this fact within our personal relationships is part of the project undertaken in this book (see part IV). Nonetheless, there are many occasions of drug use, in addiction as much as apart from it, where absolutely nothing is done that is morally wrong. Recognizing this helps us realize what should anyway be obvious: Addiction stigma comes from us. It comes from the cultural air we breathe, which is saturated with a history of religious moralism about drugs; from the criminalization of drug possession, which forges an invidious association between drugs, deviancy, and crime; and from the intersection of our ideas about drugs and drug users with discriminatory class, race, and gender stereotypes.

Demonizing drugs is the easy way out. It stops us, as individuals and as a society, from questioning, and thereby potentially changing, our own stigmatizing and stereotyping attitudes.

Notice that no matter the good intentions of its champions, the brain disease model implicitly demonizes drugs rather than encouraging such questioning. The brain disease model aims to address stigma and foster care for people with addiction by providing them with a simple, blanket excuse. Drug use is not voluntary in addiction, because people with addiction have a brain disease that compels them to use drugs. For this reason, no one is straightforwardly responsible or to blame for using drugs, once addicted. But here's the rub: We only need an excuse if we are doing something morally wrong. The felt need to provide an excuse for all drug use in addiction betrays an assumption that all drug use is morally wrong. Otherwise, we could proceed—as I argue we should at the end of this book—case by case (see chapter 19). First, simply by pausing, taking a breath, and reflecting on why we are inclined to blame someone and whether we are right to do so. Second, if indeed they have done something morally wrong—or if not morally wrong, then so upsetting to us personally that we can't pretend that nothing is wrong between us—only then by considering how we want to address what has occurred and relate to that person. As part of this, we can consider whether any of the wide panoply of reasons, excuses, justifications, and mitigating circumstances—including but by no means limited to compulsion—might reduce or even eliminate their responsibility or temper our tendency to blame. But this is hardly the only thing that matters when people hurt us and relationships go wrong.

The second clarificatory point pertains to the distinction between drug and behavioral addictions. In one sense, I reject this distinction: Using drugs is a behavior—something we do—just as much as is eating, gambling, exercising, working, watching porn, or having sex. I see no reason to think that people cannot be addicted to all these behaviors. Indeed, there is some reason to think that people can be addicted to just about any behavior, including, famously, eating carrots.[48] But the paradigm proposed in this book is intended as a paradigm for drug addiction, which is evidently different from other addictions given that—notwithstanding important differences between classes of drugs[49]—the ingestion of drugs typically affects the brain differently than, say, the ingestion of carrots. I would be delighted if the paradigm proved to offer insight into other behavioral addictions, and I will very occasionally discuss other forms of addiction, but I make no claim to offer a new paradigm for understanding all possible addiction. There are important differences between

the many things to which we can become addicted—drugs, food, games of chance, exercise, work, porn, sex, and no doubt more—and each should be treated in its own right.

But that said, the third and final clarificatory point is that, despite proposing a new paradigm for drug addiction, I do not provide a watertight definition of "drug."

The *Oxford English Dictionary* (OED) defines "drug" thus:

*a*. Originally: any substance, of animal, vegetable, or mineral origin, used as an ingredient in pharmacy, chemistry, dyeing, or various manufacturing processes. In later use: *spec.* a natural or synthetic substance used in the prevention or treatment of disease, a medicine; (also) a substance that has a physiological effect on a living organism.[50]

*b*. A substance with intoxicating, stimulant, or narcotic effects used for cultural, recreational, or other non-medicinal purposes. In later use frequently: *spec.* a controlled substance used illegally and often habitually. Frequently in *plural*.[51]

A workable definition of "drug" for the purpose of understanding drug addiction can be extricated from the latter part of *a* combined with the first part of *b*. A drug is a substance with what we can summarily call a "psychoactive" effect on the mind and consequently the behavior of living organisms, at least in part because it has a physiological effect on bodies, in particular, brains; and which is typically used for cultural, recreational, and other nonmedicinal purposes as well as medicinal purposes. We need to specify that the psychoactive effect is due to a physiological effect to rule out pure placebos. But the caveat "in part" is essential since it is evident that much of the psychoactive effect of drugs is due to the expectation of the effect—hence not purely physiologically caused, but psychologically mediated.[52] Drugs can also clearly be used for medicinal and nonmedicinal purposes—in so far as the line between these can coherently be drawn at all. The scope of medicine is far from self-evident, including as it does conditions that are, or are caused by, diseases, and conditions that are not diseases at all, such as pregnancy and many natural effects of aging, like menopause—just to give some obvious examples. Doctors therefore routinely prescribe drugs for conditions that are not diseases, and people who are not doctors self-medicate with drugs for conditions that are. Importantly, however, the legality or habituality of use should be no part of the definition of "drug." Nothing becomes a drug through criminalization or ceases to be one through decriminalization, and drugs that are used infrequently or

irregularly (say, because they are hard to produce, or only used by a culture as part of a yearly religious ritual) are no less drugs for that. This definition is potentially prey to counterexamples (for example, does it mean that sugar is a drug—and is that a problem?), so not watertight. But it is nonetheless a viable gloss on what we mean by "drugs" and will include all of the following common kinds: alcohol, amphetamines, ayahuasca, barbiturates, caffeine, cannabis, benzodiazepines, cocaine, fentanyl, GHB, heroin, inhalants, ketamine, khat, kratom, LSD, MDMA, mescaline, morphine, nicotine, PCP, psilocybin, Z-drugs, and many other kinds of prescription opioids, stimulants, and sedatives, as well as over-the-counter medicines, such as dextromethorphan and loperamide.

We know well enough what drugs are. Our problem is that we don't know what addiction is. This, then, is where the paradigm begins: by explicating addiction.

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