### ADDICTION IS NOT A BRAIN DISEASE

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En los últimos años se ha ido consolidando la idea en el campo médico-psiquiátrico de que la adicción es una "enfermedad cerebral", como ya así recoge el DSM-5. En este articulo se analiza cómo ha surgido y se ha consolidado esta idea, las críticas que ha recibido, las consecuencias profesionales si este modelo se hace hegemónico, junto a los intereses subyacentes al mismo. Se concluye defendiendo la necesidad de mostrar como psicólogos nuestras claras aportaciones al campo de las adicciones, como el de las variables psicológicas que son necesarias para la comprensión de las adicciones, para su prevención, junto con el papel central del tratamiento psicológico por su eficacia en las mismas. También debemos denunciar los reduccionismos, como el que representa el modelo de enfermedad cerebral frente a un modelo biopsicosocial de las adicciones.

Palabras clave: Adicción, Drogas, Enfermedad cerebral, Psicología.

The idea that addiction is a "brain disease" has gradually been consolidated in the medical-psychiatric field over the last years, as it appears in the current DSM-5. In this paper we analyse the way this idea has arisen and been consolidated, as well as the criticisms that it has received, the professional consequences if this model becomes hegemonic, and the underlying interests. The conclusion defends the need to show, as psychologists, our clear contributions to the field of addictions, and the psychological variables that are necessary in order to understand and prevent addictions, as well as the central role of psychological treatment due to its effectiveness. We must also denounce the reductionism that the model of brain disease represents in comparison with a biopsychosocial model of addiction.

Key words: Addiction, Drug, Brain disease, Psychology.

n volume 507 of the prestigious journal Nature, a letter to the editor was published, on 6<sup>th</sup> March 2014, entitled "Addiction: not just brain malfunction" signed by Derek Heim (2014). In the footnote there were 94 signatories, relevant researchers, clinicians, addiction journal editors, treatment centres, etc., from various countries, criticising the considering of "addiction as a brain disease" because "substance abuse cannot be divorced from its social, psychological, cultural, political, legal and environmental contexts; it is not simply a consequence of brain functioning "(p. 40). They insisted that "such a myopic perspective undermines the enormous impact people's circumstances and choices have on addictive behaviours. It trivializes the thoughts, emotions and behaviours of current and former addicts" (p. 40). Some of the signatories are well known people, such as Gerard Bühringer, Nick Heather, Jerome H. Haffe, Stanton Peele, Tim Rhodes, Stephen Rollnick, Robin Room, Roland Simon, Tim Stockwell, etc.

Correspondence: Elisardo Becoña. Universidad de Santiago de Compostela. Departamento de Psicología Clínica y Psicobiología. 15782 Santiago de Compostela. España. E-mail: elisardo.becona@usc.es This is an important issue, central to the conceptualisation of addiction, and one that has clear implications for drug prevention, treatment and policy. Also on the professional role of different professions, such as that of psychology. Unfortunately, in recent years the biological-brain conceptualisation of addiction has taken a reductionist path, because it is being subjected to the clear interests and pressure groups surrounding it and because of the rupture, or distancing, which we are witnessing after decades of fruitful collaboration between different disciplines in the field of addictions.

In these pages we analyse the facts that have led to the current situation and what the future holds from a psychological perspective.

#### WHAT HAS BROUGHT US TO THIS SITUATION? The first approaches to addiction as a brain disease

There have been several models that have dominated the field of addictions throughout history until it became a major social problem, between the 1960s and 1980s in the majority of developed countries.

Already in the 19<sup>th</sup> century different neurologists began to consider that addiction was a brain disease, an idea

which remained partly in force during the 20<sup>th</sup> century in the medical and psychiatric field, especially applied to alcoholism (Kushner, 2010). In the case of alcohol a distinction was made between people who controlled their consumption and those who were not able to do so, the latter beginning to be considered as sick (Jellinek, 1960) and with a genetic predisposition to alcoholism. In later years it was shown that the cause of alcoholism or drug use was multiple (e.g., Edwards, 2002) moving to a biopsychosocial explanatory model (Melchert, 2015).

A more recent origin of this concept of addiction as a brain disease comes from research studies on opiates, carried out especially on animals, since the middle of last century. Subsequently, this was aided by the discovery of brain receptors; the funding of studies within the US government's drug war focused on finding a biological cause for them; and the need to investigate the "responsibility" of individuals (if the individuals are brain sick then they are not responsible for their actions; if they lose their will power or self-control then they are not responsible) (Vrecko, 2010).

#### The North American background concerning the NIDA

No doubt, those who have allowed this model to appear, develop and become established are the North American NIDA (National Institute on Drug Abuse) and several of its directors or individuals related to it since its inception, such as Jerome H. Jaffee, Alan Leshner, Charles P. O'Brien and its current director Nora Volkow. In 1971 Jerome H. Jaffee first occupied the post of Head of the Special Action Office on Drug Abuse Prevention (SAODOP), better known as the Drug Czar. At that time, the United States was at war in Vietnam and had a serious problem of heroin use among returning soldiers. Jaffee thought it would be a tactical victory for addiction to be deemed a brain disease, as this would help to convince the senators of his proposals, using a pragmatic model (Satel & Lillienfeld, 2014).

An important milestone occurred in 1977 when Alan Leshner (1977), director of the NIDA at the time, published an article in *Science* in which he suggested that the best way to conceptualise addiction would be to consider it as a chronic brain disease characterised by relapse. Although he indicated that the onset of drug use was voluntary, its use entailed brain changes at the neurochemical level, with the result that when people wanted to stop using drugs they had problems in succeeding. Therefore the behaviour became compulsive and they relapsed quickly. For him what identified addiction as a brain disease were the changes in the brain structure and function of the individual, so the treatment should be both behavioural and pharmacological. In addition, he attached importance to the social context in drug use because, interestingly, he used the example of what had happened to the soldiers of the Vietnam War who had stopped using heroin upon their return home. Therefore, the use of the expression *psychobiological disease* appears in different parts of that article, including biological, behavioural and social or contextual elements.

Among the most influential American researchers who consider drug use to be a disease is Charles P. O'Brien, a prestigious researcher in the field of psychiatry. For him, addiction is best conceptualised as a disease, although he does acknowledge that not all drug users become addicted and he believes that the best treatment is one that combines medication with behavioural therapy (O'Brien & McLellan, 1996).

But no doubt the person who has most favoured the creation and consolidation of a brain disease model of addiction is Nora Volkow, director of the NIDA since 2003. In 2007, the NIDA published its informative manual "Drugs, brain and behaviour. The science of addiction" which also has a Spanish version (NIDA, 2008) and was updated in 2010 and 2014. It says that "addiction is defined as a chronic, relapsing brain disease that is characterized by compulsive drug seeking and use, despite harmful consequences. It is considered a brain disease because drugs change the brain-they change its structure and how it works. These brain changes can be long lasting, and can lead to the harmful behaviors seen in people who abuse drugs" [...] "Addiction is similar to other diseases, such as heart disease. Both disrupt the normal, healthy functioning of underlying organ, have serious harmful the consequences, are preventable, treatable, and if left untreated, can last a lifetime" (p. 8).

The initial decision to take drugs is voluntary; but when it becomes drug abuse, the individual's ability to exercise self-control becomes extremely poor. This is attributed to the brain changes that affect judgment, decision making, learning, memory, and behaviour control, leading to the compulsive and destructive behaviours that are a result of the addiction. They also consider the existence of risk and protection factors for addiction, recognising that there is no single factor that determines that someone will become

a drug addict. They also consider that genetic factors contribute only 40 to 60% of vulnerability to addiction and indicate that frequent drug abuse leads to the appearance of various mental disorders (the well-known dual pathology defended by Spanish psychiatrists).

On the positive side, addiction is viewed as a treatable disease, but with insistence on its chronicity and the relapse process. Interestingly, when talking about what treatment is effective, they recommend the combination of drugs, when available, with behavioural therapy. It should be noted that for the treatment of most drug addictions there are no effective pharmacological treatments, only psychological treatment (e.g., cocaine, cannabis, etc.), and when there are pharmacological treatments it is usually necessary to use them together with psychological treatment.

In summary, the NIDA has clearly opted to consider addiction as a chronic brain disease characterised by relapse, in a social context, with a clear genetic component (or, more precisely a gene-environment-stress interaction), with significant comorbidity with other physical and mental disorders (Courtwright, 2010; Volkow & Morales, 2015), and much of its data based on animal research. It stresses the central claim of this model that the persistent use of a drug produces long-term changes in brain structure and function.

#### The DMS-5. Addiction is a brain disease

The NIDA model is clearly reflected in the DSM-5 and its conceptualisation of the substance use disorder (SUD): "An important feature of the substance use disorder are the underlying changes in brain circuits that persist after detoxification and occur especially in people with severe disorders. The behavioural effects of these brain changes are shown in repeated relapses and the intense desire to eat when exposed to drug-related stimuli" (APA, 2014, p. 483).

The DSM-5 has introduced significant changes to the DSM-IV (Becoña, 2015; Compton, Sawson, Goldstein & Grant, 2013; Hasin et al, 2013). The three main changes are as follows: a) the cut-off point proposed for the SUD, 2 out of 11 criteria. Several studies indicate that this is a very low cut-off point and should be increased to 4 or 6 criteria, depending on the substance. b) The introduction of the criterion of craving, which has been made by "consensus" and because there are "drugs" for it, even though there is no evidence that it is a central aspect in the case of some drugs. This was put in writing by the

members of the group that developed the DSM-5 for addictions (Hasin et al., 2013). c) The major limitation involved in delimiting in clinical practice whether the person has a SUD due to consumption of a psychoactive drug prescribed by the doctor or if they have it due to taking the drug on their own ("self-medicating") or if they are really an addict (e.g., in the case of morphine). In addition, there is the underlying question of why, in cases where the person takes a drug prescribed for them, they are not diagnosed and if it was not prescribed for them can a person be considered to have a SUD? Where is the reliability in the diagnosis in each case?

Note also that the DSM-5 talks of a disorder, whereas the NIDA talks of brain disease. Clearly this is a huge leap.

### CRITICISM OF THE CONSIDERATION OF ADDICTION AS A BRAIN DISEASE

In recent years there has been strong criticism of the consideration of addiction as a brain disease. The most important article criticising this is by Hall, Carter and Forlini (2015), published in *The Lancet Psychiatry*. It reviews the evidence that exists on the disease model of addiction, analysing studies on animals, neuroimaging studies of people with addictions and research on the role of genetics in addiction, focusing the criticism on five aspects.

The first is whether addiction is a chronic disease. Hall et al. (2015) consider that it is not, since many people with addictions recover without treatment, which is known as "natural recovery" (Stea, Yakovenko & Hodgins, 2015). The best known case, which has already been mentioned, is that of the American soldiers addicted to heroin in the Vietnam War, most of whom stopped using without resorting to treatment when they returned (Robins et al., 2010). Similarly, we have evidence that people addicted to recreational drugs respond to small changes in their personal situations, as shown with the use of incentives (Heyman, 2009). In addition, a significant amount of those who use drugs in adolescence stop using them in adulthood, especially after the age of 25, at which time adult roles are assumed (Becoña, 2002).

The second concerns the animal models of addiction. The existing models of addiction using rats are usually for heroin, with models of self-administration of opioids in standardised and controlled conditions, which bears little resemblance to human behaviour in every situation. In addition, when animals are in enriched environments they have different patterns of self-administration of drugs. For example, rats trained to self-administer drugs refrain from doing so when they can access natural support, such as food or coupling (Ahmed et al., 2013).

The third aspect is about the genetics of addiction. Addiction is not a disorder that occurs only in those who have so-called addiction genes. Studies indicate that genetic prediction is the same as a simple family history of consumption (Gartner et al., 2009). Therefore, genetics is not very informative with regards to addictions today.

The fourth aspects relates to neuroimaging studies in humans. Although these studies show that addicts differ from non-addicts, this appears to be due, at least in part, to the bias produced by the sample sizes and the size differences. In addition, case-control studies do not show whether addiction is a cause or a consequence of the differences in brain structure and function or some combination of the two (Ersche et al., 2013).

The fifth is the increasing complexity of the neurobiology of addiction, with many neurotransmitter systems and many brain structures involved. Therefore epigenetics (changes in gene expression in the brain system that can be caused by drug use) are increasingly important (Volkow & Morales, 2015).

Although one would expect that this model would lead to the development of effective drug treatments, this has not happened. Let us remember failures such as the vaccines for different drugs, newer drugs with low results (e.g., Nalmefene), ineffective brain surgery for addicts, etc. Huge amounts of money are involved in this research and it is forgotten that simple and inexpensive measures, such as restrictive legislation regarding alcohol or tobacco, or measures such as increasing taxes, are effective, efficient and cheap (Babor et al., 2010).

Another notable criticism of the brain disease model of addiction is that of Satel and Lillienfeld (2014). For them, this model wrongly implies that the brain is the most important level of analysis and most useful in understanding and treating addictions. This obfuscates the dimension of choice in addiction, the ability to respond to incentives, and the fact that people use drugs for various reasons. This is exemplified with the aforementioned study by Robins et al. (2010), which points out that only 5% of soldiers addicted to heroin who returned from Vietnam, relapsed within 10 months of returning home, and 12% relapsed briefly in a follow-up of 3 years. At the time these results were considered revolutionary, but it seems that today their importance has been forgotten, since the definition of addiction based on the conceptualisation of brain disease implies the chronicity of this condition.

Articles

Satel and Lillienfeld (2014) criticise psychiatry for using the terms disorders or syndromes, and not diseases, for psychiatric disorders in general, so it does not make sense to talk about brain disease, but rather brain disorder. The brain and the mind cannot be considered independently, as if one were on one side and the other on the other. A feeling, a thought, a desire, produces a change in neurons and brain circuits, and the brain does not act on its own. Anyway, the DSM-5 is already going in another direction.

Other criticisms in the same line can be found in Hammer et al. (2013), Levy (2013), Pedrero (2015), Trujols (2015), etc.

#### WHY HAS THIS MODEL ADVANCED SO FAST?

It is strange that a model that is so weak due to the data that support it, as we have discussed, while very suggestive, due to its simplicity and reductionism, has advanced so quickly. In our opinion, after it was formulated and sponsored by the NIDA in the United States, it has expanded both there and in other countries, including Spain, for several reasons, which we indicate briefly below.

1) Generous funding, from the NIDA, to research that supports the model of brain disease and the clear assumption of a medical model of addiction, based on a biological substrate in the brain.

We have already mentioned that the NIDA is prioritising research in this field and in this line especially as it is the agency that finances 85% of all drug research worldwide. In addition the DSM-5 of the American Psychiatric Association and the majority of scientific societies in the field of addictions have assumed this model, and these are usually biologicists, with all that this implies. In Spain the situation is similar, with an enormous advance of this model due to the underlying financing, its simplicity, the interest of pharmaceutical companies and the revolution in genetics accompanying in parallel to this model.

### 2) The interest of the pharmaceutical industry to consolidate this model.

Pharmaceutical companies have fertile ground in this model, as there are a large number of addicts and it is a good business opportunity, so much effort has been

devoted to it over the years. However, the results of drug therapy have been disappointing, since no new molecules appear to be useful for the treatment of addictions. And, at the same time frequent conflicts of interest appear among scientists and researchers as their declarations go beyond what the data indicates.

As Allen Frances (2013), chairman of the working group DSM-IV and an internationally renowned psychiatrist, says in his book *Are we all mentally ill?* "the commercialisation of disease cannot occur in a vacuum, it requires the pharmaceutical companies to have the active collaboration of the physicians that issue prescriptions, the patients that request them, the researchers that invent new mental disorders, ... A constant, ubiquitous and well-funded campaign in favour of "raising awareness of the disease" can create diseases where none previously existed. Psychiatry is especially vulnerable to the manipulation of the lines separating normality from disease because it lacks biological tests and greatly depends on subjective judgments that can be influenced by clever marketing" (p. 50).

The field of addictions is one of the fields in which it is easiest to find conflicts of interest with the pharmaceutical industry. The relationships of associations with the industry are often built with people who exercise leadership in these associations (Lichter, 1998). Often part of the curriculum of these leaders has been achieved based on their personal relationship with the industry, in so-called "special interest groups" (e.g., boards of scientific and professional societies, scientific journal editors or editorial boards, members of the elaboration of clinical guidelines). Thus, in the DSM-5 there have been significant problems of conflict of interest with many participants who were linked to the pharmaceutical industry (Cosgrove & Krimsky, 2012).

## 3) The social construction processes of diseases and the case of addictions.

It is society that gives the label of disease to a particular condition; that is, the disease is a social construction. In recent years we have witnessed a growing creation of new diseases or disorders and the resulting increasing medicalisation of abnormality (e.g., ADHD, bipolar disorder, Internet addiction, etc.). Therefore, the idea that we have socially regarding drugs will lead to the adoption or not of social measures, to the medicalisation or not of their consequences, to considering whether or not they are a disease, whether their consumption entails negative

It is the individuals and groups who contribute to constructing the reality and perceived social knowledge (Berger & Luckman, 1966). Unlike the medical model, which assumes that diseases are universal and unvarying in time and place, the social constructionists emphasise how cultural and social systems shape the meaning and experience of falling ill (Conrad & Barker, 2015). This is especially clear in mental disorders, because getting ill, being ill, has both biomedical and experiential dimensions; some diseases are eminently social or cultural, some are stigmatised and others are not; some are considered disabilities and others are not. For example, dependence on antidepressants is authorised and dependence on other drugs is not (Kushner, 2010); the same occurs with Ritalin, a drug stimulant for the treatment of hyperactivity; SSRIs and ecstasy both act on the same receptors of serotonin. One would not produce a brain disease and the other would.

This has clear social and health implications, such as the recognition of disabilities, access to health care, creating research on the "disorder" or "disease", etc. But when it is not a "real" disease there is a risk that this will lead to its medicalisation. This has been encouraged in recent years by the pharmaceutical industry (Loe, 2004) which even goes as far as to create the need for its products in individuals through aggressive advertising (of drugs, of course). A current example is the DSM-5 conception of alcoholism.

## 4) The psychological processes underlying the proponents of this model.

The people who opt for the brain model of addiction have previously been professionally trained to understand people, their patients and the world in a certain way, usually facilitating biological reductionism or seeking the ultimate cause of a phenomenon in biological functioning. This in itself is neither good nor bad. But when the model is not entirely clear (e.g., it is not the same to say the cause of the flu is a specific virus, as it is to say the cause of addiction is the abnormal functioning of dopamine in the brain), and when individual professional and commercial factors may be present, it can lead to bias. For example, professional biological identification facilitates more a praxis guided by this model, together with professional prestige, with a specific methodology

and therapy, different from others, and with a biological interpretation of the results obtained.

In this sense, recent years have seen the passage of a growing number of medical field professionals working in addictions from a biopsychosocial explanatory model of "addiction" to a biological model (that of brain reductionism). Having a specific model, when it is useful, is good; but when it is reductionist and only partially explains part of the phenomenon, it is often inadequate and harmful to the users. This has been favoured because North American psychiatry -and also official psychiatry in Spain- is opting clearly and resoundingly for addiction as a brain disease, and although we know that this is not the opinion of all psychiatrists and physicians working in addictions, it is the dominant one at this time in the official documents of various associations and scientific journals on addictions. The worrying thing about it is the attempt to psychiatrise the conceptualisation of addiction and treatment, as if it were just another biological illness. We experience a clear example of this in Spain with the dual diagnosis, because if the person has a "disease" then psychiatric treatment (pharmacological, naturally) is "always" justified for the condition; which means forgetting about the problems that come with psychiatric overmedicating, increasingly criticised (Whitaker, 2015). But when a person assumes a model, due to their life history, learning, necessity or consistency, there are

nistory, learning, necessity or consistency, there are several psychological processes that accompany them, and that we psychologists know well, such as selective attention, the effect of conformity (to the dominant group) and social pressure, confirmatory bias, selective attribution, self-fulfilling prophecy, moral license, the group identity (professional), decision-making and, above all, the process of reinforcement.

As an example of the above, the power of reinforcement applied to the actors involved in expanding this model is clear: they tend to be comfortable and consistent with it (learning history), with the idea of reducing all the symptoms to one illness, to be in a clearly identified professional group (reductionism and simplicity); and, most importantly, there is a clear reinforcement to assume it, in the form of self-reinforcement and external reinforcement (from colleagues, society, the pharmaceutical industry, patients, etc.). If they do not accept the dominant model they will face negative consequences or exclusion. In addition, there is a modelling effect because the people with the most prestige in their profession are the leaders of the movement.

This does not mean we do not recognise the value and the role the individual biological weight clearly has on having or not having an addiction. But it is not the only "cause" nor is it possible to explain all aspects of addiction only through biology. What we are criticising is precisely the reductionism of this model and the forgetting of the central weight of other factors, such as cultural, social and environmental factors (e.g., availability, social attachment), psychological factors (e.g., expectations, learning, self-control, personality), individual factors (e.g., sex, age), etc.

## THE FUTURE OF THIS MODEL FROM THE PSYCHOLOGICAL PERSPECTIVE

There have been many contributions of psychology to the understanding, assessment, prevention and treatment of addictions. Naturally, from a psychological or biopsychosocial model, our professional training leads us to understand human beings in a comprehensive way, not biased or reductionist. The psychological contribution to the understanding and treatment of addictions has been and remains clear, highlighting for example motivational techniques, techniques of psychological dishabituation and techniques of relapse prevention, among others (Becoña, 2016). Therefore, the brain model of addiction, due to its reductionism, is not acceptable from the psychological perspective, and although we do not deny the role of biology, we do deny its exclusivity and its simplistic attempt to understand the complex phenomenon of addictions. As Hall et al. (2015) say, "Addiction is a complex biological, psychological and social disorder that needs to be addressed by various clinical and public health approaches" (p. 109).

The future is always open and we cannot predict it exactly, but if this continues, we will see in the short term a biological-brain reductionist conceptualisation of all addictions. Some embrace this model almost like a religion and silence the critical voices, of which there are many, but they are not the ones with the power, the money, the means, or the public access. What we mentioned at the beginning of this article is very striking, that 94 important scientists and clinicians from different countries around the world would write a letter to the editor of *Nature* denouncing this model and the attempt to make it predominant. It would be strange for thousands of intelligent scientists, professionals and clinicians to be wrong about the cause of addiction. It therefore seems that sometimes we are facing more of



an ideology than a consistent model or paradigm (Vreckro, 2010). Although we are optimistic in the long term, because in the end reason always seems to prevail, this process can take years, which means an increase in the suffering of people with addictive disorders. In psychology it is clear that we cannot accept this model as formulated, because it is simple, biased, interested, reductionist, not based on the existing scientific data on addiction or the biopsychosocial model and also it does not help the interests of consumers or addicts. This model skirts around the main issues, leaving in second, third or fourth place, the role of the environment, psychological factors, etc., denying the reality of the scientific information accumulated over decades and decades of research.

It is curious that the dominant perspective in the field of addiction not so many years ago was psychological. But psychology is aimed at helping the human being, not at creating a technology that can make a profit or patents, or create products based on it. Nor was it believed that some of the people who assumed the biopsychosocial model, upon which the science of addiction has been based in recent decades, had the audacity to propose such a radical reductionism or mask such reductionism within a biased approach by indicating that there are always individual or social factors that frame this brain disease. But it has happened, with no consistent arguments being heard and even without anyone to argue from this reductionist model that the biopsychosocial model anachronism must be abandoned (Cabanis, Moga & Oquendo, 2015).

We believe the data should prevail over beliefs and interests, so we conclude that the psychological contribution to addictions has been central and will remain so in the future. Brain-centred biological reductionism is not justified nor is it useful or appropriate for people with addictive disorders or for preventing addiction. In addition, this model cannot explain the entire complex phenomenon of addiction, but we must take it into account and, at the same time, produce our own data, more forcefully and more publicly and using the media, and we must not be fooled by a very well organised marketing campaign in favour of this model, in which it seems that what they are presenting is real and the other explanations for this complex problem do not exist. This is a new task that psychologists have to undertake in an urgent, persistent and incisive way.

#### REFERENCES

- Ahmed, S., Lenoir, M. & Guillen, K. (2013). Neurobiology of addiction versus drug use driven by lack of choice. *Current Opinion in Neurobiology*, 23, 581-587.
- American Psychiatric Association [APA] (2014). DSM-5. Manual diagnóstico y estadístico de los trastornos mentales [Diagnostic and Statistical Manual of Mental Disorders]. Madrid: Editorial Médica Panamericana.
- Babor, T., Caulkins, J., Edwards, G., Fischer, B., Foxcroft,
  D., Huamphreys, K. et al. (2010). La política de drogas y el bien público [Drug policy and the public good].
  Washington, DC: Organización Panamericana de Salud.
- Becoña, E. (2002). Bases científicas de la prevención del consumo de drogas [The scientific basis for drug prevention]. Madrid: Delegación del Gobierno para el Plan Nacional sobre Drogas.
- Becoña, E. (2014). Trastornos relacionados con sustancias y trastornos adictivos [Substance-related disorders and addictive disorders]. Cuadernos de Medicina Psicosomática, 110, 58-61.
- Becoña, E. (2016). Trastornos adictivos [Addictive disorders]. Madrid: Síntesis.
- Berger, P. & Luckmann, T. (1968). La construcción social de la realidad [The social construction of reality]. Buenos Aires: Amorrortu Editores.
- Cabanis, D, K<sub>"</sub> Moga, D. E. & Oquendo, M. A. (2015). Rethinking the biopsychosocial formulation. *Lancet Psychiatry*, 2, 1-2.
- Compton, W. M., Dawson, D. A., Goldstein, R. B. & Grant, B. F. (2013). Crosswalk between DSM-IV dependence and DSM-5 substance use disorders for opioids, cannabis, cocaine and alcohol. *Drug and Alcohol Dependence*, *122*, 38-46.
- Conrad, P. & Barker, K. K. (2015). The social consequences of illness: Key insights and policy implications. *Journal of Health and Social Behavior, 51* (Supl.), 67-79.
- Cosgrove, L. & Krimsky, S. (2012). A comparison of DSM-IV and DSM-5 panel members' financial associations with industry: A pernicious problem persists. *Plos One*, *9*, e10011990.
- Courtwright, D. T. (2010). The NIDA brain disease paradigm: History, resistance and spinoffs. *BioSocieties*, *5*, 137-147.
- Edwards, G. (2002). Alcohol: The world's favorite drug. New York, NY: St. Martin's Press.



- Ersche, K., Williams, G., Robbins, T. & Bullmore, E. (2013). Meta-analysis of structural brain abnormalities associated with stimulant drug dependence and neuroimaging of addiction vulnerability and resilience. *Current Opinion in Neurobiology, 23,* 615-624.
- Frances, A. (2014). ¿Somos todos enfermos mentales? [Are we all mentally ill] Madrid: Ariel.
- Gartner, C. E., Barendregh, J. & Hall, W. D. (2009). Multiple genetic tests for susceptibility to smoking do not outperform simple family history. *Addiction*, 104, 118-126.
- Hall, W., Carter, A. & Forlini, C. (2015). The brain disease model of addictions: is it supported by the evidence and has it delivered on its promises? *Lancet Psychiatry*, *2*, 105-110.
- Hammer, R., Dingel, M., Ostergren, J., Partridge, B., McCormick, J. & Koening, B. A. (2013). Addiction: Current criticism of the brain disease paradigm. AJOB Neuroscience, 4, 27-32.
- Hasin, D. S., O'Brien, C. P. Auriacombe, M., Borges, G., Bucholz, K., Budney, A., ... Grant, B. F. (2013). DSM-5 criteria for substance use disorders: Recommendations and rationale. *American Journal of Psychiatry*, 170, 834-851
- Heim, D. (2014). Addiction: not just brain malfunction. Nature, 507, 40.
- Heyman, G. (2009). Addiction: A disorder of choice. Cambridge, MA: Cambridge University Press.
- Jellinek, E. M. (1960). *The disease concept of alcoholism*. New Haven, CT: Hillhouse Press.
- Kushner, H. I. (2010). Toward a culture biology of addiction. *BioSocieties, 5,* 8-24.
- Leshner, A. I. (1997). Addiction is a brain disease, and it matters. *Science, 278,* 45-47.
- Levy, N. (2013). Addiction is not a brain disease (and it matters). *Frontiers in Psychiatry, 4,* article 24.
- Lichter, P. R. (2008). Debunking myths in physicianindustry conflicts of interest. American Journal of Ophthalmology, 146, 159-171.
- Loe, M. (2014). The rise of viagra: How the little blue pill changed sex in America. New York, NY: New York University Press.

- Melchert, T. P. (2015). Biopsychosocial practice. A science-based framework for behavioral health care. Washinton, D. C.: American Psychological Association.
- NIDA (2008). Drugs, the brain and behavior. The science of addiction. Rockville, MD: National Institute on Drug Abuse, National Institutes of Health.
- O'Brien, C. P. & McLellan, A. T. (1996). Myths about the treatment of addiction. *Lancet*, 347, 237-240.
- Pedrero, E. (2015). Salud mental y adicción [Mental health and addiction]. Madrid: Atenea.
- Robins, L., Helzer, J., Hesselbrock, M. & Wish, E. (2010). Vietnam veterans three years after Vietnam: How our study changed our view of heroin. *American Journal of Addiction, 19,* 203-211.
- Satel, S. & Lilienfeld, S. 0. (2014). Addiction and the brain-disease fallacy. *Frontiers in Psychiatry*, 4, article 141.
- Slapak, S. & Grigoravicius, M. (2006). "Consumo de drogas": La construcción de un problema social [Drug use: The construction of a social problem]. Anuario de Investigaciones, 14, 239-249.
- Stea, J. N., Yakovenko, L. & Hodgins, D. C. (2015). Recovery from cannabis use disorders: Abstinence versus moderation and treatment-assisted recovery versus natural recovery. *Psychology of Addictive Behaviors, 29,* 522-531.
- Trujols, J. (2015). The brain disease model of addiction: Challenging of reinforcing stigma? *Lancet Psychiatry*, 2, 292.
- Vrecko, S. (2010). Birth of a brain disease: Science, the state and addiction neuropolitics. *History of the Human Sciences, 23,* 52-67.
- Volkow, N. D. & Morales, M. (2015). The brain on drugs: From reward to addiction. *Cell, 162,* 712-725.
- Whitaker, R. (2015). Anatomía de una epidemia. Medicamentos psiquiátricos y el asombroso aumento de las enfermedades mentales [Anatomy of an epidemic. Magic bullets, psychiatric drugs, and the astonishing rise of mental illness in America]. Madrid: Capitán Swing.