Theories of Addiction

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The term "addiction" derives from the Latin "*addicere*", meaning 'bound to' or 'enslaved by'; a term and meaning that have become synonymous with impaired control over substance use, though non-substance-related disorders are increasingly been included as well. Today, the definition of addiction is as varied as the personal or professional interests or biases of its authors. English and medical dictionaries define addiction broadly as a "persistent compulsive use of a substance known by the user to be harmful," [1] a "psychological or physiological dependence on a drug," [2] or "the loss of control over drug use, or the compulsive seeking and taking of drug regardless of the consequences." [3] In these and in most common definitions, the recurring theme is still "drug" or "substance", reflecting a prevailing mindset that explains the focus of current research being centered on drug-brain interactions and viewing addiction as a brain disease. In a broader sense, addiction has been defined as, "a recurring compulsion… often reserved for drugs but… sometimes applied to other compulsions, such as problem gambling and compulsive overeating." [4,5] However, using words such as "problem" to define addiction adds confusion though professional organizations' definitions might not be clearer. For instance, the American Psychiatric Association's DSM-IV-TR* nomenclature includes, not the specific term "addiction", but the categories of "Substance Use Disorders", "Impulse Control Disorders", and Obsessive Compulsive Disorders. These disorders share some features including impulsivity and impaired control over use, and it has been suggested that certain Impulse Control Disorders such as compulsive gambling, compulsive shopping, compulsive computer use, and compulsive sexual behaviors are "behavioral addictions" or "addictions without the drug," [6] implying that drug addiction is not the only "real" addiction and that all are behavioral. Likewise, the WHO's ** International Classification of Diseases and Related Health Problems (ICD-10) lists Impulse Control Disorders under "Habit and Impulse Disorders" and includes drug abuse under "Dependence syndrome". [7] It defined the latter as "A cluster of behavioral, cognitive, and physiological phenomena that develop after repeated substance use and that typically include a strong desire to take the drug, difficulties in controlling its use, persisting in its use despite harmful consequences, a higher priority given to drug use than to other activities and obligations, increased tolerance, and sometimes a physical withdrawal state." [8] This bewildering array of classifications and definitions of compulsive, impaired use control, reward-seeking behaviors that interfere with major areas of life functioning compound their understanding and management. Theories on the causes of addiction have been based on moral, biological, sociological, psychological, and social learning models, among many others. Consequently, addiction is said to be the domain of the police officer, the neurobiologist, the psychiatrist, the social worker, the priest, or the policy maker depending of the proponents' point of view. Each stance can affect the income and fate of professionals and organizations engaged in addiction prevention, control, or treatment, and the type of support offered to or punishment imposed on addicts. While it is not my purpose to reconcile these viewpoints through a "grand unifying theory", I subscribe to the view that they can be reduced to two opposing camps: the disease vs. the behavioral model advocates. In addition to identifying the two core themes underlying all theories and definitions of addiction, these opposite views carry profound clinical, policy-making, and legal implications. Advocates of the disease model view addicts as "victims" of an alleged defective neurocircuitry implicated in drug abuse hence promote the development and use of drugs as the basis for its management. On the other hand, proponents of the behavioral model consider addiction a choice

* Diagnostic and Statistical Manual of Mental Disorders, 4th edition
** World Health Organization

and promote behavior modification methods as the most suitable means to treat substance and non- substance abuse behaviors, at best, or incarceration, at worst. These two diverging views of addiction have divided the scientific community and the public at large to the point that each has become a dogma and as such, a non-negotiable polarizing issue to its proponents. In the next few pages, I will examine the pros and cons of each model in an objective and rational fashion.

1. The disease (victim) model

Although references to drunkenness as a disease can be found in ancient Egyptian and Greek writings, Scottish physician Thomas Trotter (1760-1832) is credited as the first to characterize excessive drinking as a disease in modern times. In America, drunkenness continued to be viewed as a condition that bridged morality and medicine. For instance, in his "Inquiry into the effects of ardent spirits on the human mind and body" [9], American physician Benjamin Rush (1745-1813), considered the "Father of American Psychiatry", introduced the idea that the loss of control characteristic of alcoholism was a disease "induced by vice" but caused by the "inherent properties" of alcohol. Likewise, in his "Six Sermons on the Nature, Occasions, Signs, Evils, and Remedy of Intemperance" [10], reverend Lyman Beecher referred to "intemperance" as a "sin", an "evil habit", and "a disease as well as a crime". However, of greater interest is Dr. William Sweetser's 1829 insightful argument that "intemperance" is "a disease produced and maintained by voluntary acts, which is a very different thing from a disease with which providence inflicts us", though one that causes a "morbid alteration" of the body. This view would be resurrected in support of the disease theory of addiction over a century later, as discussed below. Likewise, doctor Samuel Woodward described "intemperance" as a "physical disease which preys upon health and spirits ... making him a willing slave to his appetite." [11] He also believed that heredity played a role in drunkenness. Swedish Physician Magnus Huss (1807-1890), who considered the condition as a chronic, relapsing disease first coined the term alcoholism, in 1849. Hence, the disease concept of alcoholism was already taking shape in the middle of the 19th century, including the features of impaired control, craving, tolerance, and a presumed predisposition to drinking. Its biological, psychological, and social consequences also were being recognized. However, New York-born E. Morton Jellinek (1890-1963) was to propel to center stage the "disease concept of alcoholism," and by extension drug of abuse he classified into 5 stages - from 'alpha' to 'epsilon' phases. Jellinek's father, a Hungarian, had taken his family back to Budapest when young Jellinek was a pre-school boy. From his sister's account, we know that he first served as a captain in the Hungarian Red Cross in WWI before going bankrupt as a currency speculator. He reappeared in 1920 working for a steamship line in Sierra Leone under the name of Nikita Hartmann, then as a banana researcher in Honduras for the United Fruit Company before becoming a biostatistician at Worcester State Hospital in Massachusetts, in 1931. In 1939, age 50 he was hired to manage an Alcohol study called the Carnegie Project. According to his sister, he "knew very little about alcoholism but he was interested so he got some books on the subject and spent a weekend in bed studying." [12] Jellinek devoted the rest of his life to the field and earned the respect and admiration of his peers unaware that he had fabricated his degrees. Indeed, his claim to a Doctor of Science degree from the short-lived University of Tegucigalpa in Honduras was never substantiated and the "Honorary" prefix of a Doctor of Science degree he claimed the University of Leipzig bestowed upon him soon disappeared from his publications. Yet, he became an Associate Professor of Applied Physiology at Yale University (1941 - 1952) and subsequently a consultant on alcoholism to the WHO in Geneva. In 1958, he joined the Psychiatry departments of the Universities of Toronto and Alberta and, in 1962, moved to Stanford University where he died a year later. Because alcohol was the most obvious drug of abuse in Jellinek's time and often ran in families, genetics was suggested as a possible cause, though according to this criterion child abuse and religion that also run in families would qualify as addictions. In attempts to separate environmental from genetic factors, cohort studies of

adopted children from alcoholic parents were compared to adopted children from non-alcoholic families. Some studies suggested up to a 3- to 4-fold increased incidence of alcoholism among the former. [13,14] However, such studies failed to account for non-genetic factors (e.g. ethnicity) that impact on incidence rates and could not explain how is alcoholism inherited. Subsequently, the focus shifted to the search for predisposing factors that confer the bearer an inherited vulnerability to alcoholism rather than a predetermined alcoholic destiny. Some of the metabolic defects claimed to predispose to alcoholism included: an inability of alcoholics to discriminate blood alcohol levels, which presumably leads to lesser effects from alcohol and to increased drinking, an altered alcohol metabolism associated with higher levels of acetaldehyde and related symptoms and with decreased drinking; [15] or an aberrant brain circuitry that reinforces drinking. [16] For instance, a mutant ALDH-2 gene protein that metabolizes acetaldehyde, a product of alcohol elimination, causes its tissue accumulation more slowly than normal inducing flushing, dizziness, and nausea, which are thought to reduce the risk of alcoholism in carriers. Indeed, approximately 50 percent of Asians born with this mutant gene drink less than their normal counterparts even after migrating to the US, suggesting that genetics might be the stronger predisposing influence in that population. However, children of immigrant Asian Americans drink more that their parents and the generational difference in drinking level is greater than that observed between carriers of the mutant and of the normal ALDH-2 gene. These findings conclusively prove that the drinking behavior of first generation Asian Americans is impacted more by environmental factors than by heredity. Likewise, sensitivity to blood alcohol levels, variations in alcohol metabolism, and abnormal brain circuitry in response to alcohol intake do not predict or correlate with alcoholism, [17] nor do they differentiate the occasional or social alcohol user from an alcohol-dependent individual. Another interesting historical episode on the presumed inheritance of deviant behavior is associated with Cesare Lombroso's now discredited theories described in his influential book "l'Uomo delinquente" (the delinquent man). [18] Born in Verona in 1835, Lombroso studied medicine in Pavia, Padua, Vienna, and Genoa and taught legal medicine and public hygiene at the University of Turin. His interest in psychology and psychiatry along with his studies on brain anatomy and physiology ultimately led to his anthropometric* analysis of criminals and the mentally disturbed. Years of postmortem examinations and anthropometric studies of criminals, the insane, and normal individuals, convinced Lombroso that, contrary to the prevailing view that crime was a characteristic of human nature, he proposed the existence of the "reo nato" (born criminal) that was anatomically identifiable by certain physical stigmata**. They included a sloping forehead, handle-shaped ears, high cheekbones, hawk-like noses or fleshy lips, prognathism,*** and excessively long arms. These physical features, he thought, indicated a throwback to a primitive form of humans whose behavior was inevitably contrary to the rules and expectations of modern civilized society. Not unlike some of today's researchers who seem convinced of the genetic basis of addiction, he reluctantly admitted the influence of the environment in the etiology of crime but continued to believe in the predominant role of "predisposing" organic and genetic factors. While Lombroso's notions gained many adepts throughout Europe, they were eventually discredited by Charles Goring in his book The English Convict, published in 1913, where he documented that anatomical differences found in criminals were minimal at best. Since the decade of the 1960s, when illicit drugs began their ascent, addiction has become synonymous with drug abuse and its study expanded to include neuroscientists, molecular biologists, pharmacologists, psychologists, geneticists, and even circadian rhythm* theorists. Inevitably, each discipline brings a special one-dimensional approach and conclusions to the study of addiction that confounds the understanding of this highly complex issue involving behaviors that transcend drug abuse. For example, it has been

* The study of the measurement and proportions of the human body
** A mark or characteristic indicative of a disease or abnormality
*** A lower jaw that projects forward

suggested that, "all drugs of abuse converge on a common circuitry in the brain limbic system… [especially] …the ventral tegmental area (TVA) of the midbrain… [and] …their target in the nucleus accumbens (NAc). The VTANAc pathway is one the most important substrates for the acute rewarding effects of all drugs of abuse… [though] …additional brain areas that interact with the VTA and NAc are also essential for acute drug reward and chronic changes associated with addiction." [19] From study results such as this, it is concluded that addiction is "a chronic, relapsing brain disease… because drugs change the brain... and can lead to the harmful behaviors seen in people who abuse drugs" [4]. However, even if drugs of abuse change brain chemistry reinforcing the reward circuitry that perpetuates the cycle of addiction, the question remains: what leads someone to use drugs in the first place. That is, what is first; behavior affecting the brain or brain changes that affect behavior? Likewise, if drugs change the brain, why is the vast majority of drug use transient and sporadic and why can most drug abusers who choose to quit do so, more often than not, without third party assistance? In fact, no neurocircuits or neurotransmitters can compel anyone to abuse drugs for even the most drug-centered and self-destructive addicts retain a certain degree of control on how much drug they take at any given time, keeping some supply for the next high. Moreover, neurocircuitry and chemical reward theories of addiction are unable to account for the wide spectrum of addiction, ranging from not only licit or illicit drugs, prescription medications, and chemical products, but extending to non-substance based activities such as gambling and others that are normal, ordinary, and non-addictive for most people such as drinking coffee, eating, and having sex. Hence, "addiction" is linked to the individual, not to any intrinsic addictive property of the substance or activity abused, or their effect on the brain. Furthermore, the suggestion that the VTA-NAc pathway also mediates "the acute positive emotional effects of natural rewards such as food, sex, and social interactions" [19] does not validate viewing addicts as victims of their brain chemistry. The above arguments suggest that drug-receptor interactions theorized as the underlying mechanism for a self-reinforcing "reward" pathway blamed for addiction are at best simplistic, for they reduce behavior to simple neurobiochemical processes excluding free choice as a factor, or at worst seek to support preconceived notions intended to absolve drug abusers of responsibility. In fact, the reward pathway is an evolutionary brain circuit that reinforces behavior that makes us feel good and ensures our survival. Moreover, no brain pathways or neurotransmitters need be discovered to confirm what daily living teaches us: experiences are sought after if enjoyable and avoided if unpleasant. Seeking to recreate pleasurable experiences is a normal and common reaction of all living creatures from the lowest of worms to rodents to humans and is therefore incidental to the deviant and often self-destructing abuse behavior we call addiction. The issue is not whether seeking pleasurable experiences is reinforced through a reward pathway, which it is, but what factors underlie the type of impaired control behavior that initially leads to drug seeking and later to compulsive use. In short, since chemical rewards have no power to dictate human behavior, the key question is what leads some casual users to become addicted whereas the vast majority doesn't; a pivotal step that neurobiologists cannot explain. Simply put, "our brains do not make us do it". Likewise, theorizing that opiates and other drugs of abuse are inherently addictive or that repeated exposure will inevitably lead to addiction, while ingrained in popular folklore, have no scientific basis. Indeed, some of the "most addictive" drugs of abuse induce tolerance and withdrawal symptoms (e.g. heroin), some induce one but not the other (e.g. cocaine, fentanyl), and others induce neither (e.g. Levo-Alpha Acetyl Methadol), suggesting that these properties are not central to addiction as it is claimed. Moreover, the notion that certain drugs are inherently addictive ignores the medical evidence that taking narcotics round-the-clock for months to years for pain relief does not induce addiction [20-22], and that most casual users of licit and illicit drugs do not become addicted [23] and those who do can give them up if they choose to. Finally, few would consider ordinary foodstuff as inherently addictive. Yet, it is undeniable that many

* Recurrent biological patterns claimed to influence drug efficacy, behavior, etc.

individuals with "compulsive eating disorder" are in fact addicted. Yet, a most forceful proponent of the disease theory of addiction is the National Institute on Drug Abuse (NIDA), the Federal agency whose mission is "to lead the Nation in bringing the power of science to bear on drug abuse and addiction", which it does by steering research on addiction through selective allocation of funds. It defines addiction "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" [5]. In an August 2007 online educational presentation, it likens drug addiction to several common somatic* diseases on the basis that they all exhibit genetic and behavioral components and are not self-inflicted. It claims, "Addiction, like heart disease, cancers, and type II diabetes, is a real and complex disease… no one chooses to be a drug addict or to develop heart disease… sometimes people do choose behaviors that have undesirable effects… addictive behaviors have clearly implicated both environmental and genetic influences" [4]. These analogies were carefully chosen to convey a sense that if heart disease, cancers, and type II diabetes are acknowledged diseases despite being the unintended outcome of poor lifestyle choices (e.g. overeating, a sedentary lifestyle, and cigarette smoking -in the case of lung cancer-, respectively), addiction must also be classified as a disease. The parallel appears strengthened by invoking a genetic link based on the proposition that APOE** and the μ-opioid receptor contribute to heart disease and heroin addiction, respectively. However, the comparison is inappropriate because there is no such a thing as cancer or heart disease but a diverse group of over 200 cancers and a plethora of heart diseases caused or contributed to by factors as diverse as heredity and behavior. Examples of the former include Hereditary, non-polyposis colorectal cancer***, and Hypertrophic cardiomyopathy****, respectively. Examples of behavior-related cancers and heart diseases include tobacco-induced lung cancer and atherosclerosis* linked to obesity, respectively. Moreover, the comparison would remain disingenuous even if it were limited to behavior-related heart diseases and cancers, as it steers the reader's attention to the outcome of addictive behavior in the case of the somatic diseases cited, to the addictive behavior itself in the case of drug abuse. The appropriate comparison should be between heavy smoking and heavy drug use; both being addictions albeit to products arbitrarily placed on opposite sides of the law, or between lung cancer and HIV infection; both being disease outcomes often associated with the addictive behavior of smoking and IV drug use, respectively. Similarly, overeating to the point of developing long-term health consequences, including morbid obesity, atherosclerosis, type II diabetes, or colon cancer is also a form of addiction albeit one not yet sanctioned by the politically correct medical establishment or acknowledged by the public. To assert that addiction is a disease because drug users do not intend to become addicts is as ludicrous as claiming that heavy smoking is a disease because smokers don't plan to develop lung cancer. Smoking, overeating, and drug abuse are all addictions albeit with a different focus; the major difference being that the latter has been made into an illicit behavior. Likewise, to claim that the very existence of μ-opioid brain receptors proves or supports the genetic basis of heroin addiction is tantamount to suggesting that the human brain evolved such a receptor in anticipation of its usefulness as a mediator of heroin addiction centuries later. In fact, the term opioid receptor derives from neurobiological studies on narcotics [24] and is unrelated to the primary function of a receptor humans share with mice, rats, bullfrogs, chicken, cattle, and several fish species suggesting an evolutionary function that certainly transcends addiction.[25] Finally, it has been reported that "genetic variants of the μ-opioid receptor OPRM1 play a role in pain perception and in the susceptibility to substance abuse."[26]

* Related to the body
** Apolipoprotein-E, an apoprotein involved in heart disease
*** A hereditary type of cancer that affects mainly the colon and rectum
**** A familial thickening of the heart muscle

However, susceptibility does not mean inevitability and subscribing to this claim requires believing that, out of tens of millions of pain suffering patients taking opioids for pain relief, only 0.03% of them are endowed this variant receptor and become addicted, an unlikely proposition indeed. [20-22] Sorting out the specific and individual effect of a multitude of factors impacting addictive, compulsive, or obsessive behavior, and indeed any human behavior, is a highly complex, uncertain, and daunting process. Moreover, limitations in the tools available for study, flaws in the design, methodology, analysis, or conclusions of many studies, and the mind-set and pre-conceptions of many addiction researchers are further impediments in the search for reliable answers. For instance, the authors of a recent study of the possible genetic bases of nicotine dependence and abstinence [27] concluded, "the ability to abstain from nicotine has polygenic genetic components that overlap, in part, with those that contribute to vulnerability to nicotine dependence." To put it simply, contrary to the authors' assertion, that means that the set of genes that predispose you to smoke also predispose you not to smoke! Hence, this study supports the view that to smoke or not to smoke is the result, not of genes, but of a conscientious decision - a choice - made by the smoker, albeit one that is influenced by a multitude of psychological, familial, social, environmental, and genetic factors, as are all human decisions. Similarly, a study report on the role of genetics in human sexuality claimed having found the "gay gene." [28] Given its social, political, legal, and eugenics* implications, the report and its lead gay author became highly controversial. The report identified a genetic marker in region 28 of the long or "q arm of the X chromosome" **, shared by 33 (or 83%) of the 40 pairs of homosexual, non-twins brothers in which both members of each pair were gay and concluded that male homosexuality is X-linked. The Xq 28 marker, not itself a gene, is now known as "GAY-1". The report was initially hailed as the scientific foundation that supported prior claims of inherited sexual orientation based mainly on a prior study showing size differences of midbrains of homosexual young men and on several studies showing concordance*** in homosexual rates among monozygotic**** twins raised apart (100% concordance indicates a 100% inheritance). However, none of these studies are conclusive. First, many of the studied homosexuals with smaller midbrains died of AIDS*****, a condition that affects the brain. Second, concordance rates in identical male twin studies ranged between 0% and 100% suggesting flaws in subject selection or in the design, implementation, or interpretation of such studies. Finally, a linkage between homosexuality and the Xq28 region was not confirmed in a careful study of 52 gay male sibling pairs. [29] In contrast to claims that link certain deviant behaviors to hypothetical "genetic causes", there are approximately 4,000 diseases directly linked to chromosomal changes or gene mutations***** and can be inherited or acquired. Examples of the former include Ataxia Telangiectasia and Bloom syndrome. Acquired genetic mutations are caused by environmental "mutagens"****** and take the form of microscopic structural chromosomal changes such as multi-copy (e.g. Downs syndrome), translocations (e.g. Burkitt's lymphoma), partial deletions (e.g. acute myelogenous leukemia), or point mutations (e.g. sickle cell disease). [30] Hence, it is evident that genetic mutations can and do cause disease and there are genetic components in everything we are and in everything we do. However, evidence to date shows that behavior is modulated by a myriad of non-genetic factors (e.g. educational, religious, social, familial, psychological, and environmental) superimposed to a non-specific and non-determining genetic makeup. Indeed, while genes govern the biological processes of all living organisms in a direct, predictable, and deterministic manner, they have a more circuitous and subtle effect on human behavior that influences rather than eliminates free

* The science of improving genetic characteristics
** The female gene
*** Sameness with regard to a particular trait or characteristic
**** Derived from a single ovum
**** Autoimmune Deficiency Syndrome
***** Alterations in DNA or gene sequences
****** Agents or substances that cause genetic changes

will through difficult-to-assess gene-brain link. Simply put, "our genes do not make us do it."

2. The behavioral (choice) model.

While drug addiction is not a disease and is not caused by genetic abnormalities or a compelling abnormal brain reward circuitry, it can be viewed as the outcome of a "process" that begins with an encounter with drugs followed by casual drug-use that can progress to drug abuse in certain individuals, often to the detriment of self. [31] However, this process, which is similar for any substance of abuse or activity of abuse, is not a continuum for something intervenes to lead few casual users to become addicted whereas most do not. Perhaps the best-known example is alcohol, the most indulged drug that a large segment of the population (48% worldwide) consumes casually, socially, or daily. While most consumption is moderate and without adverse effects, a minority of users become addicted (9.7 million Americans were alcoholics in 2001-2002 [32]) and suffer the medical, psychological, economic, and social consequences of their excessive drinking. Likewise, as discussed in section 2 of the previous chapter, the natural history of drug abuse suggests that following peaks of problem behavior in adolescence and young adulthood, unassisted recovery is the norm for most casual and not-so-casual users. Indeed, while the "30-day" prevalence rate* for any illicit drug use by the 18 to 26 year-old cohort exceeded 1 in 5 in 2005, it dropped to less than 1 in 10 by age 45, despite the fact that approximately 80% of them admitted lifetime experience with illicit drugs [33] (chapter 2, figure 1). Additionally, as discussed in detail in the next chapter, the prevalence rate of licit and illicit drug use among US servicemen during the Vietnam War was rampant (alcohol, 90%; marijuana, 80%; opium, 38%; heroin 34%; amphetamines and barbiturates, 25% each). However, a strict DoD** ban on drugs, enforced by compelled urine testing and the threat of more disciplinary action for recidivists including extending their Vietnam tour and a negative urine test required to be discharged from service were the main incentives, rather than treatment, for most users to abandon drugs. A three-year follow up study showed a relapse rate of only 12%, that in most cases was short-lived. [34] Such extraordinarily high remission rate and continued abstinence contradict the still widely held belief that addiction is essentially irreversible, especially without treatment. These two studies, conducted 25 years apart, and many in between and since, demonstrate that most users "mature" out of their drug dependence or find sufficiently compelling motivations, often associated with love or religion, to override or compensate their cravings for drugs, and do so without professional or other assistance.[35] Users who become addicted and remain addicted constitute a very small subpopulation within the user population. Most, if not all, are trapped in personal, familial, and psychosocial problems that contribute to placing drug reinforcers*** in control of a significant portion of their behavior, which some neurobiologists call the "hijacking" of the reinforcement pathway by drugs.[36] The reinforcing stimulus can be euphoria from drugs, a feeling of satiety from food, an expectation of financial gain from gambling, or an adrenaline rush from high-risk activities. Although such stimuli might not become preeminent while the activities in question remain moderate rather than all consuming, they can progressively escalate to eventually dominate behavior when competing activities within the available repertoire provide less potent reinforcing stimuli, especially in individuals with impaired self-control or with non-supportive environments. Alternatively, when exposed to reinforcers incompatible with drug abuse (e.g. marriage, drug-free employment, etc.), addicts are able to substitute drugs for the new activity, achieving a more satisfying and often life-changing type of behavior reinforcement. As a corollary, it can be envisioned that the segment of the population that never tries drugs is made of individuals for who drugs of addiction or activities of addiction do not fit in their value system; a value system based on discipline and self-control that provides one or multiple reinforcers of non-

* Rate prevailing within the timeframe indicated
** Department of Defense
*** Stimuli that increase the strength of behavioral responses

deviant behavior. Hence, the brain reward circuitry proposed by some neurobiologists as the cause of addiction is only a conduit common to most, if not all, reinforcing stimuli whether resulting from normal or deviant behavior. What determines whether casual or moderate use will become compulsive and eventually all consuming depends on the individual's access to, handling of, or adherence to alternative reinforcers and on his/her level of discipline and self-control. Motivation is pivotal to the type of reinforcement achieved and whether addiction will ensue. Consequently, relief of chronic pain accompanied by a greater sense of wellbeing and a higher level of functioning are both reinforcers of "opioids-for-pain-relief" seekers, whereas interference with major areas of functioning is the hallmark of compulsive seeking "opioids-for-pleasure". As a result, pain patients experience no euphoria from taking opioids, have no reasons to continue their use once pain is relieved, and very rarely become addicted.[20-22] Similarly, it has been postulated that addicts who do not "mature" out of their addiction might not have access to alternative reinforcers, might not seek them out, or find them unsuitable replacement for the more potent reward derived from drugs or other deviant behavior. In either case, selection of reinforcers is a choice heavily dependent, not on genetics, but on personal, familial, and psychosocial factors, all of which contribute to forming an individual's personality, itself made of traits such as self-control, discipline, and willpower. Self-control is the restraint exercised over one's own impulses, emotions, desires, and actions that are the foundation of behavior. Self-control, which embodies the concepts of character, will power, and discipline, is a personality trait that is learned mostly from parents but also from family members, teachers, friends and acquaintances, and from a variety of life experiences. Depending of the individual's environment, this learning process can lead to a strong or to an impaired self-control with positive or negative consequences, respectively. For instance, a child well nurtured by dedicated and loving parents in a stable family and social environments is likely to develop robust character traits suitable for making appropriate decisions emulating learned positive patterns of behavior conducive to personal success and to the avoidance of deviant choices later in life. In contrast, a child neglected by uncaring parents, themselves lacking strong character traits, and surrounded by a perfidious environment is unlikely to develop personality traits protective of the many life pitfalls, is less well prepared to face psychosocial challenges, and is therefore more likely to engage in deviant behavior later in life. [37] One pioneering study that supports the importance and predictive value of early development in self-control is known as the "marshmallow" test [38] conducted in the 1960's by Walter Mischel to examine preschooler's ability to forego immediate gratification waiting instead for a larger but delayed reward. In the study, a group of 4- year olds were given a marshmallow and promised another if they would wait 20 minutes before eating the first one. Following the progress of each child into adolescence, via surveys of their parents and their teachers, the researchers found that ability (self-control) to delay gratification was both predictive of favorable personal outcomes (e.g. higher SAT* scores and educational attainment and better social and cognitive competence), and protective against a variety of potential vulnerabilities later in life (e.g. lower drug use). Jellinek first described the concept of "loss of control", in the context of alcoholism, as an inability to stop drinking leading to binge drinking. However, the concept has evolved to include the inability to refrain from substance use (or from engaging in other deviant behavior) and to terminate use or activity once begun. Yet, from Jellinek's time "loss of control" is widely believed to be relative rather than absolute. That is, addicts are capable of exercising some degree of control over their behavior, at least some of the time.[39] How "loss of control" relates to the behavior reinforcing effects of the stimulus, whether drug or activity, or to the addict's expectation of pleasure from a particular impaired control behavior is largely unsolved and hotly debated. Neurobiologists and electro-physiologists are now searching for neural correlates of self-control and of behavior that might explain how the brain

* Scholastic Aptitude Test

assigns relative value to different incoming stimuli in order to select the most appropriate. For example, alcohol effects on the brain include impaired frontal lobe tasks as judged by PET* scans, by metabolic studies, and by a neuropsychological test battery designed to assess cognitive functions and processes.[40] Frontal lobe dysfunction, whether associated with decreased or increased activity, has also been documented for many psychoactive agents. However, the key question is whether drug-induced frontal lobe dysfunctional changes predate abuse, which would support genetic predisposition, or are the result of abuse, which would not. That is, whether they are the cause or the result of impaired self-control. Partial recovery from chronic alcohol-induced frontal lobe dysfunction following an extended period of abstinence suggests dysfunctional frontal lobe changes to be, not the cause, but the result of impaired control and of abuse behavior. Hence, biological correlates of abuse are expected to complement our understanding of the neurobiological processes associated with the brain's handling of reinforcing stimuli but are unlikely to account for addiction and other deviant behaviors. In the meantime, "behaviorists reject the prevalent neuro-scientific notion that drugs themselves are responsible for the development of addiction, and see addiction not primarily as a 'brain disease' but as a behavioral disorder that cannot be separated from the prevailing and historical contingencies of reinforcement" [31]. Simply put, like any deviant behavior addiction is a deliberate "choice" that, while influenced by genetics, converges and mirrors an individual's formal and informal education, and encompasses all previous life experiences. The notion that addiction is born of learned behavior and life experiences is not only common sense but has been corroborated by two decades of research.[40-47] Moreover, the behavioral model surpasses the disease theory of addiction in three major aspects. First, it accounts for the behavior of a majority of individuals who seek drugs and of the millions of casual users and pain patients who do not become addicted despite repeated or protracted drug exposure. Second, it provides a foundation for understanding and explaining all forms of addiction, whether substance or non-substance related. Third and most importantly, it promotes prevention, self-control, and treatment modalities aimed at restoring addicts' discipline and willpower, empowering them to develop new behavioral patterns instead of perpetuating the myth that they are powerless victims, as embodied in the disease model of addiction. Hence "solutions [for controlling addiction] that do not take into account the basic motivations and propensities underlying addictive behavior are destined to failure."[48] In conclusion, scientific evidence shows that addiction cannot be blamed on an alleged intrinsic addictive power of drugs, on genetics, or on neurocircuitry reward pathways, and is not a disease or an indication of moral turpitude, as it is often claimed. There are at least three lines of reasoning that support the view that addiction is not substance-dependent but behavior-driven. First, an extensive repertoire of addictive behaviors extends well beyond drugs. Indeed, the addiction spectrum includes not only licit and illicit drugs (e.g. alcohol, narcotics), prescription medications (e.g. painkillers, tranquilizers), and chemical substances (e.g. inhalants, glues) but extends to a variety of dietary products (e.g. caffeine, chocolate), certain activities (e.g. internet browsing, gambling, exercise, sex), or even ordinary foodstuff, which the vast majority of the population indulges in moderation. Second, no substance or activity is intrinsically addictive. For instance, narcotics and foodstuff are not addictive when taken for pain relief and nourishment, respectively, but can become reinforcers of addiction when indulged for pleasure. Third, the vast majority of casual drug users do not become addicted and most who do eventually free themselves from their addiction, most often without help. Likewise, no specific genetic mutations or brain pathways have been identified that doom the carrier to addiction, and no chemical rewards have been shown \to compel a casual user to engage in substance or non-substance abuse and become addicted. In the words of a noted critic of the disease theory of addiction, "The idea that addiction is a disease is the greatest medical hoax since the idea that masturbation would make you go blind."[49] However, addiction can lead to certain diseases and even to self-destruction as possible outcomes. Examples include liver cirrhosis from alcoholism, lung cancer from smoking, AIDS in addicts who share needles, and death from any of them. Equating cause

to effect of addiction has been and continues to be exploited by proponents of the disease theory of addiction in part out of conviction but also driven by personal self-interest and to protect a multitude of highly lucrative businesses that profit from it. However, while addiction is not a disease addicts are not criminals to be persecuted and incarcerated as it is done the world over, or stigmatized as sinners. Indeed, addiction is not a crime (though certain drugs of abuse have been criminalized worldwide), nor is it a sign of moral or spiritual decay, or a sin (though it is so viewed in most religious circles). Rather, "People take drugs because it makes sense for them to do so given the choices available, rather than because they are compelled by the pharmacology of the drugs they take."[50] Indeed, evidence shows that addiction is a chosen behavior, just as are all choices that addicts and non-addicts make on a daily basis. In fact, everybody makes unwise choices in life that can lead to adverse outcomes such as addiction, a failed relationship, a disease, a financial loss, and even death. As in any decision-making, unwise choices most often are based on an analysis of available information that is influenced by personality traits of self-control, discipline, and willpower developed from one's life experiences superimposed to a non-determining genetic background. That being the case, three key questions arise: Should society enact social policies to prevent individuals from making unwise choices? When individuals make bad choices, should society intervene? When unwise choices lead to addiction, should addicts be held accountable? Abandoned to their fate? Treated and rehabilitated at society's expense?

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