SUPPLEMENT TO

CHAPTER 5, V1[1]

EMOTION

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ADDICTIONS

Clinical psychologists frequently see people who are substance-dependent, addicted, or who live with someone who is. Understanding addiction is therefore an important topic for clinical psychologists to know about. Cognitive neuroscience substantially informs our understanding of addictions to drugs, alcohol, gambling, food, and sex. This supplement emphasizes that addictions can be understood as an extension of the mechanisms that generate learning as a memory formation process. Addictions are mediated by the limbic system where dopamine is the primary neurotransmitter. The nucleus accumbens is central to the reward system. The limbic system has remained essentially unchanged over the last 100 million years and is found is mammals including monkeys, dogs, cats, rats, and mice which is why research on these species generalizes to humans.

Tryon (2014) discussed learning as a memory formation process based on the experience-dependent plasticity neuroscience principle where synaptic transmission is strengthened on the basis that **neurons that fire together wire together**. These events can be summarized by the phrase **Use It or Lose It**.

All processes and mechanisms have limits. At some point the brain makes an adaptive response to excessive stimulation by weakening synaptic transmission in two ways. 1) The amount of neurotransmitter[2] is reduced and 2) the number of post-synaptic receptors is reduced. Together,

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[1] V1 stands for Version 1 which implies that subsequent chapter updates will become available.
[2] The following URL provides basic neurotransmitter information:
http://en.wikipedia.org/wiki/Excitatory_neurotransmitter

these two physical changes jointly reduce the post-synaptic impulse. Hence, the same stimulus (drug, alcohol, gambling, food, sex) produces a lesser effect. This effect is known as **tolerance**. The typical behavioral response to tolerance is to increase the stimulus by taking a higher dosage, drinking larger quantities of alcohol, gambling more, eating more, and/or having more sex or watching more porn. These behaviors further reduce transmitter release and prune more post-synaptic receptors thereby exacerbating the underlying neural network problem. This negative downward spiral can be summarized by the phrase **Over Use It and Lose It**.

Effective treatment requires that the stimulus of choice no longer be applied. Abstinence can create withdrawal symptoms but these stimuli are just what is needed to activate the synthesis and release of more neurotransmitter[3] and to stimulate the regrowth of post-synaptic receptors but people typically seek to escape and avoid these symptoms by restarting their habit. This process is aggravated by **sensitization**; the fact that the desire to restart the habit increases as a result of over stimulation; i.e., the threshold to restart the habit in the presence of classically conditioned cues is reduced. Sensitization occurs because ΔFosB, a member of the Fos family of transcription factors, accumulates in the cells of the nucleus accumbens and striatum as a consequence of over stimulation. Unlike other transcription factors, ΔFosB is highly stable and promotes reward motivation to relapse over an extended period of time (Nestler, 2013; Nestler, Barrot, & Self, 2001). Addiction is the term we use for the combination of tolerance and sensitization effects. Successful treatment of addiction entails careful management of how the dissonance induced by withdrawal symptoms and sensitization is reduced. This is another example that **Principle 8: Dissonance Induction / Reduction** characterizes all effective therapies.

Epigenetic effects compromise recovery. For example, Tryon (2014, p. 199) reported that a single administration of cocaine has been shown to activate nearly 100 genes in the nucleus accumbens; the reward center of the brain. These genes will return to normal within a week unless reactivated by more cocaine which can persistently over activate many of these genes. Addiction is therefore a behavior-induced genetic-mediated brain reward center modification problem. The unanswered clinical question is how long it will take for these epigenetic changes to reverse.

The URL www.yourbrainonporn.com contains an informative six-part video tutorial that illustrates these physical changes in the context of Internet porn induced erectile dysfunction; a

[3] Dopamine is the major transmitter.

topic of increasing relevance to men of all ages and their sexual partners. High speed Internet porn is among the more recent wide spread addictions. In men, extensive viewing of highly erotic images can result in erectile dysfunction (ED), low libido, and disinterest in sex with real women. Porn-induced ED occurs now in young men including teenagers. The basic process is as follows. Each highly erotic image viewed releases dopamine that activates the nucleus accumbens in the limbic system. The normal "Use It or Lose It" principle does not operate here. Unfortunately, the opposite principle of "Over Use It and Lose It" principle is the case. The brain adapts to the high levels of dopamine in the two synergistic ways described above. These dual actions gradual reduce the ability to have an erection under all but the most extreme and novel circumstances. The typical male reaction is to seek out more extreme and novel porn and/or more exciting circumstances for having sex. Such actions may temporarily work but the predictable result is less dopamine and fewer post-synaptic dopamine receptors. The result is that young men and even some teenager's experience ED.

REFERENCES[4]

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[4] References not included in this list are in the reference section of the Tryon (2014).