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ADDICTION, CHRONIC ILLNESS, AND RESPONSIBILITY*

ADICCIÓN, ENFERMEDAD CRÓNICA Y RESPONSABILIDAD

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ABSTRACT

Some theorists have argued that we should understand the notion of *free will* from a functional perspective: free will just is our ability to choose effectively and adaptively in an ever-changing environment. Although far from what many philosophers normally mean by free will, those who adopt this biological-evolutionary perspective can clearly define and defend a notion of personal responsibility. One consequence of this point of view is that addicts become responsible for their actions, for at each choice point, there is a real sense in which the addict could have elected not to use or abuse. As a result, it has been argued that addiction is not a disease, that addictive behavior is voluntary, and that sometimes it is even rational. This paper defends a different way of thinking about addiction, one that aligns it with other complex chronic illnesses. The perspective put forth here suggests that these discussions about responsibility and free will represent an over-simplified and neuropsychologically inaccurate portrait of basic human capacities for behavioral choice.

Keywords: addiction, chronic illness, decision, free will, responsibility.

RESUMEN

Algunos teóricos han sugerido que la noción de libre arbitrio debe entenderse desde una perspectiva funcional: el libre arbitrio es nuestra habilidad para elegir de manera efectiva y adaptativa en un entorno que cambia continuamente. Aunque se distancian bastante del modo en que muchos filósofos entienden la noción de libre arbitrio, quienes adoptan esta perspectiva biológica-evolutiva claramente pueden definir y defender una noción de responsabilidad personal. Una consecuencia de este punto de vista es que los adictos se vuelven responsables de sus acciones, puesto que cada vez que hay que tomar una decisión, hay un sentido real en el que el adicto pudo haber elegido no usar o abusar. Por consiguiente, se ha argumentado que la adicción no es una enfermedad, que el comportamiento adictivo es voluntario, y que algunas veces hasta es racional. Este artículo defiende una manera distinta de pensar la adicción, acercándola a otras enfermedades crónicas complejas. La perspectiva aquí propuesta sugiere que estas discusiones sobre responsabilidad y libre arbitrio brindan una imagen demasiado simplista y neuropsicológicamente incorrecta de las capacidades humanas básicas para tomar decisiones.

Palabras clave: adicción, enfermedad crónica, decisión, libre arbitrio responsabilidad.

Addiction, Chronic Illness, and Responsibility

Several philosophers (and at least one neurobiologist) have argued that we should understand "free will" from a pragmatic, functional perspective: free will just is our ability to choose effectively in an ever-changing environment (*cf.* Banja 2015; Dennett 1984; Fuster 2013; Nahmias 2006). It reflects of our capacity to select the most adaptive behaviors from a range of possible behaviors before us. Of course, such a view of free will is quite far from what philosophers normally mean when they discuss the concept. Perhaps Banja puts this incompatibility best:

> In positing a contextualized, embodied, adaptive, improvisatory, recurrently vectored interaction of sensory inputs and behavioral outputs, the evolutionary account redefines the "free" of free will in a way that bears little resemblance to the philosophical tradition's compatibilist versions relying on contra-causal willing. (Banja 2015 8)

However, even though this perspective on free will is quite far from philosophy's usual interpretation, those who adopt it are perfectly clear that one can both define and defend a notion of personal responsibility using this biological-evolutionary perspective. Insofar as organisms can alter their behavioral choices in response to new environmental information such that they can still select the most adaptive, then that ability points to a version of responsibility that these organisms have. They are responsible in so far as they can choose behaviors that promote survival over those that do not (or do not as much).

One consequence of this point of view is that addicts become responsible for their actions, for at each choice point, there is a very real sense in which the addict could have elected not to use or abuse. Such a perspective is not unusual –indeed, most psychologists believe that addicts are responsible for the consumption of their drugs of choice (*cf.* Buckwalter 2014; Uusitalo 2015), and most community-based treatment programs are predicated on the idea that addicts can and do choose to use (*cf.* Heyman 2009; Pickard 2015). Hanna Pickard in particular advocates for this conclusion. She urges that a) addiction is not a disease; b) addictive behavior is voluntary; and c) in many cases, it is rational as well (*cf.* 2012 §40-49).

We take exception to this perspective on addicts and addiction. In what follows, we shall advocate for a different way of thinking about addiction, one that aligns it with other complex chronic illnesses, like heart disease, diabetes, obesity, and the like. Adopting this alternative perspective suggests that these sorts of discussions about responsibility and free will miss the mark: they represent an over-simplified and neuropsychologically inaccurate portrait of the basic human capacities for behavioral choice. We shall describe why a different approach to understanding addiction is warranted and what this approach tells us about personal

responsibility and decision-making in general. Finally, we shall gesture towards some larger conclusions from this discussion, *viz.,* that discussions of free will and responsibility are, at bottom, empirically bankrupt.

Disease Models of Addiction

The so-called disease model of addiction has received widespread support across a range of expert institutions, including the U.S. National Institute on Drug Abuse (2009), the World Health Organization (2004), and the American Psychiatric Association (2013). The position of the U.S. National Institute on Alcohol Abuse and Alcoholism is that

> alcoholism is a disease in which voluntary control of behavior progressively diminishes and unwanted actions eventually become compulsive. It is thought that the normal brain processes involved in completing everyday activities become redirected toward finding and abusing alcohol. (2013)

This view has also made its way into lay approaches to explaining addiction, including what is advocated in Alcoholics Anonymous, Narcotics Anonymous, and other similar community support groups. For example, the on-line popular medical site, MedicineNet.com, asserts,

> Alcoholism is a disease. The craving that an alcoholic feels for alcohol can be as strong as the need for food or water. An alcoholic will continue to drink despite serious family, health, or legal problems. Like many other diseases, alcoholism is chronic, meaning that it lasts a person's lifetime; it usually follows a predictable course; and it has symptoms. The risk for developing alcoholism is influenced both by a person's genes and by his or her lifestyle. (2015)

For each of these institutions, addiction is seen as a chronic, neurobiological disease that robs its victims of their ability to control their behavior with respect to their substances of abuse.

In contrast, Hanna Pickard (2012), John Banja (2015), and perhaps Daniel Dennett (1984), hold that addiction is not a disease that takes voluntary control away from anyone (*cf.* Heyman 2009, Kloss and Lisman 2004).It is not a disease of the will. For it is not a disease at all, nor does it circumscribe the will. Of the three, Pickard has the most thorough defense of this position. She musters two lines of argument in support of the notion that addiction is incompatible with the disease model. We believe that each of these lines misrepresents what the data are telling us about addiction.

The Trajectory of An Addict

First, Pickard points out that U.S. survey data show unequivocally that alcohol and substance abuse peaks in late adolescence and early adulthood, "by the late twenties or early thirties" (2012 2) indicating, she

believes, that "addicts tend to 'mature out' as the responsibilities and opportunities of adult life increase" (*ibd.*).The exceptions to these trends are addicts who also suffer from additional mental disorders. In those cases, addiction can prove to be much more recalcitrant. She concludes:

> The large-scale national survey data do not suggest that, for the general population, addiction is correctly characterized as a chronic, relapsing condition of any sort, never mind a chronic, relapsing, neurobiological disease. On the whole, addiction is only a chronic, relapsing condition for psychiatric patients. (Pickard 2012 2)

However, the studies that she cites support neither her interpretation of the data nor her conclusions. While it is true in the United States that a greater percentage of people with a drug use disorder are between the ages of 18 to 29 than those aged 30-44 (48% vs. 34%), we note that the death rate for opiate overdose is highest among the 45-54 age group, followed by those aged 35-44 and 55-64 (U.S. National Center for Health Statistics 2015). Moreover, the difference between those with alcohol abuse disorder aged 18-29 and 30-44 is quite small (38% vs. 37%) (*cf.* Dawson *et al.* 2005 281-292). (A cross-cultural analysis of data from Colombia, Mexico, Lebanon, Nigeria, Japan, and the People's Republic of China suggest that we see similar trends globally.) Given that over four times the number of people abuse alcohol over other drugs, it is difficult to make a meaningful claim that abuse *tout court* peaks in late adolescence and early adulthood.

It is also true that about 75% of people with some sort of dependence on alcohol do reduce or stop consumption on their own, without any intervention, community group support, or professional treatment regimen. However, of the 75% who no longer meet the criteria for alcohol dependence, 39% of them continue to engage in patterns of drinking that put them at risk for relapse or only in partial remission (*cf.* Dawson *et al.* 2005 281-292). Alcohol dependence can and does swing between heavy and lighter drinking, even abstinence, and then back again; and not surprisingly, the severity of dependence influences any trajectory toward recovery (*cf. ibd.*). Indeed, Koob and Valkow describe relapse as a "hallmark of addiction" (2010 218). It also seems that those who stop drinking naturally and permanently have relatively mild disorders to begin with (*cf.* Dawson *et al.* 2005; Weisner *et al.* 2003).

At this point, it is quite unclear why we see a significant drop in alcohol dependence and abuse after the age of 44. It oversimplifies the data to claim that maturation levels and increased responsibilities or opportunities can account for recovery trends. Consider: three of the eleven diagnostic criteria in *DSM-5* for alcohol use disorders are concerned directly with use interfering with significant life responsibilities

and events,[1] while another three are indirectly tied to use significantly impacting life's major activities.[2]Thus, it is highly likely that anyone diagnosed with a substance dependence disorder has already experienced at least some of the "responsibilities and opportunities of adult life," without it affecting their addiction. Indeed, data suggest that many of the putative associations between significant life events and likelihood of recovery indicate selectivity and not causation. That is, things like marriage and job promotion are just less likely to happen to people who are alcohol or drug-dependent instead of the life events themselves prompting recovery. In other words, while important life events might "contribute to" the spontaneous recovery from alcohol dependence for some people, they do not "fully explain" them (*cf.* Dawson *et al.* 2006 195).

In short, current data do not support the claim that addiction is not chronic. While some addicts with milder forms of dependence do appear to fully and permanently recover, significant numbers do not. Moreover, recovery does not seem to be tied to any discernable natural life trajectory or set of events.

Impulses and Compulsions

Second, Pickard claims that addictive behaviors are not compulsive. She notes that "drug-seeking and drug-taking behavior appears to be deliberate, to be flexible, and to involve complicated diachronic planning and execution" (2012 4). The notion of compulsion, however, denotes an irresistible desire, one "so strong that it is *impossible* for it not to lead to action. The compelled person has no power to do otherwise" (Pickard 2012 3). True actions –and not reflex or automatic movements– require alternatives in behavior. True actions require choice, but genuine choice belies the notion of compulsion.

She supports this conceptual argument with biological considerations as well. She contends that there is nothing in neurobiology that

1 These criteria are the following: (5) "Recurrent alcohol use resulting in a failure to fulfill major role obligations at work, school, or home." (6) "Continued alcohol use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of alcohol." (7) "Important social, occupational, or recreational activities are given up or reduced because of alcohol use" (*cf.* American Psychiatric Association 2013).

2 These criteria are the following: (3) "A great deal of time is spent on activities necessary to obtain alcohol, use alcohol, or recover from its effects." (8) "Recurrent alcohol use in situations in which it is physically hazardous." (9) "Alcohol use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by alcohol" (American Psychiatric Association 2013).

suggests that there is something fundamentally different about desires for a substance of abuse than any other desire, though they might be stronger and more insistent than ordinary wants (*cf.* Dill and Holton 2014). Nor does the neurophysiology of addiction suggest that control gets lost.

Let us look more carefully at the neurobiology of addiction. Pickard focuses her arguments on how and whether addiction impacts desire, which we all agree it does. However, this focus obscures the far-reaching cognitive, emotional, and behavioral implications of addiction due to a pattern of dysregulated neural plasticity and cell death (*cf.* Hyman 2005). The dopamine receptors morph in the brains of people abusing a wide range of substances, including cocaine, methamphetamine, alcohol, and heroin. While these receptors are linked to motivation and reward, they are also, importantly, tied to choosing the most salient action in a given circumstance; that is, they are activated in response to the action most relevant for the person at the time (National Institute on Drug Abuse 2008). Brain-imaging studies of addicts show cell death in areas of the brain associated with judgment, decision-making, learning, memory, as well as with inhibitory control (*cf.* Fowler *et al.* 2007). Cortical degradation in addicts underlies impairments in problem-solving and cognitive flexibility, which are also relevant to understanding why addicts behave the way they do (*cf.* Pfefferbaum *et al.* 2001).

We suggest, again, that Pickard's description of addiction is over-simplified. Generally, when psychologists and psychiatrists speak of compulsive behaviors, they are not referring to a behavior that cannot be controlled under any or even most circumstances. Even the tics of Tourette's syndrome or Obsessive-Compulsive Disorder can be resisted for a while. Instead, they use "compulsion" to refer to behaviors that persevere despite adverse consequences or despite being the incorrect response in choice situations; it can also refer to the persistent re-initiation of a habitual behavior (*cf.* Everitt and Robbins 2005). This sense of compulsion is tied to negative reinforcement mechanisms. Patients with compulsive disorders feel great anxiety and stress before committing the compulsive act and then relief from the stress after performing the act (*cf.* Koob and Volkow 2010). (This contrasts with impulsive behaviors, which are linked to positive reinforcement: impulsive patients feel tension or arousal before committing the impulsive act and then pleasure or gratification after).

More to the point: what is significant about an addict's behavior is perhaps not the question of whether the drug-seeking behavior is compelled in any interesting sense, but rather that the affective-cognitive functioning of the addict as a whole is significantly impaired, which impacts not only the rate, amount, and time of consumption, but a whole

range of other activities. For example, intoxication, reduced impulse control, and aggression are highly correlated for a range of chemicals. A quick glance at arrest records confirms this. Almost half of first-time violent offenders in the United States have history of drug use, and more than 80% of offenders with five or more convictions do. Depending on the study, somewhere between 40% and 60% of men arrested for violent crime test positive for illegal drugs. Upwards of three-quarters of state prisoners with a mental health problem and over half of those without are dependent on or abused alcohol or drugs (*cf.* Bureau of Justice Statistics 2006a). We see similar statistics and ratios in the jails (*cf.* Bureau of Justice Statistics 2006b).

Some researchers consider a failure to maintain inhibitory control over drug-seeking and drug-consuming behaviors definitive of addiction (*cf.* Fillmore and Weafer 2004; Finn *et al.* 2000; Lyvers 2000). But, of course, saying that someone is aggressive or impulsive is just as uninformative as claiming that a behavior is compulsive –these are all multifaceted constructions that belie easy reduction to psychological testing or neural circuits. For example, "impulsivity" is operationalized in the laboratory to a lack of inhibitory control in responses when subjects are required to withhold reacting under certain circumstances, *e.g.*, a go/no-go task. Both test subjects under the influence of alcohol and alcoholics themselves show premature motor preparation and difficulty inhibiting a cued or primed behavioral response (*cf.* Oscar-Berman and Marinkovic 2007).

But on top of difficulties inhibiting cued responses, addicts also have deficits in decision-making, beyond making poor choices regarding consumption of their substance of abuse (*cf.* Fein *et al.* 2004; 2006). They have difficulties with cognitive evaluations of their environment and selecting the most effective response strategies (*cf.* Oscar-Berman and Marinkovic 2007). In short, substance abuse impairs executive and motivational functioning in general, which, in turn, affects self-regulation and goal-directed behaviors.

It might be helpful to compare an addict's cognitive capacities to those of an old person –their impairments are similar. Both addicts and the elderly show impaired working memory, visuospatial abilities, and gait and balance, which correspond to corpus callosum and hippocampus degradation, cerebral atrophy, and cerebral sulci enlargement (*cf.* Pfefferbaum *et al.* 2006). The frontal lobes in particular are vulnerable in alcoholics. Estimates are that 15%-23% of frontal neurons are lost with chronic alcohol consumption (*cf.* Harper 1998 101-110). As a result, just like many elderly persons, alcoholics have difficulty with memory, fluency, cognitive flexibility, and perseveration (*cf.* Dirksen *et al.* 2006; Oscar-Berman *et al.* 2004).

Or, we could compare an addict's brain to that of a child –the frontal lobe system and its concomitant ability to engage in long-term planning and goal setting are underdeveloped compared to a normal adults. There

are very few children who can resist an easily available treat, even when they know that resisting for just a few minutes will bring more treats later. The brain structures just are not there to make those sorts of choices. Children cannot act in their own best interests. Nor can addicts. Addicted brains lose their ability to refuse or ignore proposed responses, even when those actions are strongly contra-indicated.

Or, we could even compare addicts' brains to the brains of chronic pain patients (*cf.* Apkarian 2012; Hardcastle 2014). We know that the transition from normal consumption to genuine dependence involves changes in the connectivity among the nucleus accumbens (NAc), the amygdala, and prefrontal cortex (*cf.* Gilpin and Koob 2008). The NAc encodes everything from salience to reward to aversion. Both chronic pain patients and addicts show increases in connectivity of NAc to medial prefrontal cortex, which contributes to increased reinforcement of drugs and alcohol in addiction and the transition from acute pain processing to a chronic pain syndrome in pain patients.

In addition, the amygdala is connected to processing salience or reinforcement in addicts. Nociceptive pathways also terminate in the amygdala, which, as part of the limbic system, processes the emotional aspects of pain. Just as with addicts, amygdala activity is increased in chronic pain patients, which leads to increased negative reinforcement of painful experiences. Activation in the amygdala in both addicts and chronic pain patients indexes changes in medial prefrontal cortex. In both cases, we see concomitant deficits in decision-making.

The point in all of this is to illustrate that calling addiction a compulsion, or denying that it is, mischaracterizes the cognitive and behavioral deficits that addicts have. In deciding whether to label addiction as a disease, the issue is not whether addicts can resist the siren calls of their substances of choice, but that their functioning is impaired along multiple dimensions, which impacts not only their substance use, but also many facets of their existence. Regardless of how one comes down on the question of whether addiction is a disease, addicts have significant impairments in their abilities to analyze environmental stimuli and then respond in an adaptive fashion.

Is this impairment enough to conclude that addicts are therefore not responsible for their addictive behaviors?

Addiction as Voluntary Behavior

Banja (2015) argues that addicts are indeed responsible because they could engage in practices that would diminish the chances that they would act on their cravings. He approvingly describes Pickard as

> roundly fault[ing] most addicts for not adverting to …choice modifiers by suggesting that they don't want to exert the effort to reshape their

> choice repertoire in ways that would make their drug use more cumbersome, unwieldy, unpleasant, and less likely. (8)

While this view is quite common, it also assumes a view of personal freedom that Banja, Pickard, and the rest are specifically arguing against.

Evolutionary perspectives on free will suggest that philosophical versions of freedom, the "freedom to have acted or chosen otherwise under identical conditions," are empirically moribund (because, of course, under the same conditions, the same biological organism would respond to the same stimuli in the same way). Instead, freedom to choose just means that the brain has a variety of behavioral options before it to address its particular circumstance, and it tries to maximize its potential for survival in whatever response it selects. In many respects, this view is aligned with a compatibilist's notion of freedom: freedom is the absence of constraint. However, as we allude to above, addicts are cognitively and affectively constrained in exactly such a way as to deny them this sort of evolutionary freedom. The only way to assert that addicts could be responsible for doing things that would lessen their addictive tendencies is to assume that they could have chosen otherwise in the very same circumstances in which they did not, which is exactly the view of freedom that is being denied.

To get to this conclusion, let us first look at the sort of behavioral control being discussed here. Some philosophers agree that, under the right conditions, responsibility for some outcome can be traced back to some suitable moment in history; an agent's responsibility for an action does not solely depend on the decision just prior to the outcome (*cf.* Vargas 2005 269). In some cases, we can trace an agent's responsibility for a later outcome back to that agent's control over earlier actions, even if the agent did not have control at the time of the later action that led to the outcome in question (*cf.* Fischer and Raviza 1998; Fischer and Tognazzini 2009). Prior control might entail that we are later responsible for our actions. This in particular is the way the criminal justice system justifies holding drivers responsible for driving under the influence of drugs or alcohol: even if they were not in control of their decision-making faculties at the time of their arrest, they were in control back when they were sober and decided to consume with their car keys available.

I might forgo purchasing chocolate at the grocery store now to prevent myself from absent-mindedly consuming it later in the evening while watching TV. I have deliberately arranged my environment such that acquiring candy at a later decision-point becomes more onerous, which would influence my decision about eating the sweets. The suggestion is that we are responsible for those sorts of environment-arranging

activities, which then trickle down into our being responsible for the later outcomes of our environmental arrangement. Banja, Pickard, and others, argue that addicts are responsible for arranging their environments in such a way that they can't use.

Can addicts actually choose in this manner? We shall argue that they are just as free (or as not free) as the rest of us are in those circumstances. Which is to say: most people do very poorly in trying to arrange their environment so that they force certain choices or behaviors later.

One of the oddities about addiction is that while most agree that it is a chronic illness, by and large we regard it as though it were a social problem and not a medical one. We expect law enforcement and social support groups to manage the problem instead of our health care system using evidence-based treatments. Most community counselors for addiction have no actual medical training –quite often they rely only on their personal experiences as former attendees in whatever program they are supporting– and most medical schools do not require coursework in addiction (*cf.* McLellan *et al.* 2000). Indeed, most physicians do not screen for alcohol or drug dependence during routine office visits (*cf.* Fleming and Barry 1991; Spitoff and Turner 2015).

The other oddity about addiction is that we do this, despite the fact that this approach clearly does not work. An estimated 8.9% of the U.S. population needed treatment for alcohol or drug use in 2012, which, though less than the 9.3% needing treatment a decade ago, is not substantially different from the percentages across the intervening years (*cf.* U.S. Substance Abuse and Mental Health Services Administration 2013). Our treatment strategy clearly has not been successful, yet during this same time period we have done little to change it.

Directly opposed to Pickard, we advocate taking the idea that addiction is a complex chronic illness *more* seriously, and this means that it should be treated in a fashion similar to other complex chronic illnesses. Consider: Substance use addiction has been tied to a complex interaction among genes, individual choices and behaviors, and the surrounding environment, which results in very specific pathophysiologic responses (*cf.* Levy 2013). So have type 2 diabetes mellitus, hypertension, and adult-onset asthma (*cf.* McLellan *et al.* 2000). Data from twin studies indicate that the genetic contributions to the risk for type 2 diabetes, hypertension, asthma, and addiction are all comparable (*cf.* Fragard *et al.* 1995; Karo *et al.* 1992; Nieminen *et al.* 1991; True and Xian 1999; Tsuang *et al.* 1996, Van den Bree *et al.* 1998). Tolerance (or intolerance) for alcohol, for example, appears heritable (*cf.* Chao *et al.* 1994; Newmark *et al.* 1998; Schuckit 1994; Schuckit and Smith 1996). However, the risk factors for diabetes and hypertension (*e.g.*, obesity, stress, and inactivity) are also all strongly linked to family traditions, culture, and personal preferences

(*cf.* Mitchell *et al.* 1996; Svetkey *et al.* 1996), just as are addicts' original decisions to consume alcohol or drugs. In all these cases, while the initial choice to consume or eat excessively or forgo exercise is perhaps voluntary, genetic inheritance as well as the sociocultural environment amplify and shape the effects of these decisions.

Moreover, all of these ailments are intermittently exacerbated. Importantly, diabetes, hypertension, and asthma require continued care through the patients' lifetime. There are medical treatments for them, to be sure, but, similar to recovery from addiction, treatment success also depends upon a patient's willingness to adhere to particular regimes. And compliance is an issue across these illnesses. Less than 30% of patients with adult-onset diabetes, hypertension, or asthma observe the diet and behavioral changes required to reduce the risk factors for recurrence (*cf.* Clark 1991; Dekker *et al.* 1993; Graber *et al.* 1992).[3] More importantly, "relapse" rates are similar across these illnesses as well. Up to 50% of adults with diabetes and somewhere between 50% and 70% of adult patients with hypertension or asthma have recurrent symptoms each year that require medical care (*cf.* Clark 1991; Dekker *et al.* 1993; Graber *et al.* 1992; Schaub *et al.* 1993). These rates are virtually identical to what we find with addicts: somewhere between 40% and 60% of patients treated for alcohol or drug dependence return to active use within a year of some treatment intervention (*cf.* Finney and Moos 1991; Hubbard *et al.* 1997; McLellan and McKay 1998).

The point here is that addicts' traced-control of their decision-making regarding whether to consume is virtually identical to what we find in other complex, chronic illnesses. None of these patients are very good at arranging their environments to encourage compliance with their treatment regimens. Of course, one could also conclude so much the worse for all chronically ill patients. One reason that they are all sick is that they have repeatedly made very poor decisions about their behaviors and now have to suffer the consequences. Not only do we have fat-shaming and addiction-shaming, but perhaps we should have diabetes, hypertension, and asthma-shaming, too.

But wait: there is more. If we look at other cases of putative historical control that do not involve illness, we still see similar patterns of failure. For example, almost 16% of professional football players in the U.S. file for bankruptcy during their first 12 years after retirement, despite having earned an average of $3.2 million (in 2012 dollars) (*cf.* Carlson *et al.* 2015). Neither the

3 And, just as with addiction, outcomes are poorest among those with low socioeconomic status, few family or social supports, or other psychiatric disorders (*cf.* Gerstein and Harwood 1990; National Institute of Drug Abuse 1991; McLellan *et al.* 1994; Moos *et al.* 1990).

amount of money earned nor the years spent playing affected the likelihood of filing for bankruptcy. This rate is comparable to the bankruptcy rates for all Americans of the same age. Even though pro-football players, unlike most young adults, accumulated the great wealth, many fail to organize their environments such that they would have appropriate resources upon retirement. And they did this, despite knowing full well that their sports careers were likely to be brief.

But there is nothing special about football players' lack of ability to translate sudden wealth into financial security. Lottery winners are even worse; they file for bankruptcy at twice the rate of the broader population (*cf.* Hankins *et al.* 2011). The U.S. Certified Financial Planner Board of Standards estimates that nearly a third of lottery winners will go bankrupt at some point after winning (*cf.* Anderson 2012).

When we think about how many people begin diets on 1 January, only to have them end on 2 January, how many people have idle gym memberships, unused running shoes, yet plan for regular exercise; when we consider that almost 70% of Americans are overweight or obese, yet less than 20% meet the federal guidelines for exercise (*cf.* National Institute of Health 2012; National Center for Health Statistics 2015), we can surely conclude that the sort of historical control that Banja adumbrates just does not reflect the abilities of the majority of humans. We just are not very good at sacrificing short-term rewards for long-term goals, even when operating at full cognitive capacity (*cf.* Ainslie 2001). Is it theoretically useful to claim that so many of us are freely guilty and irresponsible? Perhaps a different approach to understanding responsibility is warranted.[4]

Rational Addiction

Pickard claims that sometimes addicts' continuing to use can be a rational response to extreme emotional pain if the addiction is comorbid with other psychiatric disorders: addict use their substances of abuse to self-medicate. Others have already pointed out that Pickard overstates the relationship between continued use and dual diagnoses, and we will not repeat that discussion here (*cf.* Carter and Hall 2012; Skinner and Russell 2012). But beyond that, we can intuitively understand how it is that consuming a drug of choice could seem to make sense. As an addict from rural Indiana recently commented, injecting Opana (a new prescription opioid) eases his withdrawal enough to allow him to get out of bed (*cf.* Goodnough 2015). Even without being addicts ourselves, we can appreciate the painful desperation that drugs can relieve, at least for a bit. But is this behavior rational?

4 See Henden *et al.* (2013) for a similar approach to responsibility in addiction, but a different suggestion for how to understand it.

The number of people who died from a drug overdose in neighboring Ohio has quadrupled since 2002, when the state starting keeping these statistics. Last year, the number was more than double those who died in a car accident. Overdose was the most common injury-related cause of death, and it was a more common cause of death than breast cancer, prostate cancer, or Parkinson's disease. The vast majority of those deaths (about 75%) were from opiates (*cf.* Balmert 2015; Ohio Department of Health 2015). Let us couple these sorts of statistics with the fact that addicts do not want to be addicted any more than a diabetic or a heart patient wants to be ill (*cf.* Charland 2002; Hyman 2007; Leshner 1999). If we stack the chance of overdose from injecting an unregulated street drug and the desire not to be addicted against, say, calling for professional medical help, I think we can all agree that injecting the drug to dull emotional pain is not the choice most conducive to survival. In short: addictive behavior is not a rational decision, even when it might be an understandable one.

Recognizing the physical and psychological distress of withdrawal and cravings coupled with the damage done to the reward system and executive functioning, it is very easy to fathom why an addict will continue to use. However, there is a difference between behavior being comprehensible and decisions being rational, at least from a traditional philosophical perspective on rational choice, which holds that the most "choice worthy" action is the one that maximizes the expected utility (*cf.* Mill 1861). But, as noted above, continuing to use does not maximize expected utility against the background of knowledge available to the person with addiction. It does not even approach this criterion.

Before she died of a heroin overdose the day after she was released from jail, Desi Sandlin wrote, "I cry every fucking day because the thing that ruined my life I still have cravings [for] […] How can I hate it so much then still want it?" (Cherkis 2015 n. pag.). Perhaps, the problem with addicts is not just an unwillingness to behave responsibly or rationally. Perhaps Sandlin's writings express what it is like to be constrained by one's brain. Perhaps the problem is in how we actually assign blame and responsibility.

Recent work in social psychology has indicated that how we assign responsibility is tied to prior judgments of what counts as morally bad behavior, which are in turn dependent upon other, larger, social and cultural factors. Mark Alicke's culpable control model of blame is a good exemplar of this approach (*cf.* Alicke *et al.* 20011; Alicke 2000, 2008). "Culpable control" refers to the fact that our desire to blame someone intrudes on our assessments of that person's ability to control his or her thoughts or behavior.

Deciding that someone is responsible for something, which is taken to be the conclusion of a judgment, is actually part of our psychological process of assessing blame. If we start with a spontaneous negative reaction to some action, then that can lead to our hypothesizing that the source of the action is blameworthy as well as to an active desire to blame that source. This desire, in turn, leads us to skew our interpretations of the available evidence such that it supports our blame hypothesis. We highlight evidence that indicates negligence, recklessness, impure motives, or a faulty character, and we ignore evidence that suggests otherwise. In other words, instead of dispassionately judging whether someone is responsible, we validate our spontaneous reaction of blameworthiness. Data suggest that we will even exaggerate a person's actual or potential control over an event to justify our blame judgment or we will change the threshold for how much control is required for a blame judgment (*cf.* Alicke *et al.* 2008; De Brigard *et al.* 2009; Lagnado and Channon 2008).

The assignment of responsibility and blame is more about the emotional response of the evaluator than it is the rational application of objective standards. Consequently, in professional settings and philosophical discussions, we would be better off not engaging in this practice at all. We suggest that it would be more useful and more effective to understand human decision-making as the multifarious and complex process that it is, and leave it at that. At bottom, trying to divine who is how responsible for what and under what conditions is a fool's errand; it is not getting at anything meaningful or real from a psychological, sociocultural, or biological point of view.

For those who wish to adopt an evolutionary perspective on free will, it is better to recognize that our best science tells us that human choice is driven by hundreds, if not thousands, of influences and is filtered by brains that have been formed and deformed by genes, environment, and previous decisions and behavior. Never mind the fact that evolution only cares about getting genes into the next generation. It is not interested in whether individual decisions maximize survival over the long run. It is even less interested in whether decisions promote wellbeing. Philosophy's versions of free will might be moribund, but so too are the biologically driven ones. The concept itself is simply inapplicable to the complex social, psychological, biological creatures that we are.

Theoretically, under ideal conditions, our brains can select the best action under the circumstances to promote our wellbeing. But most of the time, we operate under much less than ideal conditions. Addiction is a clear example of this. So too, we would submit, are those with many of today's chronic illnesses. Or those with abrupt disruptions to their environments. Or those trying to make long-term changes in their everyday

routines… The list is long. We suspect that, in the end, it includes all of us. Evolutionary free will is a nice ideal, but we are dubious that it reflects reality.

Coda: Treating Addiction

Moreover, recognizing the truly chronic nature of addiction along with the complexity of human decision-making in real time should inform how we treat it. As Thomas McLellan notes:

> Relapse among patients with diabetes, hypertension, and asthma following cessation of treatment has been considered evidence of the effectiveness of those treatments and the need to retain patients in medical monitoring. In contrast, relapse to drug or alcohol use following discharge from addiction treatment has been considered evidence of treatment failure. (McLellan *et al.* 2000 1694)

Or, we add, evidence of the failure of the addicts themselves. Our current treatment protocols assume exactly the sort of free will and responsibility that we have been arguing against. They assume that if addicts use again, then they were not "ready" to stop. If they really, truly, sincerely, want to stop using, then, with some help, or not, they will.

But the real truth is, we currently have no good model for treating addiction, or even providing for any type of on-going managed care. We really do not know what we are doing where addiction is concerned. Consider: in a recent study, patients with substance dependence disorders were randomized into two groups. One received extensive longitudinal care coordinated by a primary care clinician, motivational enhancement therapy, relapse prevention counseling, on-site medical, addiction, and psychiatric treatment, social work assistance, and referrals –basically, this group received every possible treatment we suspect might be relevant to controlling addiction. The control group received an appointment with a primary care physician and a list of treatment resources with phone numbers. After a year, the researchers found no significant differences in drug or alcohol consumption between the care management group and the control group, nor were there significant differences in secondary outcomes, like the severity of the addiction or the health-related quality of life (*cf.* Saitz *et al.* 2013).

The way we approach addiction, like the way we approach other chronic illnesses, and the way we assign responsibility and blame, does not reflect what we know about human neuropsychology. It should. We need a new approach that accepts our limitations in rational decision-making and conforming action to long-term and short-term goals, that eschews discussions of responsibility, and that recognizes the diversity of influences on human behavior. In short: we need a new approach

that presumes humans rarely select the most adaptive path available to them, an approach that recognizes that humans are rarely free.

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