



# The conceptual basis of addiction memory, allostasis and dual processes, and the classical therapy of addiction

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## Abstract

**Purpose:** In recent years, research has yielded new information regarding the impact of intense, long-term alcohol consumption on the development of permanent changes in the central nervous system. The present study examines the mechanisms related to the existence of addiction memory, sensitization and allostasis. A dual-process model was also created, which analyses the role of conscious and automatic mechanisms in the functioning of addicts. The aim of the article is to present these mechanisms and to consider the implications of their existence for the course of therapy.

**Views:** The mechanisms analysed shed new light on some of the negative phenomena occurring during and after therapy, such as frequent abstinence after treatment, switching addictions, and returning to drinking after a long period of abstinence. The existence of these mechanisms should also change the character of addiction therapy, which has so far focused mainly on conscious aspects and ignored the existence of automatic ones. Attempts are already being made to implement the dual-process model in addiction therapy.

**Conclusions:** A better understanding of the mechanisms resulting from the dual-process model can significantly influence perspectives regarding functioning in addiction and the course of therapy. These processes merit further research, as do possible therapeutic interventions based on them.

**Key words:** dual-process model, addiction memory, addiction therapy.

## INTRODUCTION

Since the introduction of modern therapy for various forms of addiction, including to alcohol, both theoreticians and practitioners have been seeking reasons for its relatively low effectiveness. The everyday experiences of therapists testify that despite experiencing very strong aversive reactions, which can often be life-threatening, many patients fail to check their destructive behaviour. Many others return to substance use relatively quickly after treatment, even those after a period of sustained abstinence, although sometimes less severely than before. It is also common for patients to “swap” addictions (e.g., from substantial to non-substantial), and finally there are situations in which people with alcohol use disorders return to active substance use after long abstinence, with the effects being as destructive as before. In addition, many people with addiction have been observed to demonstrate apparently in-

comprehensible and illogical behaviour. As such, it is only natural to explore the reasons behind this state of affairs.

Until now, little consideration has been given to the processes involved in the changes occurring in the central nervous system as a result of an intensive intake of addictive drugs; in particular, few studies have examined their impact on the behaviour of people with addiction, both during therapy and afterwards. These processes concern, among others, addiction memory [1-6], sensitization [7-9], allostasis [10-12], and above all their resultant dual-process model [13-17]. It is worth noting that these phenomena have been described or thoroughly investigated only recently, i.e., since the 1990s.

The aim of the article is to briefly present the above-mentioned processes, together with their possible implications for addiction therapy, which may contribute to improving the effectiveness of therapy.

## NEUROADAPTATION AND NEUROPLASTICITY

To understand the role of these phenomena, it is first necessary to review their background, which is closely connected with the processes of neuroadaptation and neuroplasticity.

In the case of addictions, the term *neuroadaptation* refers to anatomical and/or physiological changes in the functioning of the reward system as it “tries” to adapt to the intensive and repeated administration of psychoactive substances. This adaptation results in changes in the “baseline” levels of, *inter alia*, dopamine, glutamic acid, gamma-aminobutyric acid (GABA) and endogenous opioids. Such changes result in the substance gaining new positive and negative effects, and thus an increase in tolerance, and the occurrence of withdrawal symptoms in the event of an abrupt discontinuation or reduction of intake.

The length of time and intensity of substance use needed to induce temporary or permanent neuroadaptation depends on genetic factors and the age of onset of use, and in some people the process may begin relatively quickly. Most importantly, neuroadaptation is considered to be one of the processes responsible for the transition from recreational to compulsive substance use [18-20].

On the other hand, neuroplasticity is understood as the ability of the brain to undergo change, create new paths aimed at consolidating memory traces and learn new information. In the case of addiction, the dopamine system may become dysregulated, resulting in changes in the activity of neurotransmitter pathways, especially when the individual is under the influence of a substance. In addition, physiological changes may change in locations where traces of the rewarding effects of alcohol are stored [21-23].

## ADDICTION MEMORY

It may be that the genesis of the phenomenon of addiction memory lies within that of neuroplasticity. It can be said that repeated use of psychoactive substances causes the formation of specific, strong and long-lasting memories regarding both the internal experience evoked by these substances and their associated environmental stimuli [2]. This is described in more detail by Anton [21], who emphasizes the role of changes in the nucleus accumbens, amygdala and frontal cortex occurring during the development of addiction in places where memory traces of the rewarding effects of the substance are stored. In turn, Di Chiara [24] report the appearance of permanent memory traces during the development of addiction. These induce dopamine secretion in response to stimuli associated with substance use, thus consolidating the neural circuits underlying drinking behaviour.

Such *addiction memory* involves a general memory of loss of control and a memory of the effects specific to

the substance. It can be activated by internal or external signals, even after a long period of abstinence; it drives conscious and unconscious desires, changes the perception (assessment) of the environment, reduces satisfaction with living in a sober way and influences the ability to plan [25-27]. In the early stages of abstinence, these processes can lead to a feeling of *hunger*. In the case of long-term abstinence, exposure to stimuli related to previous drinking, or the emergence of memories of past experiences, together with the occurrence of strong emotions, can activate the memory of drinking together with all its consequences [21, 25]. According to Lindenmeyer [15], this phenomenon occurs not only in addictions: similar hypotheses, regarding the risk of relapse due to the activation of permanent memory traces, have been described in the case of recurrent obsessive-compulsive disorders, depressive disorders, post-traumatic stress disorder (PTSD), sexual disorders, chronic pain and anxiety disorders. Similarly, Wolffgramm *et al.* [28] believe that these traces are permanent, in fact inextinguishable, and that their formation resembles that of traces in PTSD.

The most serious effect of the development of addiction memory is the permanent impairment of the participation of cortical functions in decision-making (e.g., regarding drinking control). In other words, the brain becomes particularly sensitive to, broadly understood, stimuli related to substance use, and the regulation of behaviour is increasingly taken over by the drive system and automated behaviour [3]. This seems to be in line with the definition of Wojnar [29, p. 63], who describes addiction as: “a developing imbalance between the activity of higher and lower brain structures resulting from dysfunction of the connections and communication between them (...) (there is a) weakening of cortical control mechanisms and, on the other hand, the disinhibition and uncontrollability of the subcortical centres (...). As a result, behaviour becomes increasingly impulsively or compulsively directed by the lower structures of the brain without the control and balance of the higher structures”.

This perspective significantly changes the way relapse is viewed, recognizing that the persistence of maladaptive memories associated with substance use is a key factor in achieving abstinence. These memories can sustain the search for psychoactive substances and cause unconscious relapses of certain behaviours. Thus, as defined by Milton and Everitt [27, p. 2]: “addiction can be conceptualized as a disorder of abnormal learning: creating strong memories combining actions with seeking drugs and expecting results”.

For these reasons, the development of treatments that can disrupt memories associated with drugs and alcohol is gaining importance as a goal of addiction research. However, to achieve this goal, it is first necessary to understand the basic mechanisms through which these substances regulate memory formation [6, 26].

## SENSITIZATION

Sensitization theory, another extensively tested neuro-adaptation concept, attempts to explain the commonly observed mechanism by which stimuli heralding contact with a substance trigger a strong motivation to seek and take it. This motivation is accompanied by psychomotor agitation and a focussing of attention (attentional bias) on addictive substances [22]. The most well-known concept in this area, “The incentive sensitization theory of addiction” of Robinson and Berridge [7-9, 22], assumes that the increasingly intensive and repeated intake of substances result in long-term changes in the functioning of the reward system, particularly in the elements of it responsible for wanting the substance (wanting). As the key role in this mechanism is played by dopamine, these changes occur as neuroadaptive changes in both the pre- and post-synaptic level of the dopaminergic mesolimbic-cortical system. It is these changes that, in certain situations, lead to compulsive behaviours aimed at acquiring the substance [30].

However, and most importantly, the mechanism described does not concern the pleasure resulting from receiving a reward (liking), as this is the duty of the opiate system; rather, it is associated with the mechanisms of desiring a substance (wanting) [7, 8, 29]. The problem, and the burden borne by those with addiction, is that with the increase in sensitization, the frequency and intensity of compulsive seeking behaviours (wanting) increases, but the pleasure (liking) from receiving a reward (drinking alcohol) does not; in fact, the reward fades over time, thus resulting in the vicious cycle mentioned above. Long term abuse therefore results in an increasing discrepancy between the subjective feeling of reward, and the willingness to seek and obtain it [7, 9]. In such a situation, the user must increase the dose of the substance, combine it with others, or “change addictions” to achieve the same level of reward.

When discussing sensitization and attempting to explain many of the seemingly incomprehensible behaviours associated with addiction, it is necessary to explore its varied aspects. Firstly, as emphasized by Kostowski [30], sensitization is largely influenced by the signals coming from the environment and the course of the learning process, an example of which is *context sensitization*: sensitization often does not occur if an animal is administered a substance in a different environment than the one in which it was previously exposed. Another important aspect concerns individual variation – it has been shown that sensitization is produced more easily in some animals than in others, and some animals may not become sensitized at all. In addition, sensitization is permanent, once produced, it tends to last for months or even years, although individual differences also occur in this regard [7, 9]. It should be noted, however, that while the concept is still

not free from imperfections, being based mainly on animal models [30], the authors of the model emphasise that the evidence for its validity is still growing and that the concept includes behavioural addictions [7].

## ALLOSTASIS

The concept of allostasis assumes that during the development of addiction, specific changes occur in the reward system as a result of an intensive intake of a psychoactive substance. These changes overload natural homeostatic processes, countering the excessive and “artificial” stimulation of the system by the substance, and over time these prevent the restoration of the previous equilibrium [10, 12]. The processes reach a new balance point known as *allostasis*, i.e., homeostasis on a non-physiological level. This balance is maintained as long as the substance is present in the body. However, this new “artificial” balance is very unstable, being regulated pathologically through the intake of substances. Therefore, when the substance runs out (withdrawal) there is no return to the previous “healthy” homeostasis; instead, strong and very unpleasant physiological and emotional symptoms arise that drive the individual to take the substance again. Thus, in the development of allostasis, there are both positive reinforcements (e.g., drinking for pleasure) and negative reinforcements (drinking for relief).

In the initial stages of development, the state of allostasis is associated with impulsive seeking behaviours, and later with compulsive behaviours aimed at the immediate relief of suffering. In such cases, even relatively mild stress or pain stimuli, or the appearance of external risk factors, may cause a further reduction in the level of allostasis, thereby increasing the suffering and stress of the addicted person, and thus the desire to alleviate it [10-12, 31, 32]. The mechanism of allostasis not only occurs with active substance use, but also after its discontinuation. Its consequence may be chronic anhedonia (hedonic allostasis), characterized by a lack of pleasure from natural rewards; this may be associated with craving, relapse and a tendency to “swap” addictions [e.g., 33].

## THE DUAL-PROCESS THEORY

These observations have provoked a change in thinking about the genesis and course of behaviour associated with addiction, which is best described by the dual-process model of Wiers and Stacy [13-17]. According to the model, the stimuli associated with alcohol use to which an addicted person, or heavy drinker, is exposed trigger both automatic (the aforementioned specific information processing, specific evaluation of stimuli, psychomotor agitation, attention bias) and conscious processes (controllable information processing, reflexivity, attribution of causes). While automatic processing happens

quickly and is difficult to become aware of, much less stop, conscious processing and intentional regulation of action take place more slowly; they also show connections with behavioural motives, volitional and cognitive factors, and are influenced by the developed skills (e.g., during therapy) of coping and finding alternative solutions. Importantly, the automatic and conscious systems are linked to separate brain networks: this distinction is extremely important because while addiction is frequently associated with automatic behaviour, the changes expected during treatment require conscious information processing [15].

Based on the dual process theory, the development of addiction can be seen as the progressive reinforcement of the role of impulsive and automatic behaviour, and the weakening of the self-control system, which perpetuates the cycle of continuing substance use despite an increasing number of negative consequences. The first, situational signals of this process can already be seen in people who engage in harmful drinking, as well as periodical binge drinkers [34, 35].

In turn, the behaviour of those trying to maintain abstinence can be described as functioning in a “shaky balance” of conscious and unconscious processes, and when the balance is disturbed in favour of the latter, it is easy to return to the previous addictive behaviour [13-15].

## PRACTICAL IMPLICATIONS

The above theories, which are all interconnected, explain many of the behaviours observed during addiction. Lindenmeyer [15] indicates that they can account for the seemingly incomprehensible fact that many people aware of the dramatic, and possibly fatal, consequences of their drinking, and often return to using substances in difficult and/or triggering situations, and even under the influence of relatively “trivial” reasons. Such returns can occur despite support provided by therapy and self-help groups, and despite promises and “oaths” given to loved ones. The theories also explain the frequent “addiction switch” in patients, as well as the permanence and invariability of addictive behaviours, which are often repeated in an identical way, even after a long period of abstinence and multiple therapies.

Further implications concern the course of therapy for addicts. In most cases, current approaches to addiction therapy are based mainly, and sometimes exclusively, on conscious processes. Their goal is to help patients maintain permanent abstinence and improve their physical and mental health. During treatment, the patient should gain an insight into their own thought processes, implement new, constructive behaviours, and learn to solve their emotional and life problems. Therapy comprises a number of important elements such as gaining knowledge about the condition, learning to recognize relapses and deal with them, improving contact with the environment, and es-

tablishing relationships with self-help groups [31, 36, 37]. However, adopting the dual-process perspective implies that therapeutic interventions should be extended to include those that take into account the existence and role of automatic processes [17]. Such proposals exist, and their implementation so far gives rise to cautious hope for improving the effectiveness of help in overcoming addiction.

One new approach is neuropsychological training, such as joystick training and computerized cognitive bias modification (CBM), referring to attention bias and sensitization. Such training consists of the presentation of a series of pictures of alcohol and neutral or pleasant images displayed on a computer screen (or smartphone). The patient’s task is to remove the “alcohol” images from the screen and leave the neutral images. By doing so, it is intended to create alternatives to previous reactions to substance-related stimuli, with multiple repetitions, thus contributing to the reduction of craving and the reduction of abstinence violations after treatment; its effectiveness has been confirmed in a series of randomized studies [e.g., 15: 38-40] and meta-analyses [e.g., 41]. These exercises can also be carried out with the use of virtual reality, assuming that it provides stronger, multimodal experiences that can engage brain networks more effectively than standard methods [42].

Another tested therapeutic proposal is the use of eye movement desensitization and reprocessing (EMDR). The method, initially used in the treatment of PTSD, is now also used in the treatment of addictions. In this case, the starting point is addiction memory theory. According to the adaptive information processing (AIP) model underlying EMDR, representations of high arousal events such as states of alcohol intoxication are stored in the memory along with their accompanying emotions, bodily reactions and physical sensations. EMDR therapy is believed to weaken the primary memory and disrupt the chain of associations, which can reduce the craving for substances and withdrawal of exploratory behaviour. Reports on the effectiveness of EMDR in addiction therapy are promising, although further research is needed, especially on its long-term effects [43, 44].

The use of mindfulness techniques, or mindfulness-based cognitive therapy (MBCT), also merits attention. The use of these techniques, initially used to support the regulation of emotions in the treatment of depression, and later various addictions, including alcohol, has become popular in recent years. The main reason for the interest in mindfulness as an auxiliary method in the treatment of addictions is the generally known fact that the main problem for many addicts is the dysregulation of emotions, which is not addressed by traditional therapy [45]. Mindfulness, understood as awareness arising as a result of intentional and non-judgmental attention to currently experienced emotional, mental and physiological states, is the opposite of withdrawing from experiencing aversive experi-

ences and suppressing unacceptable thoughts [46, 47]. Regular mindfulness practice not only allows more effective emotional regulation and stress reduction, but also provides the patient with the ability to identify triggering situations, and to experience hunger and deal with it without the strong discomfort, dysphoria and remorse typically experienced in such situations. As a result, the automated associations between craving/stress and abstinence violations are weakened. The effectiveness of mindfulness techniques in the treatment of behavioural and substance addictions has been confirmed in a number of randomized studies and in a 2018 meta-analysis [45].

These approaches, based on automatic processes, may be used to supplement traditional addiction therapy. However, other possibilities also exist, such as cue exposure treatment (CET), i.e., exposure to alcohol stimuli with extinction of reactions, which proceeds similarly to anxiety stimuli in cognitive-behavioural therapy. A number of reports have examined the effectiveness of this technique, although the results are currently inconclusive [15, 31]. Special forms of training are also being tested, such as cognitive control methods including goal management training (GMT) and habit reversal therapy (HRT). Such training, previously used to treat strokes, brain injuries, ADHD and Tourette's syndrome among other things, is

aimed at strengthening the ability to control thoughts, emotions, attention and behaviour and to introduce new, goal-oriented behaviours [48].

## CONCLUSIONS

Recent research has led to the creation of a number of extremely interesting theories regarding the appearance of permanent changes in the central nervous system following intense and long-term alcohol consumption. These have been combined in a dual-process model highlighting that both conscious and automatic processes are independently involved in addiction. This review describes the automatic processes and their implications for therapy. This is an important consideration, as conventional treatment of addictions has focused mainly on the existence and modification of conscious processes. However, recent years have seen a growing number of attempts to supplement traditional therapy with methods and techniques based on automatic processes, and their results appear promising. Despite this, these concepts are relatively new, and there is a need for more practical implementations of the dual-process model, as well as the creation of new interventions, and further research on the short- and long-term effectiveness of these interactions.

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