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The Neuroscience of Freedom and Creativity: Our Predictive Brain. Joaquín M. Fuster. Cambridge: Cambridge University Press, 2013, 282 pages, $29.99 paperback.

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Since the turn of the last century, an ever-increasing number of psychologists and biologists have argued that action and perception are fundamentally connected to each other. We perceive so that we can act. Joaquín Fuster has pushed this idea for most of his career in neuroscience, collecting evidence that not only do perceptual processes and decisions to act inform one another, but also that all of cortical memory is comprised of ever-changing, distributed patterns of connections among neurons that have been defined by experience. These patterns, which he calls "cognits," are hierarchically organized by depth of complexity and increasing abstraction. These memory networks connect neurons across discontinuous cortical regions of prefrontal and posterior association cortex. And they overlap each other, such that individual neurons can play a role in many different memory networks.

This view stands in contrast to what had been the mainstream cognitive science assumption: perception, thought, and action each is separable from the other. According to this view, discrete cortical areas are devoted to specific cognitive functions; cognition is modular, in other words. For example, there are single regions concerned with facial recognition or motor programming or working memory, which are only sparsely connected to one another. Fuster has repeatedly argued that his networked model, with its tightly interconnected yet fluidly determined circuits, is an empirically more viable model.

In *The Neuroscience of Freedom and Creativity: Our Predictive Brain*, Fuster takes his perspective one step further and examines what it would mean for our understanding of freedom. If the mind just is a collection of cognits, then in what sense, if any, do we have a free will, or any other sorts of freedom? Fuster outlines his answer in the book, as well as commenting on what this view means for the larger political economy.

Fuster has been an innovative force in cognitive neuroscience. His research has been groundbreaking. He is less adept at translating his science into philosophy, however. In particular, he appears to make the common error of trying to use scientific theories to justify his preconceived notions of how things should be, instead of letting the science lead him. He is not the only one who makes this type of mistake. Indeed, most people who try to save freedom from deterministic science end up being at cross-purposes with themselves.

I use addiction as a case study to explore how this plays out in *The Neuroscience of Freedom and Creativity*. I chose addiction as an example because much of what Fuster says about it is spot-on and important. But in the end, his view of addiction, like his view of

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freedom, becomes untethered from the data and winds up being over-simplified. Let us start first by reviewing how he understands decision-making and choice in our brains.

The Always Changing Mental Landscape of Cognits

Here is how Fuster views human cognition. The association areas in the posterior regions of cortex house our perceptual cognits, networks of neurons sensitive to sensory information. The frontal association cortex contains executive cognits, networks of neurons sensitive to information related to decisions and action. In both regions, cognits are hierarchically organized. At the bottom of that organization and in simpler animals, cognits are small and relatively uncomplicated, representing minimal percepts and motor actions. At the top and in humans, cognits are more complex and represent information abstracted from more basic perceptual or executive cognits.

In primitive animals, the perception–action cycle is circular, with little to no internal feedback from motor output neurons to sensory receptors. But in higher animals, posterior and frontal networks are linked by long reciprocal cortico-cortical connections, which support a dynamic perception–action cycle for sequential behavior, like those found in speech and problem-solving. The primitive sensory-motor circle has been replaced by a perception–action cycle that includes more phylogenetically and ontogenetically advanced structures, including the prefrontal cortex. This, more complex, perception–action cycle can start anywhere: in the internal or external environment, in the posterior or the frontal regions. Moreover, complex, goal-directed behavior can mobilize several perception–action cycles at the same time (or in sequence).

Cognits form the basic units of long- and short-term memory. Each new memory becomes associated and integrated with concurrent stimuli and pre-existing memories at other levels. Moreover, if sensory input starts a perception–action cycle but the subsequent action cannot be immediately completed, then short-term or working memory bridges the temporal "gap" in the cycle, holding information online until it is needed. The activity of working memory occurs in the prefrontal cortex but the items held and used by working memory are stored elsewhere. Working memories have the same structure and location as the long-term memories to which they refer, for they are just the temporary reactivation of the long-term memories, held in the cognits of cortex. In general, cognits follow Hebbian learning principles and are formed through temporally coinciding synaptic events. In Fuster's theory, however, cognits can also be created after just a single event and modified by just a single input.

The bottom line is that all decisions or choices that our cortex makes are completely embedded in some perception–action cycle or other. "In that manner . . . we choose our guiding memories and principles, our venue and means of expression, our friends and mates, our goals and rewards" (p. 108). Consequently, according to Fuster's view, as with most biologically plausible views of decision-making, there is no "autonomous and mysterious" center that is the repository of free will.

If this is the case, is there any sense in which we can say that we are free? Fuster answers yes. And the point of the book is to explain how and why.

Freedom to Choose

Fuster thinks of freedom as a freedom to choose. Our freedom is the freedom "to choose, to think, to plan, to decide, to do, to undo, or not to do" (p. 111). It is the ability of our cortex to deliberate and make choices among alternatives. "Our brain is free to choose and act, naturally within the constraints of the nervous system itself and the world in which we live" (p. 110). Our cortex selects at every moment among innumerable options. Fuster believes that the more choices we have, the more freedom we have.

Of course, each decision is nothing more than a series of causes and effects, determined by the laws of physics and chemistry. One would think that, therefore, there is little room for anything resembling traditional notions of free choice. Fuster, however, thinks that the possibility for freedom "expands" because "most neural transactions, especially in the cerebral cortex, are nonlinear and probabilistic, not strictly determined by the self, but by changes around us and by decisions of others" (p. 137). The room for free will grows even more "if we take into account the enormous multiplicity of influences weighing on, if not determining, almost all our decisions" (p. 137). It is this view that I shall challenge: that the more complex the causal pathways to a decision, the freer the decision is.

Fuster is not alone in his view. Several philosophers argue for a similar perspective: that we should understand freedom from a pragmatic, functional perspective; that free will just is our ability to choose effectively in an ever-changing environment (cf., Banja, 2015; Dennett, 1984; Nahmias, 2006). Free will reflects of our capacity to select the most adaptive behaviors from a range of possible behaviors before us. As Fuster puts it: "We are free inasmuch as the PA [perception-action] cycle, which joins us to the environment, can lead us by choice between alternatives to high probabilities of success and low probabilities of failure" (p. 110).

Such a view of free will is quite far from what philosophers normally mean when they discuss the concept. Perhaps John Banja states 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" (2015, p. 10). Instead of defining freedom as being able to do or choose otherwise under the identical conditions, Fuster, like Banja, ties freedom to current actions and actual possibilities. This does not mean that freedom is connected to conscious deliberation, however; according to Fuster, choices are "to a large extent biased if not determined by some circumstances of the moment and some unconscious motives" (p. 186). Just your brain being able to choose among alternatives is enough for freedom.

Where Fuster departs from other philosophers who promote this view is in how he applies it to particular cases. In particular, he diverges from others in the analysis of decision-making in cases of addiction. Do persons with addiction freely choose to use, or do they become slaves to their substances of abuse? Most philosophers hold that persons with addiction remain free; Fuster does not. The differences between the two analyses are instructive. A slightly deeper dive into the neuroscience of addiction will help us understand addiction's neurobiological mechanisms as well as why this view of freedom as choice is, at bottom, problematic.

The Freedom to Use

Equating freedom to choice for most philosophers means that persons with addiction are responsible for their actions, for at each choice point, there is a very real sense in which the individual could have elected not to use or abuse. Such a perspective is not out of bounds — indeed, most psychologists believe that persons with addiction are responsible for the consumption of their drugs of choice (Buckwalter, 2014; see also Uusitalo, 2015), and most community-based treatment programs are predicated on the idea that persons with addiction can and do choose to use; hence, they can choose to stop. Insofar as people can alter their behavioral choices in response to environmental information such that they are able to select the most adaptive or useful, then that ability points to a version of freedom that they have. They are free in so far as they *can* choose

behaviors that promote survival over those that do not (or do not as much), regardless of whether they *actually* make the most adaptive choice.

In contrast, Fuster holds that, "Drug addiction is the clearest example of freedom mindlessly exercised to its own demise" (p. 116). The reason is that, "As dependency increases in the addict, the PA [perception–action] cycles guiding normal behavior drop out. At the same time, the cycle of addiction to the drug restricts itself ever more tightly in drug-seeking behavior. That behavior becomes associated with concomitant sensory stimuli in the creation of new cognits. . . . By . . . associative retrieval, the stimuli by themselves trigger irresistible drug-seeking behavior; a pathological perception–action cycle driven by positive feedback" (p. 116). In sum, addicted persons' "decisions are far from free, in that there are few alternatives to their abnormal behavior" (p. 129).

Fuster's views echo the tenets of the so-called disease model of addiction. He is in good company, in that the disease model of addiction has received widespread support across a range of expert institutions, including the United States National Institute on Drug Abuse (1999, 2009), the World Health Organization (2004), and the American Psychiatric Association (2013). The position of the United States National Institute on Alcohol Abuse and Alcoholism (2013) 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."

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 (2015), asserts that "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." For each of these institutions, addiction is seen as a chronic, neurobiological pathology that robs its victims of their ability to control their behavior with respect to their substances of abuse.

Indeed, some researchers consider a failure to inhibit drug-seeking and drug-consuming behaviors the very definition of addiction (e.g., Fillmore and Weafer, 2004; Finn, Sharkansky, Brandt, and Turcotte, 2000; Lyvers ,2000). Others, however, do not find this view persuasive. Echoing the arguments of Thomas Szasz (1974a, 1974b) to distinguish between those "disabled by living" and true illnesses, Hannah Pickard, for example, claims that addictive behaviors are in fact 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, p. 43). 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" (italics hers, p. 42). True actions — and not reflex or automatic movements — require alternatives in behavior. True actions require choice, but genuine choice belies the notion of compulsion.

Pickard supports this conceptual argument with biological considerations. She contends that there is nothing in neurobiology that 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 (see also Dill and Holton, 2013). Nor does the neurophysiology of addiction suggest that control gets lost.

I believe, though, that Pickard's descriptions are inaccurate. 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, clinicians use "compulsion" to refer to behaviors that persevere despite adverse consequences or despite being the incorrect response in choice situations (the term can also refer to the persistent re-initiation of a habitual behavior; cf., Everitt and Robbins, 2005). Persons with addiction are compulsive in this sense of the word; Fuster is right about this.

However, what I find significant about addiction is not the compulsive drug-seeking behavior, but rather that the affective–cognitive functioning of the individual as a whole is significantly impaired. Hence, Fuster's suggestion that "drug addition is equivalent to adding a new drive to the organism, as compelling as any other, and more destructive than all" (p. 116) is not quite accurate either. Addiction is not a new drive in an otherwise normal brain, nor is it "an all-consuming PA [perception–action] cycle that heavily restricts the freedom of the patient" (p. 116), as he also suggests. Instead, addiction changes the very structure of the brain, altering the possibilities of perception-action cycles.

Brain-imaging studies of persons with addiction show physical changes in areas of the brain associated with judgment, decision-making, learning, memory, as well as with inhibitory control (Fowler, Volkow, Kassed, and Chang, 2001). Cortical degradation in persons with addiction underlie impairments in problem-solving and cognitive flexibility, which are also relevant to understanding why persons with addiction behave the way they do (Fein, Klein, and Finn, 2004; Fein, McGillivray, and Finn, 2006; Pfefferbaum, Desmond, Galloway, Menon, Glover, and Sullivan, 2001). In addition, they have difficulties with evaluating their environment and then 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. These changes impact the rate, amount, and time of addictive consumption, but they also affect a whole range of other activities. For example, intoxication, reduced impulse control, and aggression are highly correlated with a range of chemical addictions.

Nevertheless, these changes to executive and cognitive functioning do not support the view that persons with addiction are not free to choose, as one might think. Banja (2015) argues that persons with addiction do indeed freely choose to use because they could engage in practices that would diminish the chances that they would act on their cravings, yet they do not. Prior decisions to control behavior might entail that later decisions and actions were not compelled. 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 drivers 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 decide to forgo purchasing chocolate at the grocery store now to prevent myself from absent-mindedly eating it later in the evening while watching TV. I have freely and deliberately arranged my environment such that acquiring candy at a later decision-point becomes more onerous, which would then influence my decision about eating the sweets. The suggestion is that we freely choose 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 persons with addiction could choose to arrange their environments such that they can't use.

Can persons with addiction actually choose in this manner? I 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.

Directly opposed to Pickard and Banja, and in line with Fuster, I advocate taking the idea that addiction is a complex chronic illness very 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 (see also Levy, 2013). So have type 2 diabetes mellitus, hypertension, and adult-onset asthma (McLellan, Lewis, O'Brien, and Kleber, 2000). Tolerance (or intolerance) for alcohol, for example, appears heritable (Chao, Kiou, Chung, Than, Hsu, Li, and Yin, 1994; Newmark, Friedlander, and Thomasson, 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 (e.g., Mitchell, Kammerer, Blangero, Mahaney, Rainwater, Dyke, et al., 1996; Svetkey, McKeown, and Wilson, 1996), just as are addicted persons' 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.

Importantly, diabetes, hypertension, and asthma require continued care through the patients' lifetimes. There are medical treatments for these ailments, 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 (Clark, 1991; Dekker et al., 1993; Graber et al., 1992).[1] 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 (Clark, 1991; Dekker, Dielemann, Kaptein, and Mulder, 1993; Graber, Davidson, Brown, McRoae, and Woolridge, 1992; Schaub, Steiner, and Vetter, 1993). These rates are virtually identical to what we find with persons with addiction: somewhere between 40 and 60% of patients treated for alcohol or drug dependence return to active use within a year of some treatment intervention (Finney and Moos, 1992; Hubbard, Craddock, Flynn, Anderson, and Etheridge, 1997; McLellan and McKay, 1998).

The point here is that for persons with addiction, prior control of their decision-making regarding whether to consume, is virtually identical to what we find in other complex, chronic illnesses. These patients are not 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 ill is that they have repeatedly made very poor decisions about their behaviors and now have to suffer the consequences.

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 United States file for bankruptcy during their first 12 years after retirement, despite having earned an average of $3.2 million (in 2012 dollars). Neither the amount of money earned nor the years spent playing affect the likelihood of filing for bankruptcy (Carlson, Kim, Lusardi, and Camerer, 2015). This rate is comparable to the bankruptcy rates for all Americans of the same age. Even though pro-football players, unlike most young adults, accumulate great wealth, many fail to organize their environments such that they would have appropriate resources upon retirement. And they do this, despite knowing full well that their sports careers are likely to be brief.

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[1]And, just as with addiction, outcomes are poorest among those with low socioeconomic status, few family or social supports, or other psychiatric disorders (Gerstein and Harwood, 1990; McLellan et al., 1994; Moos, Finney, and Cronkite, 1990; National Institute on Drug Abuse, 1999).

But there is nothing special about football players' lack of ability to translate sudden wealth into financial security. Lottery winners fare even more poorly; they file for bankruptcy at twice the rate of the broader population (Hankins, Hoekstra, and Skiba, 2011). The United States 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 plans 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 (National Center for Health Statistics, 2015; National Institute of Health, 2012); 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 are not very good at sacrificing short-term rewards for long-term goals, even when operating at full cognitive capacity. Is it theoretically useful to claim that so many of us are freely irresponsible? Perhaps a different way of describing human decision-making and a different approach to understanding freedom is warranted.

Constrained Choices

Fuster is sensitive to the idea that we are not very good at the sort of cognitive control that Banja and Pickard propose. He discusses this concept in terms of "delay discounting": the idea that the depreciation of the value of a reward is positively related to the time that it takes to be received, or, perhaps simpler, it is the devaluing of future outcomes relative to present outcomes. Fuster agrees that delay discounting belies freedom; however, in line with Banja, he believes that this sort of lack of control indicates a weak will and is endemic in contemporary society, instead of reflecting normal human behavior and typical decision-making. It's what wrong with the world today — we are coddled by social programs, and, as a result, we do not learn how to plan effectively and then act upon those plans: "In a very real sense, delay discount and short-term thinking rob the liberty of the modern citizen to plan for his future. The traditional value of saving for security is sabotaged by official assurances and insurances" (p. 123). For persons with addiction, the implications can be deadly: "the lack … of responsibility leads to enslavement, a total and sometimes fatal loss of liberty" (p. 213).

Perhaps unfortunately, Fuster pushes this line to support of his own political views: "Consumer society has regressed to financial immaturity and short-term decisions aided by the welfare state with its 'entitlements'" (p. 122). But of course when one is discussing biological arrangements that have been set down by evolution, the time course in which we should consider human behavior is much, much longer than Western democratic society. That is barely a blip on our historical timeline and our behavior should be discussed and measured across our full history, not just the past 250 years. Indeed, so far as I can tell, no one really knows what causes delay discounting (cf., Angott, 2010), though its effects are well documented across a spectrum of human cultures and decision-making types. Why we discount delays in reward is, of course, an empirical question, and we should treat it as such instead of using it to support a broader agenda.

So: here is where we stand. Humans, in general, are very poor at selecting the best option for action among a range of choices. In particular, we are very poor at postponing gratification when it would be better in the long run to do so. This is true of persons with addiction. It is also true of the rest of us. For reasons unknown — though it is tempting to dream up just-so stories of how our decision-making faculties evolved when life was nasty, brutish, and short — this is just how we are. There seems to be consensus among those who advocate for a view of freedom based on making adaptive behavioral choices

from a range of alternatives including that being constrained in decision-making reduces one's freedom. The more constrained we are, the less free we are. I conclude: given this definition of freedom, and given what we know about human psychology, we are not terribly free creatures.

Let me suggest that it might be more useful for science and theoretically 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 freely deciding and under what conditions is a fool's errand; it is not getting at anything meaningful from a psychological, sociocultural, or biological point of view. 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, and then end there. Philosophy's traditional 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.

Fuster has written an accessible book that describes his views on the interconnectedness of perception, memory, and action quite well. His work has been part of a larger theoretical perspective and research agenda that has fundamentally reshaped cognitive neuropsychology over the past several decades. However, his foray into philosophical accounts of freedom is less successful. In the end, I do not see this as his failing. Rather, the concept of human freedom itself is incoherent, and Fuster's book does much to illustrate the great difficulties one has in trying to wedge this idea into science. Ultimately, I believe that this is a project that is doomed to disappoint.

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* [DSM–5, fifth edition]. Washington, DC: American Psychiatric Association.

Anderson, N. (2012). Three ways NOT winning the lottery may make you wealthy. *Forbes* 4.5.2012. http://www.forbes.com/sites/financialfinesse/2012/04/05/three-ways-not-winning-the-lottery-may-make-you-wealthy/

Angott, A.M. (2010). What causes delay discounting? Ph.D. dissertation, Department of Psychology, University of Michigan. http://deepblue.lib.umich.edu/bitstream/handle/2027.42/78754/aangott_1.pdf?sequence=1, Accessed 10 September 2015.

Banja, J. (2015). "Could have chosen otherwise under identical conditions": An evolutionary perspective on free will. *American Journal of Bioethics: Neuroscience*, *6*, 3–11.

Buckwalter, W. (2014). The folk psychological basis of dualistic psychiatric approaches. Presentation at the Early Career Scholars' Conference in the Philosophy of Psychiatry: Overcoming Mind–Brain Dualism in 21st Century Medicine, Pittsburgh, Pennsylvania.

Carlson, K., Kim, J., Lusardi, A., and Camerer, C.F. (2015). Bankruptcy rates among NFL players with short-lived income spikes. *National Bureau of Economic Research*, NBER Working Paper No. 21085.

Chao, Y.-C., Kiou, S.-R., Chung, Y.-Y., Than H.-S, Hsu, C.-T., Li T.-K., and Yin, S.-J. (1994). Polymorphism of alcohol and aldehyde dehydrogenase genes and alcoholic cirrhosis in Chinese patients. *Hepatology, 19*, 360–366.

Clark, L.T. (1991). Improving compliance and increasing control of hypertension: Needs of special hypertensive populations. *American Heart Journal*, *121*, 664–669.

Dekker, F.W., Dielemann, F.E., Kaptein, A.A., and Mulder, J.D. (1993). Compliance with pulmonary medication in general practice. *European Respiration Journal*, *6*, 886–890.

Dennett, D.C. (1984). *Elbow room: The varieties of free will worth wanting*. Cambridge, Massachusetts: MIT Press.

Dill, B., and Holton, R. (2013). The addict in us all. *Frontiers in Psychiatry, 5*, 1–20.

Everitt, B.J., and Robbins, T.W. (2005). Neural systems of reinforcement for drug addiction: From actions to habits to compulsions. *Nature Neuroscience, 8*, 1481–1489.

Fein, G., Klein, L., and Finn, P. (2004). Impairment on a simulated gambling task in long-term abstinent alcoholics. *Alcoholism: Clinical and Experimental Research, 28*, 1487–1491.

Fein, G., McGillivray, S., and Finn, P. (2006). Normal performance on a simulated gambling task in treatment-naive alcohol-dependent individuals. *Alcoholism: Clinical and Experimental Research, 30*, 959–966.

Fillmore, M.T., and Weafer, J. (2004). Alcohol impairment of behavior in men and women. *Addiction, 99*, 1237–1246.

Finn, P.R., Sharkansky, E.J., Brandt, K.M., and Turcotte, N. (2000). The effects of familial risk, personality, and expectancies on alcohol use and abuse. *Journal of Abnormal Psychology, 109*, 122–133.

Finney, J.W., and Moos, R.H. (1992). The long-term course of treated alcoholism, II: Predictors and correlates of 10-year functioning and mortality. *Journal of Studies of Alcohol and Drugs, 53*, 142–153.

Fowler, J.S., Volkow, N.D., Kassed, C.A., and Chang, L. (2001). Imaging the addicted human brain. *Science and Practice Perspectives, 3*, 4–16.

Fuster, J.M. (2013). *The neuroscience of freedom and creativity: Our predictive brain*. New York: Cambridge University Press.

Graber, A.L., Davidson, P., Brown, A., McRoae, J., and Woolridge, K. (1992). Dropout and relapse during diabetes case. *Diabetes Care, 15*, 1477–1483.

Gerstein, D., and Harwood, H. (Eds). (1990). *Treating drug problems: A study of the evolution, effectiveness, and financing of public and private drug treatment systems.* Washington, D.C.: National Academy Press.

Hankins, S., Hoekstra, M., and Skiba, P.M. (2011). The ticket to easy street? The financial consequences of wining the lottery. *The Review of Economics and Statistics*, *93*, 961–969.

Hubbard, R.L., Craddock, G., Flynn, P.M., Anderson, J., and Etheridge, R. (1997). Overview of 1-year follow-up outcomes in the Drug Abuse Treatment Outcome Study (DATOS). *Psychology of Addictive Behavior, 11*, 261–278.

Levy, N. (2013). Addiction is not a brain disease (and it matters). *Frontiers in Psychiatry*, *4*, 1–7.

Lyvers, M. (2000). "Loss of control" in alcoholism and drug addiction: A neuroscientific interpretation. *Experimental and Clinical Psychopharmacology, 8*, 225–249.

McLellan, A.T., Alterman, A.I., Metzger, D.S., Grissom, G.R., Woody, G.E., Luborsky, L., and O'Brien, C.P. (1994). Similarity of outcome predictors across opiate, cocaine, and alcohol treatments. *Journal of Clinical Consulting Psychology*, 6, 1141–1158.

McLellan, A.T., Lewis, D.C., O'Brien, C.P., and Kleber, H.D. (2000). Drug dependence, a chronic medical illness: Implications for treatment, insurance, and outcomes evaluation. *JAMA* 284, 1689–1695.

McLellan, A.T., and McKay, J. (1998). The treatment of addiction: What can research offer practice? In S. Lamb, M. Greenlick, and D. McCarty (Eds.), *Bridging the gap: Forging new partnerships in community-based drug abuse treatment* (pp. 147–185). Washington, D.C.: National Academy Press.

MedicineNet.com. (2015). Alcohol abuse and alcoholism. http://www.medicinenet.com/script/main/art.asp?articlekey=145076. Accessed 21 April 2015.

Mitchell, B.D., Kammerer, C.M., Blangero, J., Mahaney, M.C.,Rainwater, D.L., Dyke, B., Hixson, J.E., Henkel, R.D., Sharp, R.M., Comuzzie, A.G., VandeBerg, J.L., Stern, M.P., and MacCluer, J.W. (1996). Genetic and environmental contributions to cardiovascular risk factors in Mexican Americans. *Circulation, 94*, 2159–2170.

Moos, R.H., Finney, J.W., and Cronkite, R.C. (1990). *Alcoholism treatment: Context, process and outcome.* New York: Oxford University Press.

Nahmias, E. (2006). Free will as knowledge. Unpublished manuscript. http://www2.gsu.edu/~phlean/papers.html.

National Center for Health Statistics. (2015). *Health, United States, 2014: With special feature on adults aged 55–64.* Hyattsville, Maryland: Center for Disease Control and Prevention.

National Institute of Health. (2012). Overweight and obesity statistics. NIH Publication No. 04-4158. http://www.niddk.nih.gov/health-information/health-statistics/Pages/overweight-obesity-statistics.aspx. Accessed on 6 May 2015.

National Institute on Alcohol Abuse and Alcoholism. (2013). NIH study identifies brain circuits involved in learning and decision making. Press release. http://www.niaaa.nih.gov/news-events/news-releases/brain-circuits-learning-decision-making. Accessed on 21 April 2015.

National Institute on Drug Abuse. (1999). *Principles of drug addiction treatment: A research based guide.* NIH publication 99-4180. Bethesda, Maryland: National Institutes of Health.

National Institute on Drug Abuse. (2009). *Principles of drug addiction treatment: A research-based guide.* Bethesda, Maryland: National Institute on Drug Abuse.

Newmark, Y.D., Friedlander, Y., and Thomasson, H.R. (1998). Association of the ADH22 allele with reduced alcohol consumption in Jewish men in Israel: A pilot study. *Journal of Studies of Alcohol and Drugs, 59*, 133–139.

Oscar–Berman, M., and Marinkovic, K. (2007). Alcohol: Effects on neurobehavioral functions and the brain. *Neuropsychological Review, 17*, 239–257.

Pfefferbaum, A., Desmond, J.E., Galloway, C., Menon, V., Glover, G.H., and Sullivan, E.V. (2001). Reorganization of frontal systems used by alcoholics for spatial working memory: An fMRI study. *Neuroimage, 14*, 7–20.

Pickard, H. (2012). The purpose in chronic addiction. *AJOB Neuroscience, 3*, 40–49.

Schaub, A.G., Steiner, A., and Vetter, W. (1993). Compliance to treatment. *Journal of Clinical Experiments in Hypertension, 15*, 1121–1130.

Schuckit, M.A. (1994). Low level of response to alcohol as a predictor of future alcoholism. *American Journal of Psychiatry, 151*, 184–189.

Schuckit, M.A., and Smith, T.L. (1996). An 8-year follow-up of 450 sons of alcoholics and controls. *Archives of General Psychiatry, 53*, 202–210.

Svetkey, L.P., McKeown, S.P, and Wilson, A.F. (1996). Heritability of salt sensitivity in black Americans. *Hypertension, 28*, 854–858.

Szasz, T. (1974a). The myth of mental illness. In R.J. Morris (Ed.), *Perspectives in abnormal behavior* (pp. 1–11). Elmsford, New York: Pergamon Press Inc.

Szasz, T. (1974b). *The myth of mental illness: Foundations of a theory of personal conduct.* New York: Harper & Row.

Uusitalo, S. (2015). Addiction, recovery and moral agency: Philosophical considerations. *The International Journal of Alcohol and Drug Research, 4*, 85–89.

World Health Organization. (2004). *Neuroscience of psychoactive substance use and dependence.* Geneva, Switzerland: World Health Organization.