Psicothema

Psicothema 2018, Vol. 30, No. 3, 270-275
doi: 10.7334/psicothema2017.303

ISSN 0214 - 9915 CODEN PSOTEG

www.psicothema.com

Brain disease or biopsychosocial model in addiction? Remembering the Vietnam Veteran Study

Elisardo Becoña
Universidad de Santiago de Compostela

Abstract

Background: In recent years, we have repeatedly been told that addictions are a brain disease, leaving aside their classic biopsychosocial explanation. **Objective:** To describe both models and discusses the weakness and reductionism of the brain disease model following the consumption of heroin by North American soldiers in the Vietnam war in the 1970s. **Method:** A literature review of the Vietnam Veteran Study in relation to drug consumption. **Results:** The soldiers greatly increased their consumption of heroin in Vietnam, but almost all of them ceased using it upon returning home. The analysis of the environmental factors related to this self-healing is a critique of the brain disease model of addictions because it cannot explain this or other studies. **Conclusion:** The biopsychosocial model is still the best model to guide the field of addiction due to its utility, coherence, and efficacy in treatment.

Keywords: Addiction, brain disease, biopsychosocial, Vietnam Veteran Study, heroin.

Resumen

¿Modelo de enfermedad cerebral o biopsicosocial en adicciones? Recordando el Estudio de Veteranos de Vietnam. **Antecedentes:** en los últimos años se escucha cada vez más la afirmación de que las adicciones son una enfermedad cerebral, dejando a un lado la clásica explicación bio-psico-social en adicciones. **Objetivo:** describir ambos modelos y analizar la debilidad y reduccionismo del modelo de enfermedad cerebral siguiendo el Vietnan Veteram Study, sobre el consumo de heroína, en los soldados norteamericanos que participaron en la guerra del Vietnam. **Método:** revisión de la literatura del Vietnam Veteran Study en relación al consumo de drogas. **Resultados:** los soldados incrementaron de modo importante el consumo de heroína en Vietnam, pero casi todos dejaron de consumirla a su vuelta a casa. Analizando los factores ambientales relacionados con esta auto-cura se hace una crítica del modelo de enfermedad cerebral en adicciones ya que el mismo no puede explicar este ni otros estudios. **Conclusión:** el modelo biopsicosocial sigue siendo el mejor modelo para el campo de las adicciones debido a su utilidad, coherencia y eficacia en su tratamiento.

Palabras clave: adicción, enfermedad cerebral, biopsicosocial, Estudio de Veteranos del Vietnam, heroína.

Addictions as a brain disease

In recent years, we have seen psychiatrists and other neuroscientists' attempts to turn addictions into a "brain disease." This began timidly a few decades ago (Leshner, 1977; O'Brien & MacLellan, 1996), and has emerged with full force when this conception was taken on by the National Institute on Drug Abuse [NIDA] in its informative manual "*Drug, brain and behavior. The science of addiction*" (NIDA, 2007). Many authors expanded and justified it, especially Nora Volkow, Director of the NIDA since 2003, all of this coupled with generous funding of the studies in this vein. Also the American Psychiatric Association (2013) in the latest version of the DSM-5 consider substance consumption disorder along these lines, when indicating that "an important characteristic of substance use disorders is an underlying change in brain circuits that may persist beyond detoxification, particularly in individuals with severe disorders. The behavioral effect of these brain changes may be exhibited in the repeated relapses and intense drug craving when the individuals are exposed to drug-related stimuli. These persistent drug effects may benefit from long term approaches to treatment" (pp. 483). This conceptualization falls within a current notion of biological psychiatry consisting of reducing all of psychopathology to brain diseases or disorders (Insel & Cuthbert, 2015).

Volkow & Morales (2015) consider that the "advances in neuroscience identified addiction as a chronic brain disease with strong genetic, neurodevelopmental, and sociocultural components" (pp. 712). The key always lies in the fact that drugs activate the reinforcement circuits through the neurotransmitter dopamine in the accumbens nucleus, tegmental ventral area and other brain areas. And that once this process occurs, it is irreversible. A central claim is that the persistent use of a drug produces long-term and irreversible changes in the brain's structure and function (Courtwright, 2011; NIDA, 2007). Therefore, the individual cannot exercise self-control over his or her behavior. They also assign

Received: August 18, 2017 • Accepted: March 22, 2018
Corresponding author: Elisardo Becoña
Dpto. de Psicología Clínica y Psicobiología, U. de Tabaquismo y Trast. Adictivos
Universidad de Santiago de Compostela
15782 Santiago de Compostela (Spain)
e-mail; elisardo.becona@usc.es

great weight to genetics, to factors such as stress and psychiatric comorbidity. Many of their statements are based on animal research (Ahmed, 2012) with paradigms of self-administration of drugs in standardized and controlled conditions. What they propose is an exclusively biological theory of the addictive process. The other relevant components of addictions are missing. Interestingly, they suggest that, according to that model, certain pharmacological treatments would be ideal (McKay, Kranzler, Kampman, Ashare, & Schnoll, 2015; Volkow, Koob, & McLellan, 2016), but the hard clinical reality indicates that this is not so, and in many cases, the only treatment of choice that works is the psychological treatment (e.g., cannabis, cocaine, alcohol or tobacco) (Blonigen, Finney, Wilbourne, & Moos, 2015).

In recent years, also due to the many criticisms that this model has received, they propose more factors to explain the addictive process, or the recent statement that only a minority of people who consume drugs become addicted, and that such vulnerability is due to genetic, environmental, and developmental factors (Volkow et al., 2016).

There has been much criticism of this model, as can be imagined [e.g., de Leon, 2015; Hall, Carter, & Forlini, 2015; Hammer et al., 2013; Levy, 2013; Pedrero, 2015; Pérez, 2013).

The biopsychosocial model of addictions

The biopsychosocial model was proposed by Engel (1977), a specialist in internal medicine with psychotherapeutic training, considering that biological, psychological, and social factors play an important role in human functioning within the context of disease or the perception of disease. He proposed it as a holistic model, an alternative to the existing model in the biomedical field at that time. What Engel did was to transfer the center of interest from the disease to the sick individual. The final goal of the model is to perform biopsychosocially oriented clinical practice (Engel, 1980). In few years, in diverse areas of health, and specifically in drugs of abuse, this was soon accepted by professionals who were working on it, as there were professionals of all three areas -medical, psychological, and social.

The biopsychosocial model of addiction states that genetic/biological, psychological, and sociocultural factors contribute to substance consumption and should be taken into account for its prevention and treatment (Becoña, 2002; Skewes & González, 2013). The different current models of addiction considered these same factors to a greater or lesser extent (West & Brown, 2013) because we know about the mutual relation between genetic-biological factors, the environment, and individuals' thoughts and behaviors to maintain their health and in the process of becoming sick (Melchert, 2015).

To show the reductionism of the brain disease model of addiction and the current validity of the biopsychosocial model to explain the complex phenomenon of addictions, we shall examine a classic study on heroin, the Vietnam Veteran Study.

The Vietnam Veteran Study about heroin consumption. A classic study, almost forgotten nowadays

The best study carried out that clearly demonstrates the weight of the environment in drug consumption is the Vietnam Veteran Study, a classic study on how one can stop consuming heroin, without the need of treatment, when the environmental circumstances that maintain the consumption change.

In 1971, in the midst of the war, when each month about 1,000 soldiers went home, the alarms sounded, as between 10 and 15% of the returning soldiers were addicted to heroin. To find out about this situation, Jerome Jaffe, newly appointed Director of the Special Action Office for Drug Abuse Prevention of the White House, entrusted to perform a study to find out what was happening and how to face this situation. Most of the soldiers were 19 years old when arriving at Vietnam, and they returned to the United States when they were 20 or 21 years old, mostly to their parents' home (Robins, 1993).

At that time, soldiers' access to heroin in Vietnam was very easy because it was abundant, proceeding from the golden heroin triangle (the countries surrounding Vietnam), it was very pure (about 90-96%), about 15 to 30 times purer than that obtained in the USA (Stanton, 1976) and cheap, not more than 6 dollars a day for heroin (Jaffe, 2010). Moreover, most soldiers smoked it (Robins, Helzer, & Davis, 1975), they did not inject it, as occurred in United States.

Two samples were used for the study, a random one of soldiers returning from the Vietnam, and another one of returning soldiers with positive opiate urine tests. Thus, out of 13,760 soldiers who returned from 470 were randomly selected. And, out of 1,400 who tested positive in urine to opiates, 495 were randomly selected. Twenty-two soldiers who appeared randomly in both samples were eliminated. Of the general sample, 43% had used narcotics in Vietnam, 34% had used heroin, and 20% were addicted to narcotics. Between 8 and 12 months after returning home, only 10% had used narcotics, and the number of those addicted to heroin decreased to 1% (Jaffe, 2010; Robins, 1993; Robins et al., 1975). In a subsequent follow-up at 3 years, 2% was addicted to heroin (Robins, Helzer, Hesselbrock, & Wish, 2010). All this was confirmed with urine analyses at each time. We note that in Vietnam, they also consumed other drugs with a high level of prevalence such as marijuana, amphetamines, and barbiturates.

The main reason they gave for consuming heroin in Vietnam was because of its euphoric effects; the next reasons were to be able to bear army rules and to reduce homesickness, boredom, depression, and fear (Robins et al., 1975).

In contrast, alcohol consumption had the opposite pattern to heroin. Before going to Vietnam, 22% had alcohol problems and 4% met the criteria for alcoholism. In Vietnam, this dropped to 13 and 2%, respectively. But upon returning home, 8 to 12 months later, 30% had alcohol-related problems and 8% met the criteria for alcoholism (Goodwin, Davis, & Robins, 1975).

What this study showed is that heroin consumption and dependence upon returning home was similar to that reported before going to Vietnam, although there, consumption increased temporarily and importantly (Helzer, 2010). Having gone to Vietnam, the circumstances of the departure, and the context they found there explained the phenomenon. That is, variables like being away from home, in a new, strange, and hostile environment, in a war, where many of them did not know what they were doing there, thousands of miles from home, surrounded by death every day, the stress, anxiousness, fear, distress, helplessness, etc., along with high access to a substance that made it easier for them to support their daily lives. All this disappeared in most of them when they got back home. This change of the conditions, besides the attachment, affection, and comprehension they received from their parents, friends, girfriends, and the American society when they came home made almost all them go back to their pre-war status.

Is the brain model adequate for addictions?

If the model of addiction based on brain disease is true, then how does it explain the data of the Vietnam Veteran Study? In other words, a theory must fit the available empirical data and, in the case of drug use, especially with regard to its onset, abandonment, treatment, and maintenance or relapse. Does this happen in the study we just saw? No. Therefore, it can be used as one of the criticisms of the brain disease model (e.g., Hall et al., 2015).

Even the title of the article of Robins et al. (2010), one of the researchers who participated in the study, is very eloquent: "*how our study changed our view of heroin*". And, as they themselves said: "they were exposed to generous supplies of heroin for only 1 year and in an extraordinary situation –far from home and under fire" (pp. 211). Or as Helzer (2010) said: "The most obvious legacy of the Vietnam study is a change in our view of heroin and other opiate addiction. The study challenged many of the accepted beliefs about the intractability of opiate addiction. It demonstrated unequivocally that use of opiates did not necessarily and rapidly progress to regular use and addiction" (pp. 219-220).

As already stated in this study and in many others on drug dependence in humans, opiate addiction cannot be reduced to just a biological phenomenon. First of all, it is a behavior learned in a context that facilitates it. Hence, self-healing or spontaneous recovery from different drugs is possible (Heyman, 2013), or as Robins et al. (1975) said "it does seem clear that the opiates are not so addictive that use is necessarily followed by addiction nor that once addicted, and individual is necessarily addicted permanently. At least in certain circunstances, individuals can use narcotics regularly and even become addicted to them but yet be able to avoid use in other social circunstances" (pp. 961).

When beliefs guide behavior or the way of thinking, it is difficult to see reality. This already happened with this study, when Helzer (2010) indicated that "as Jerome Jaffe points out, the results of the Vietnam study were so contrary to accepted wisdow that many found them unacceptable" (pp. 221), in spite of the fact that the study has an excellent design, a representative sample, and it was performed with strict methodological control, including urine analyses in the entire sample and at all the times tested.

Reviewing this study, we clearly see how the brain model of addiction has forgotten the relevance of the environment and the human being's capacity to respond to incentives (Satel & Lilienfeld, 2014). This has marked the history of humanity. Therefore, we cannot ignore the environmental, psychological, or biological factors to explain addictions (Becoña, 2016b). For example, about availability, Robins (1993) said that: "Their remarkable rate of use was a response to market conditions—both the high availability of opiates and the lack of alternative recreational substances, to the absence of disapproving friends and relatives, and to the fact that serving in Vietnam was not seen as part of their real-life career" (pp. 1052).

Also, the genetics of addictions partially fails in this study because it explains only a part of the problem (Maze and Nestler, 2011), as becomes clear when observing that some people can begin to consume drugs (heroin) and later on stop consuming it by themselves. In this case, environment is more relevant to explain the behavior, not only genetics or biology (Tsuang, Bar, Harley, & Lyons, 2001).

And such low prevalences upon returning were not due to treatment. As said Helzer (2010), "The low rates of opiate involvement after return from Vietnam do not reflect treatment results. Less than 1% of the general sample of veterans had any interest in receiving treatment and only 5.5% of those who had failed the exit urine screen had an interest" (pp. 219).

This study has shown how many people, addicted in Vietnam, have ceased to consume substances by themselves and have gone on to live a normal life. This has also been found in other studies. It is called spontaneous remission (Heyman, 2013; Satel & Lilienfeld, 2014). The error of the brain model of addiction is that it extrapolates what probably happens with a reduced percentage of addicts to the rest of the addicts, or it is only based on animal studies in which environment and cognitions do not have the same relevance as in humans (Ahmed, 2012).

Clearly, addiction is not a neurological disease, and addicts do not have a brain injury, and no place has been located for such an injury. The onset of drug consumption is not biological, but social. Stress contributes significantly to becoming addicted (Volkow & Morales, 2015), as shown in the study of Vietnam. Clearly, biology is not able to explain all of human psychological functioning (Schwartz, Lilienfeld, Meca, & Sauvigé, 2016). However, let us not forget that psychotherapeutic interventions also change the brain's functioning and in some cases, also the brain's structure (Matto et al, 2014). The same thing can happen with the environment. Hence, spontaneous remission of addictions does occur (Heyman, 2013; Lopez-Quintero et al., 2011), as the study of Vietnam has shown.

It is nowadays well established that not everyone who consumes drugs will become addicted. And, in many cases, consumption may be a normal part of people's lives and they consume drugs because the drugs' effects are useful for their personal goals. This was probably what was happening in Vietnam. We cannot ignore the sociocultural explanations of drug use. As Chien-Chang (2011) says "our brains are not har-wired and could evolve and adapt with the requirement of sociocultural practices" (pp. 327). In the case of alcohol, we know that social influence leads to changes in its ingestion (Prestwich et al., 2016). Treatments with components of social influence reduce alcohol consumption. The same has been found in the case of tobacco (Boddanovica, McNeil, Murray, & Britton, 2011). We believe that the same thing happened in the study of Vietnam, where the availability of heroin, the stress and relaxing properties of the drug contributed to such a high consumption over there, and not upon coming home. For example, the introduction of urine controls in Vietnam reduced positive testing from 8-9% to 4-5% in one month (Jaffe, 2010).

Lenoir et al. (2013), in rats, has shown that heroin is more addictive than cocaine (51% compared to 15%, respectively, in a paradigm of self-administration of drugs, where rats preferred heroin or cocaine to sucrose). But this occurs in rats, not in humans. As says Ahmed (2012)), in his review of the models of self-administration of drugs in rats, "it appear that resilience to cocaine addiction is the norm in rats" (pp. 107). This is due to the fact that few rats are vulnerable to addiction. The same thing happens in humans. And it occurs when the rat has more possibilities of choice than cocaine. When it can choose between cocaine and performing another behavior, most choose another behavior, which means that it has not lost control over self-administration of cocaine. Therefore, this is similar in humans. It would also explain why, of those who consume drugs, only a few become addicts because the great majority has control over their behavior.

Vulnerability to dependence has been studied from two basic paradigms: the pharmacological or "drug-focused" approach of the model of exposure, where dependence is in the cerebral substrate; and the individual-focused approach, where the center of interest is the individual, where addiction is a behavioral disorder that occurs in a vulnerable phenotype (Swenson & Le Moal, 2011). Therefore, part of this explanatory capacity of the brain model of addiction in studies like the one mentioned is that there is currently a lot of technology to study the influence of drugs on the brain but little conceptualization of why this occurs. Thus, we discover the simplicity of the model and its lack of explanatory elements applied to the social reality of humans who consume drugs. As says Le Moal & Swendsen (2015) "brain science are still in a state of great ignorance, trivial for such a young enterprise" (pp. 600).

In spite of the insistence of the supporters of the model of addiction as a brain disease, addiction does not meet the specified criteria for a disease (Holden, 2012). For instance, addiction is self-acquired and is not transmissible, infectious, autoimmune, hereditary, degenerative, or traumatic. The treatment consists of little more than ceasing to perform the behavior. In addition, it turns out that most effective treatments in addictions are psychological, not pharmacological (Becoña, 2016b; Blonigen et al., 2015; Melchert, 2015).

Conclusion: brain disease model or biopsychosocial model?

In a recent article Volkow et al. (2016) wondered about "nonetheless, despite the scientific evidence and the resulting advances in treatment and changes in policy, the concept of addiction as a disease of the brain is still being questioned" (pp. 364). We believe that currently, it is not sufficiently questioned. Due to its reductionism, the brain model of addictions is not acceptable either from the psychological or the social perspective or from the biomedical field and, although we do not deny the role of biology, we do deny brain model's exclusivity and its simplistic attempt to understand the complex phenomenon of addictions all by itself.

Unfortunately, there are pressures and interests to consolidate this brain-centered model. For example, recently 94 relevant scientists and clinicians from different countries of the world wrote a letter to the editor of *Nature* denouncing this model and the attempt to make it predominant (Heim, 2014). It would be very strange for thousands and thousands of wise scientists, professionals, and clinicians to be mistaken about the cause of addiction. Therefore, it sometimes seems that we are facing an ideology and not a model or consistent paradigm (Vrecko, 2010).

The model of brain disease in addiction, as currently formulated, is simple, biased, profit-seeking, reductionist, not based on the existing scientific data on addiction or on the biopsychosocial model and, moreover, it does not serve the interests of consumers or addicts. This model addresses the central themes of addiction, 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 (Becoña, 2016a). This model views the mind and the body as separate and as not affecting each other. Really, the available data fit the biopsychosocial model and not a model of biological reductionism.

Biological brain-focused reductionism is not justified or useful or suitable for people with addictive disorders or for the prevention of addiction (Hall & Weier, 2017). Moreover, this model cannot explain the entire complex phenomenon of addictions (Edwards, 2010), but we must take it into account, and, at the same time, provide data of the different aspects that do explain addictions, with more forcefulness, publicity and media resonance, and not be fooled by a very well organized marketing in favor of this model that appears to present the reality, and the rest of the explanations of this complex problem simply do not exist.

We consider the attempt of the social construction of addictions as a brain disease and the interests underlying this effort to be very serious (Frances, 2014). Thus, it is noteworthy that in recent years some people have proposed that the biopsychosocial model should be abandoned because it is anachronistic (Cabanis, Moga, & Oquendo, 2015) or due to its lack of utility (Ghaemi, 2011). Proposing to ignore all the psychological and social factors that we know very well influence the acquisition, maintenance and abandonment of drug use is extreme reductionism (Skewes and Gonzalez, 2013). We believe that the following words of Hall et al. (2015) reflect the opposite viewpoint from the model of brain disease, and it is the one that most professionals of addictions assume: "addiction is a complex biological, psychological, and social disorder that needs to be addressed by various clinical and public health approaches " (pp. 109).

Intermediate proposals have been attempted, like that of Pickard, Ahmed, & Foddy (2015), who believe that it would be better to consider to addiction as a choice. The extremes of the perspective of normalization and brain disease would thus be avoided. This would provide the same attention and weight to the historical, contextual, and biological factors that are significant for addiction and that are the ones that constitute the classic biopsychosocial model. Thus, we would have a global conceptual framework in the field of addictions.

As Griffith Edwards (2010) said, with regard to the ideas about alcohol that have existed for the past 200 years, one of the inferences that could be made with confidence was that of "alcohol problems are multi-functional in origin, with history warning against any tendency to see the problem with drink as resident at the level of brain science alone. Psychological, social and economic aspects are also inherent" (pp. 803). The same is applicable to the Vietnam Veteran Study and to the brain model of addictions.

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