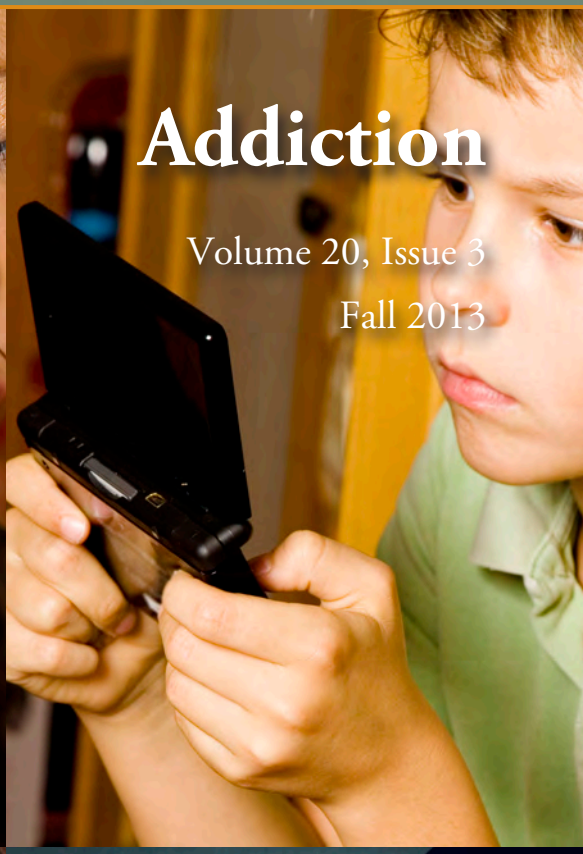
Vital Link

The journal of the Canadian Association of Naturopathic Doctors

Feature Articles

- The Psychology of Addiction: A Literature Review
- Addiction, The Song and Dance
- Naturopathic Treatment of Addictions
- Electronic Media Addiction: A Literature Review
- Current Concepts in Food Addiction



Addiction

Volume 20, Issue 3

Fall 2013

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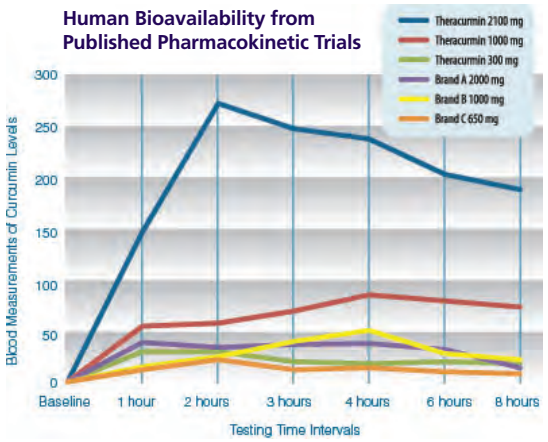

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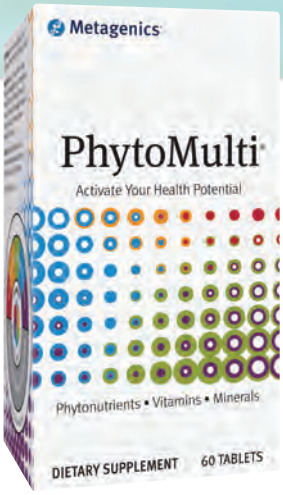

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Vital Link

Volume 20, Issue 3, Fall 2013
Addiction

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Managing Editor and Advertising
Alex McKenna • amckenna@cand.ca

Publisher
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Layout and Print
J Sutton Communications • jsuttoncom.com

Mailing Address
Vital Link c/o Alex McKenna
CAND 20 Holly St., Ste. 200 • Toronto, Canada M4S 3B1
Phone 416.496.8633 • Fax 416.496.8634 • cand.ca

The *Vital Link* is the professional journal of the Canadian Association of Naturopathic Doctors (CAND). It is published primarily for CAND members and features detailed reviews of specific causal factors: philosophical and research-based papers, clinical practice articles and case reviews, as well as international updates on the profession. The *Vital Link* has an outreach to other health care professions and promotes qualified naturopathic doctors to corporations, insurance companies and the Canadian government.

Forthcoming Themes

Spring 2014 Assessing the Health Impacts of Pharmaceutical Medications
Summer 2014 True Cancer Prevention
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When writing for the *Vital Link*, keep in mind its broad readership and outreach to other professions. Your contribution to the *Vital Link* will benefit the naturopathic profession as a whole and provide you with personal professional exposure. Previously unpublished material is preferred. Please contact the managing editor for submission guidelines.

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The *Vital Link* is published three times per year and is distributed to over 2000 qualified Canadian NDs and students of CNME-accredited naturopathic programs in Canada and the U.S. The *Vital Link* is also distributed to the CAND's corporate members and in our media kit. The journal is available in print and e-formats, by paid subscription.

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Naturopathic Notes

Dr. Iva Lloyd, BScH, BCPP, ND

Few topics cause as much confusion as the subject of addiction. In this edition of the *Vital Link* we explore the world of addiction, addressing the current research, the debates and considerations regarding definitions and the role of naturopathic medicine in the assessment and treatment of addiction.

Dr. Aaron Van Gaver explores the world of alcohol and illicit substance abuse. He addresses such questions as: What is the line between occasional or recreational drug use and a true addiction? What are the telltale signs that your patient might be struggling with a drug or alcohol addiction? How to assess for addictions as part of a naturopathic intake? Alcohol and recreational drug-use is rampant in our society. One of the roles of naturopathic doctors is to help patients understand the health impacts of their substance use, and assist them in recognizing when "occasional use" is becoming, or has become, problematic.

Dr. Maureen Horne-Paul shares her experience as an addictions-recovery practitioner. She explores the myths associated with addiction and the role of neurotransmitter balance in addictions. She emphasizes the importance of addressing the root causes of any addiction. Dr. Horne-Paul's article expands on the labs indicated in the assessment and treatment of addictions and the various naturopathic treatment options available.

The notion of an addictive personality was first introduced in 1965. Dr. Nicole Daniels takes us through the research, history and debate about addictive personality disorders. She reviews the traits or risk factors which predisposes someone to addictive behaviours, including genetic, psychological and environmental factors. Dr. Daniels provides details on the neurobiological impairment and dysfunctions associated with addictions and also treatment guidelines for practitioners. Understanding the multi-factorial aspects of addictions is beneficial when working with patients with any form of addiction.

Naturopathic doctor candidates, Kaeli Sweigard, Sara Ip and Marie-Jasmine Parsi look at the growing concern about electronic media addictions. The article reviews the latest research on the habitual use of the Internet, video games and cell phones. Not only is heavy electronic media use accepted, but it is sometimes expected and even encouraged across all age groups. The article looks at pathological or problematic internet use and how it can affect behaviour and mood. The high comorbidity of internet addiction with other disorders such as anxiety, depression, gambling, substance abuse and ADHD is explored.

Video game addiction is a relatively new concept that has gained attention over the last couple of decades. This article expands on a growing concern about video game use and how it affects not only children but also adults. The issue of cell phone addiction has been complicated by the introduction of various media platforms, including text messaging, internet browsing, and game playing that are now available on cell phones. The problematic use of cell phones causes a number of issues ranging from risky behaviour, social harassment and social and work life disruption to depression, anxiety, radiation exposure, oxidative lens stress and musculoskeletal pain. Exploring the impact of electronic media is becoming an essential part of a thorough naturopathic intake.

Another area of addiction loaded with confusion and misunderstanding is food addiction. Drs Penny Kendall-Reed and Stephen Reed explore the world of food addiction and provide a detailed overview of the current theories behind appetite control, food cravings and addiction. By detailing the neurophysiology of these conditions, the authors lay the groundwork for an approach to diagnosis and treatment. The factors contributing to food addiction, such as stress, emotions and serotonin, food choices and genetics are also explored. By integrating the research on the psychological and biochemical aspects of food addictions, these contributors provide a comprehensive review of food addictions and a four-step approach to treatment.

Historically addictions and addictive behaviour have been associated primarily with alcohol, illicit drugs and gambling. It has since expanded and encompasses many different aspects of life. Educating patients about addictive behaviour and its ability to affect so many different areas of a person's life is an essential aspect to naturopathic care.

The next *Vital Link,* to be published in spring 2014, will explore the topic of prescription medications. If you are interested in contributing to our next edition, please contact us. As always, we welcome your opinions and questions.

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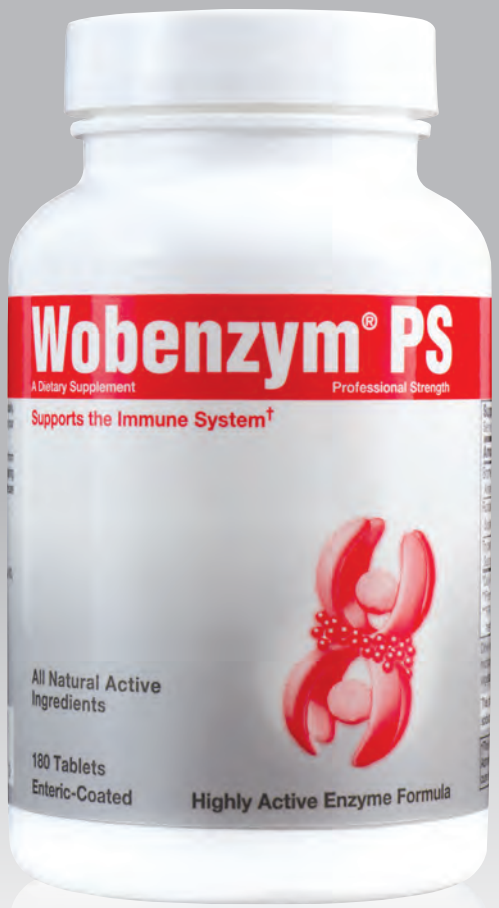

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The Psychology of Addiction: A Literature Review

Dr. Nicole Daniels, ND, MSc, BMSc

According to the Canadian Mental Health Association (CMHA); one in ten Canadians over the age of 15 report symptoms of addiction or substance dependence.[1] Addiction is defined as a chronically relapsing disorder characterized by a compulsive dependence on a substance or behaviour, the loss of control of limiting intake, and negative emotional symptoms, such as anxiety and irritability, when the substance is removed.[2]

The most common substance dependency is alcohol, followed sequentially by *Cannabis sativa*, prescription painkillers, nicotine, and illegal drugs.[1] In addition to drug abuse, other common addictive behaviours include, exercise addiction, binge eating and food addiction, compulsive buying, sex addiction, obsessive tanning, computer addiction, self injury and gambling.

The existence of an "addictive personality", first introduced by VanKamm in 1965,[3] has been debated and discussed by numerous researchers.[4] Their consensus is that there is no definitive "addictive personality"; however, there are certain traits or risk factors, which predisposes one to addictive behaviours. Addiction is a multi-factorial syndrome, resultant from a combination of genetic, psychological, and environmental factors ultimately creating neurobiological impairment and dysfunction.

Addiction Risk Factors

Biological or genetic factors

The estimated heritability of substance dependence is approximately 50%, where the impact of genetic factors tends to increase in adolescence.[5] Linkage and association-based genome-wide studies have identified a number of chromosomal regions containing variants that confer susceptibility to substance dependency, most commonly variation in the mid-region of human chromosome 7.[6] Population-based transmission studies suggest common genetic factors contribute to substance dependency equally in both genders. Genetic variations predominately affect dopamine levels in the mesocorticolimbic brain reward pathway.[6]

The mesocorticolimbic pathway is a complex neural pathway essential in higher order cognition. It consists of dopamine-producing cells that originate in the ventral tegmental area of the midbrain, which project to various forebrain regions including the nucleus accumbens (NAcc), medial prefrontal cortex (mPFC) and amygdala. This highly conserved neural circuit is thought to play a critical role in the assignment of motivational value to biologically relevant stimuli, resulting in the production of adaptive behaviors.

Polymorphic variations of dopamine receptors lead to increased dopamine signalling in the ventral striatum, where aggregates of multiple dopamine receptor single nucleotide polymorphisms (SNPs) are associated with addictive behaviour. In addition to genetic variability in dopamine systems, gene studies have also linked genetic modification of opioid and GABAergic systems in addiction.[4] These findings suggest that the genetic factors involved in addiction are cumulative, where an individual with greater genetic variation will have a higher chance of developing addictive behaviours.[4,7,8]

Genetic predisposition to addictive behaviour has been demonstrated in family-based studies - including family/siblings, adoption and twin studies. Bierut *et al.* reported elevated rates of alcohol dependence, compared to control, between siblings, 50% for men and 25% for women.[9] Similar trends were noted for cannabis, cocaine, and nicotine dependence.[6] Merikangas *et al.* reported an eightfold increased risk of drug dependent disorders among biological relatives.[10] Similarly, studies have reported that alcoholism is familial and having an alcoholic parent is associated with a fivefold increase in the risk of alcoholism.[11,12] Rates of substance dependence was found to be significantly higher in adoptive children with biological parents with addictive behaviours, providing support for a genetic component.[13] Twin studies also show genetic susceptibility to addiction, estimating the heritability of alcohol and nicotine dependency around 50-70%, and, cannabis dependency around 34-78%, respectively.[14]

Environmental Factors

Both genetic and environmental factors are co-involved when evaluating the probability whether an individual develops an addiction. Most studies find a greater environmental role on the initiation of substance use and greater genetic component in heavier substance use.[13-15] Adolescents are primarily influenced by their peer

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group; hence, environment and social pressures are a dominant factor in determining substance dependency. *Cannabis sativa* is the most abused substance in adolescents, arguably because of peer pressure to maintain a certain image.[16] Availability and cost of substances also influence dependency. Alcohol is the most readably available and affordable substance, hence it is the most abused substance in all ages. Other important environmental influences in the development of substance dependency, revealed through adoption studies, which are able to isolate the influence of environmental exposures from potential genetic confounds, are parental divorce and parental psychiatric disorder.[17]

Social experiences and the quantity and quality of social attachments and interactions during early development and throughout life greatly influence the susceptibility to addiction. Another important environmental factor, maternal neglect or childhood trauma, is correlated to addictive behaviours later in life.[18] *In vivo* animal studies have shown neurotransmitter system modification within one week of maternal separation. When animals were exposed to prenatal stress they exhibited increased risk and characteristics of addiction.[18] Studies of drug addicts have repeatedly shown high percentages of childhood trauma (sexual, physical, emotional or abandonment) where for each traumatic event the initiation of substance abuse increased two to four fold, and seven to ten fold for more than five traumatic events.[19] Traumatic events alter neurobiological stress mechanisms in the child's brain making them hypersensitive to stress and more susceptible to substance abuse.

Psychological factors

There are a multitude of psychometric scales measuring significant psychological traits to predict addiction predisposition. The most accurate and widely used scale is the Addiction Scale of the Personality Inventory by Eysenck.[20] Personality is comprised of the following dimensions: extroversion/introversion, neuroticism/stability, and psychoticism/socialisation.

Psychoticism is one of three traits used by the psychologist Hans Eysenck in his psychoticism, extraversion and neuroticism (P-E-N) model of personality. Psychoticism refers to a personality pattern typified by aggressiveness and interpersonal hostility.

Addiction is correlated with psychoticism, and the specific Addiction Scale measures levels of emotional reactivity, proneness to stress, impulsivity, and negative effect. Elevated levels on the Addiction Scale are positively correlated with predisposition to addictive behaviours.[21] Psychotic characteristic traits seen in addiction are aggression, coldness, egocentricity, impersonalizing, impulsivity, antisocialism, no empathy, creativity, and tough-minded.[22]

In addition to common psychological traits, there is a distorted cognitive belief system shared by addicts.[19] This belief system involves polarized "all or none" irrational thoughts and negative self talk (such as "I am not good enough"). This distorted belief system also dominates in individuals with depression and poor self esteem, and may be the causal link between the high occurrence of depression together with substance abuse.

Neurobiology of Addiction

Genetic susceptibility and environmental cues augment an individual's neural circuitry by down regulating specific receptors and/or neurotransmitters, leading to increased occurrence of addictive behaviours and substance abuse. Specific neurotransmitter and/or receptor deficiencies lead to dependency on specific substances. The neurobiological systems commonly altercated in addiction include the dopamine and opioid circuits, the limbic or emotional brain, and the stress apparatus and impulse control cortical area. As the addiction continues, the brain undergoes further modification and neuroadaptive changes, further increasing substance dependency through positive reinforcement.[23]

The dopamine and opioid circuits

Dopamine is a neurotransmitter inside the brain which regulates motor control, motivation, arousal, cognition and reward. The brain reward cascade starts in the hypothalamus (the mesolimbic system) where serotonin activates endorphins.

Endorphins are released in the hypothalamus stimulating mu receptors in the substania nigra that contains gamma-aminobutyric acid (GABA). $GABA_B$ receptors project onto the ventral tegmental area (VTA) brain region to activate dopaminergic neurons to release dopamine at the nucleus accumbens (reward site of brain).[24,25] Substances that increase dopamine levels will increase the feeling of reward, hence leading to addiction.

Most studies have show that reduced dopamine levels are a predisposing factor for addiction.[25] Genetic studies have shown that polymorphisms in the alleles of dopaminergic receptors, subsequently leading to their down regulation and decreased dopamine levels, are a genetic risk factor in addiction.[7] Also, animal studies have shown that when separated/abandoned by their mothers, rats have reduced dopamine receptors/levels and increased substance dependency, and monkeys have reduced levels of serotonin and increased aggression and tendency to consume alcohol.[26,27]

However, some studies argue that heightened dopamine function is responsible for addictive behaviours. Their reasoning being that low dopamine levels in Parkinson's Disease are associated with low incidence of addictive behaviours; whereas, when medicated with dopamine they develop compulsive and addictive behaviours.[28] Also, there is a positive correlation with childhood trauma, a risk factor in addiction, and seizure activity caused by increased dopamine levels.[29]

All abusive substances in some way exert their rewarding effects by augmenting dopamine levels in the nucleus accumbens. The specific pathway modulated depends on the substance abused. Opioid

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neurotransmission, affected by heroin, is crucial for signalling in the neural reward circuitry, modulating acute and chronic responses to other abusive substances.[30] Increased GABA neurotransmission, affected by alcohol, is the main inhibitory neurotransmitter and synergistically affects the rewarding effects of other substances.[31] Increased nicotine neurotransmission synergistically increases the levels of dopamine, norepinephrine and serotonin by decreasing monoamine oxidase activity, leading to the euphoria and relaxation.[32]

Limbic Brain

In addition to the mesocorticolimbic dopamine system and its projections, specific components of the basal forebrain and amygdala have recently been identified with neuroadaptations to acute drug reward. Ventral corticolimbic control pathways incorporate fast associative learning that is adaptive in low-predictable environments, suggesting an innate tendency to urgently react or give attention to novel addictive stimuli. This physiologically explains why impulsivity is an addictive personality trait. The reward response is the default mechanism in the limbic brain and needs to be actively retrained in order to control addictive tendencies.

Another major contributing factor to the development and maintenance of addiction is the ability of substances to disrupt the neurobiological learning and cellular plasticity, exerting long lasting influences on behaviour. Brain regions are often involved either directly or indirectly. Directly through the prefrontal cortex and hippocampus which form long-term and episodic memories. Indirectly through the amygdala, involved with emotional modulation of memories, and striatum, involved in reward-based memories and decision making in declarative and episodic memory processes.[33,34] The ability of drugs to alter this system may be one factor why they can exert such a strong control on behaviour, becoming the central focus of an addict.[35] Numerous studies have shown smoking addiction to remain prevalent in addicts because of the effect and control of addictive behaviours on memory. Additionally, smoking is a habitual social activity and hence involves negative modification of oxytocin-dopamine interaction.[32]

Oxytocin increases resilience against addiction by facilitating the processing of social and attachment-related information and its consolidation in internal working models, thus promoting a shift from novelty seeking towards preference of social familiarity through the cortical route.[36] Oxytocin is a neurohypophysial hormone released during and after childbirth which facilitates contractions, stimulates lactation, and promotes maternal bonding. Recent studies have shown the role of oxytocin in social recognition and bonding.[36] Oxytocin and dopamine work synergistically, where increases in level of oxytocin from social attachment and love increases expression of dopaminergic neurons and consequently positively affecting neural plasticity and memory.[29] Animal studies have shown that maternal abandonment permanently decreases the production of oxytocin, leading to impaired social skills, anxiety, aggression, trust issues, and increased substance abuse.[29] Recent studies indicate that social attachments protect against addiction and health consequences of stress; whereas, drug abuse and chronic stress can undermine social attachments.[36] Therefore, a healthy social support network is crucial for modifying addictive behaviours.

Stress response mechanisms

Stress is a real or perceived threat, ultimately activating a "fight or flight" response. This response results from the activation of the autonomic sympathetic nervous systems and the hypothalamic-pituitary-adrenal (HPA) axis. Activation of the HPA axis is characterized by pupil dilation, increased heart rate, bronchodilation, and increased sweat. Infants have no ability to regulate their own stress apparatus, hence a child is completely dependant on the relationship with their parents to regulate and dissipate their stress. Maternal deprivation in times of stress will result in permanent high levels of cortisol, which damages the midbrain dopamine system and shrinks the hippocampus, inhibiting memory and emotional processing.[37]

Cortisol is a steroid hormone produced in the adrenal cortex secreted in response to stress, primarily to increase blood sugar and aid in metabolism.

Early stress (such as childhood trauma and/or abandonment) establishes a low set point for a child's internal stress system, leading them to become hypersensitive and overactive to stressful situations, exponentially so when trauma is experienced in adulthood. A brain pre-set to be hypersensitive to stress may actively seek out short-term relief from stress, turning to addictive substances as a form of stress relief. Addiction is an ingrained and learned response to stress, where abusive substances are used to numb or "cope with" stress. However, it should be noted that childhood trauma is not the only determinate of addiction and not all people with childhood trauma will turn to addiction.

Additionally, stress is an influential factor in continued drug use and relapsing. Exposure to stress is the most powerful and reliable experimental manipulation used to induce reinstatement of alcohol or drug use.[38] Stress renders one vulnerable and susceptible to self-medicate or relapse. Animal studies have shown that physical and psychosocial stressors evoked a relapse to habitual addictive behaviours, causing neural atrophy and increased dopamine transmission.[19] Cohort studies have shown that chronic administration of abusive substances deregulates both the hypothalamic-pituitary-adrenal (HPA) axis and brain stress response mechanisms.[39]

Considerations for practitioners treating addiction

Self Disclosure and Childhood Experiences

Many addicts are reluctant to share their life histories with practitioners, requiring trust to be first established.[40] Individuals may be unaware of the link between their childhood experiences and their addictive behaviours, and others will have no memory of trauma. The memory may be purposely undisclosed, or the memory

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may be repressed as a defence mechanism to deal with pain and emotion. Acknowledging and working through childhood traumas are necessary for healing and should be addressed as they are an integral part of the addiction psychology.

Withdrawl, Relapse, and Stress Coping Mechanisms

When treating addiction, withdrawl from substance dependency and occasional relapsing are common occurrences. Human studies have shown that the presentation of drugs themselves, or the stimuli previously associated with their drug delivery, or the state of stress, increased the likelihood of relapse as well as self-reports of craving and motivation to engage in drug-taking.[2] Therefore, it is important for the addict to remove all temptation of substances, including replacing their social network with healthy and supportive social interaction. Taking away an addict's abusive substance is like taking away their "stress coping mechanism". This is why perceived stress is a large determinant of an addict relapsing. Before withdrawing the dependant substance, a healthier mechanism of coping with stress needs to be adopted and practiced. Stress coping therapeutic tools included guided meditation, cognitive behavioural therapy, art/music therapy, social interaction, reading, and exercise.

Technology and Adolescence

Substance dependence peaks in young adults aged 18-25 years-old, and has doubled in youths aged 15-24 years-old during the past five years.[1] Young adults are the most susceptible age group to environmental cues such as peer pressure and parental conflict, hence, prevention and education are key. Part of a health practitioner's role is to educate parents on implementing healthy stress coping skills, and fostering healthy self-esteem to decrease the likelihood of their children developing long-term addictions.

Technological addictions are becoming more prevalent in adolescents. Common technological addictions include use of the Internet, video games, cell phones, TV and social media. Adolescents with Internet addiction, seen in approximately 10% of elementary students, have higher rates of depressive mood, subjective stress, suicidal ideation, anxiety, and other mental health issues.[41] Social network site addiction is associated with health, academic, and interpersonal issues such as depression, anxiety and poor self esteem.[42] Cell-phone addiction is most prominent in females aged 7-13 years-old with low self esteem, and is often associated with depression.[43] Smart phones combine phone technology with Internet, video games, and texting, where the user can access these services at anytime, from anywhere, making smart phones highly addictive. Since the average age a child acquires a cell phone is 7 years-old, preventative measures need to be instated early.[44] Educating parents on establishing healthy boundaries for their children surrounding technology usage and encouraging social interaction at a young age will be protective against addiction.

Summary

Addiction does not have a concrete "personality"; it is a multifactorial syndrome influenced by quantitative genetics, environmental influences, psychotic personality characteristics and childhood trauma. These factors combine to influence and modify the neurochemistry and circuitry of an individual, leaving them susceptible to addictive behaviours and substance abuse. Neurobiological modification includes changes in dopamine levels and the reward circuitry, changes in the limbic brain, and dysfunction of stress response mechanisms. Although these factors predispose an individual to addiction, they do not guarantee that addiction will absolutely occur. In light of predisposition, addiction is a learned and reinforced behaviour which can be prevented and avoided if proper stress coping skills and healthy self esteem are implemented early enough in development.

About the Author

Dr. Nicole Daniels, ND, MSc., BMSc. is a naturopathic doctor working in Newmarket, Ontario. In addition to her naturopathic doctorate she also holds a Masters of Science in Biology. She believes in addressing and investigating all levels of health (physical, mental, emotional, spiritual, and energetic) and also in eclectic treatments to guide patients back to a harmonious healthy balance. Nicole has a special interest in women's health and mental health. With remarks pertaining to the article, please contact Nicole at nd.nicoledaniels@gmail.com. She can also be followed on twitter @NicoleDanielsND and her blog HealthyHappyNaturopathy.com.

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Addiction, The Song and Dance

Dr. Aaron Van Gaver, ND

Imagine the following scenarios:

1) A 32-year-old male patient presents to your office for an overall health assessment and divulges that he consumes 1-2 beer a night.

2) A 23-year-old female comes to you for general health advice, and tells you that she 'parties' the occasional weekend, using the drug ecstasy.

3) A 40-year-old male tells you he smokes marijuana every day, doesn't drink alcohol or use other "drugs" and comes to you because he wants to stay healthy.

As naturopathic doctors we treat a wide variety of patients with a broad spectrum of health concerns and – whether we are aware or not – our patients have a spectrum of issues, and addiction (whether it is to alcohol, tobacco, cocaine, sugar or TV) is often among them.

Regardless of the reason for their initial presentation, all of the scenarios listed above describe patients with a possible addiction, and as naturopathic doctors, many of us have patients who are facing this struggle. Many of us believe addicts are the vagrant "street beggars" who approach us on the street. We don't always think of our patients who have steady employment, a family and social network as having addiction issues. Defined as "…a persistent, compulsive dependence on a behavior or substance"[1] addiction is an individual's inability to stop using a substance or engaging in a behavior, even when the individual knows there are negative consequences to its continuance. Many times, drug or alcohol dependence is silent and unacknowledged for years'.[1] Addicts become good at lying to family, friends and co-workers; especially as an addiction progresses. The sense of shame that comes with addiction inevitably sets up an addict to lie to themselves and their loved ones;[1] in the end, however, the only people they end up deceiving are themselves.

Naturopathic doctors need to remain observant and be aware of some tell-tale signs that their patient may be struggling with a drug or alcohol addiction:[1]

- Mood swings
- Red or glassy eyes
- Social withdrawal
- Changes in behaviour (quick to anger, apathy, missed appointments, irritability)
- Sniffles or runny nose (especially with cocaine abuse)
- Careless grooming
- Changes in sleep pattern
- Changes in schedule

It is important for naturopathic doctors not only to be aware of these outwards signs of addiction, but also to be prepared to discuss addiction with our patients despite the difficulty and discomfort it may cause. Certainly some individuals will have personal experience in dealing with a loved one's addiction; but if you as a practitioner haven't had much experience with addiction, it is important to get comfortable asking the hard questions. These are all factors to consider when addressing addiction with patients.

Let's return to the young female patient mentioned in the introduction. Before making a decision about the potential harm of her 'recreational' ecstasy use, there are many factors to consider. Some questions for the patient:

1. What are the patient's experiences with ecstasy?
2. What is the patient's recovery time? Does the patient's weekend spill over into the week? Has his/her work/career/school been affected?
3. What precautions is the patient taking? Is (s)he also drinking alcohol? Is (s)he hydrating well?
4. Does the patient know the risks of taking 'bad' ecstasy?
5. Is the patient partying with friends or solo?
6. Does the patient's partying lead into anything else that could be problematic, such as risky sexual behaviour?

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If the patient's response to these questions showed their drug use was limited to a 24-hour period, twice a month, then the focus should be on educating her on the safety around using the drug. There have been a number of 'ecstasy'-related deaths during the past few years, and while some of those deaths might have been mitigated by proper hydration, there is no guarantee of the purity of an illicit substance. Therefore, the patient must be made aware of these additional associated risks.

It is important to have an open dialogue with our patients and approach the conversation in a way that does not impart judgment. This enables us to be able to have frank conversation with the patient in the future should she disclose that her behavior/habits have changed or that she has increased her frequency of use of the drug. This is true of any addiction and the same questions should be given careful consideration whenever we are faced with a patient that is struggling with addiction to other substances, including, for example, alcohol or prescription pills. As naturopathic physicians we must always be assessing the harm that a 'habit' is doing.

Let's try a different approach with this same patient. Consider how the patient would react if pushed to seek treatment for her drug use. One possible response could be that she might close off, and deny having a problem; because she feels judged, she might leave your office and not return.

In our first scenario, we create a relationship with our patient, educate her and make her feel she is included in the process. We let her know that we are listening to her, which enables us to gain her trust, helping ensure she will be open to talking in the future. If a practitioner can get a patient to articulate what they themselves need to do, as opposed to the practitioner dictating to them, the success rate for sobriety is higher.[2]

When using this questionnaire to assess a patient with a possible addiction to a substance other than alcohol, simply just substitute the word "drinking" for "substance use" when asking the questions. If your patient answers yes to **1** or more of these questions, further exploration is warranted.[3]

Often, our patients do think they have a problem and have questioned their use or behavior related to a substance before entering our office setting. The proper questioning from a caring professional may be all that is required for them to admit their concerns and disclose the problem. A valuable tool, the CAGE questionnaire, although specific for alcohol use, can be used as a guide for other addictions.[3]

C – Have you ever felt you needed to Cut down on your consumption?

A – Have people Annoyed you by criticizing your drinking?

G – Have you ever-felt bad or Guilty about your drinking?

E – Have you ever had a drink first thing in the morning ("Eye-opener") to calm our nerves? OR Have you ever had to use substances to Ease your withdrawal symptoms.

One of the most useful interviewing styles for assessing and helping our patients with addictions is motivational interviewing (MI). Motivational interviewing is a counseling style based on the following assumptions:[2]

- Ambivalence about substance use (and change) is normal and constitutes an important motivational obstacle in recovery.
- Ambivalence can be resolved by working with the patient's intrinsic motivations and values.
- The alliance between practitioner and patient is a collaborative partnership to which you each bring important expertise.
- An empathic, supportive, yet directive, counseling style provides conditions under which change can occur. (Direct argument and aggressive confrontation may tend to increase patient defensiveness and reduce the likelihood of behavioral change.

Thankfully, as NDs, we tend to spend more time with our patients than other practitioners. The relationship begins in the initial visit – this is the time you start to get the information and ascertain a patient's use of caffeine, tobacco, alcohol, marijuana, and other substances. Often their answers to these questions will be indicative of how truthful/open they are ready to be with their health care provider. Many times when we question the patient about their substance misuse, they will express a desire to quit and list all the positive reasons they should, but an 'ambivalence' is created and a patient can just as easily counter the reasons for quitting by producing all the reasons why they cannot or do not wish to quit.

Let's examine at how this often plays out.

A 40-year-old male patient who smokes marijuana daily is asked about his usage.

He responds "yes" when asked if he has ever thought about cutting down or quitting and provides the following reasons:

• Healthier to quit, will save money, will eat less junk food, home won't stink of smoke.

When asked why he hasn't yet quit if there are clear benefits to doing so, he replies:

• Helps me socialize and sleep, makes me happy and more pleasant to be around, cuts down on my alcohol consumption, doesn't cost THAT much.

Ambivalence is "the coexistence of opposing attitudes or feelings, such as love and hate, toward a substance."[2] It is like a teeter-totter, and all we hope for is that one-day our patient weighs the health benefit more than its social benefits – and if we can keep open communication, then when the opportunity arises we can

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help create change in our patients.[5] By encouraging our patients to vocalize what change can do for them ("**I can change** my habits, **I can quit** smoking pot"), the more likely they are to make that change.[2]

Some key principles of Motivational Interviewing that can be used as a guide to help direct patient visits are:[2]

1. Express empathy through reflective listening.
2. Develop discrepancy between patients' goals or values and their current behavior.
3. Avoid argument and direct confrontation.
4. Adjust to client resistance rather than opposing it directly.
5. Support self-efficacy and optimism.

While all the principles above are useful, it is the author's opinion that identifying the discrepancy between what our patient wants versus their present status can be most effective in eliciting change. A standard 'MI' question often asked first visit is "If you had a magic wand, and could change anything about your health right now – what would it be?"[2] I usually give a patient 3 wishes. This provides an opportunity for the patient to tell me where they would like to be so that we can work on a plan to change these habits using small, measurable goals.

Revisiting our 32-year-old male patient, we must assess whether his consumption of 1-2 beer per night is an alcohol dependency. In order to do this, some further questions should be considered:

1. Do you drink every day? And if so, are there days you drink less, and days you drink more?
2. Does your drinking affect your home life and/or work life?
3. Have any of your family members expressed concern about your drinking?
4. Have you ever felt the need to watch your alcohol intake?
5. Have you ever opened a bottle in the earlier part of the day (or upon waking)?
6. Have you ever felt the need to cut down?
7. What is your diet like? Do you exercise?

The above questions contain the CAGE questionnaire[3] as well as some questions to give you an idea about whether his personal/professional life is being affected. If our 32-year-old patient went to the gym regularly, ate a healthy diet, didn't miss work and truly enjoyed the social and relaxation aspect of his drinking, then we would probably educate him on recognizing the signs of when his habit becomes an addiction. Does he start missing work because he is 'hung over' the next day? Does he start needing a drink to "take the edge off"? We would want to ensure that he was maintaining adequate nutrition through diet and supplementation, and possibly taking liver protecting herbs and nutraceuticals. We should certainly encourage reducing his intake and taking days off from drinking; but if he has been drinking for many years, then taking days off could be dangerous without medical detoxification. In fact, if our patient has never thought of his drinking as being an issue, and if it isn't affecting him or others negatively, until he comes expressing a desire and need to eliminate alcohol, our role is to support him nutritionally and emotionally.

One of the biggest mistakes a clinician can make is confronting and attempting to force treatment on a patient. While there are certainly instances when this is necessary (i.e., suicidal patient, risky use), attempting to force someone into treatment, especially if they do not see themselves as having a problem, could not only not influence their behavior in a detrimental way, but could result in the termination of the therapeutic relationship altogether.

It is important for naturopathic physicians to consider what is our comfort level with respect to addictions. It is even more important that we understand our deficiencies in assessing and treating addiction, so that we can further educate ourselves in this area.

As a starting point for naturopathic doctors wishing to learn more about addictions, consider reviewing the websites of local Alcoholics Anonymous (AA www.aa.org) or Narcotics Anonymous (NA www.na.org) chapters. Al Anon (www.alanon.org) is an organization supporting friends and family of addicts. Smart Recovery (www.smartrecovery.org) is a non-religious/non-spiritual approach to understanding addiction recovery.

While it is not always clear whether a patient is struggling with addiction, a safe approach is always to use non-judgmental language with our patients. Without a doubt, patients grappling with addiction are more likely to open up to a physician who is compassionate and empathetic. It is important to be aware of how our responses might be interpreted when a patient tells us about their health and diet, as a patient might sense that you are being judgmental. And lastly, if you have any doubt in your ability to help your patient, then ask a colleague experienced in this area for guidance. You may also need to consider referring out to another professional. It all comes down to how you as a doctor feel about addiction.

Dr. Gabor Maté (www.drgabormate.com) has written an excellent book entitled, In the Realm of Hungry Ghosts, offering a unique view into the life of a doctor working with addictions in Vancouver's downtown east side. I found the information he gave and the research he has done in this field, incredible. If you are at all uneasy with treating addiction, this is a good place to start your journey.

continues page 22

About the Author

Dr. Aaron Van Gaver, ND works in Clinical Practice out of the Sinclair Wellness Centre (www.sinclairwellnesscentre.com) in downtown Vancouver. Practicing for 10 years, he has focused his practice in working with Mental Health issues such as Addiction and Depression, as well as Hormone Balancing. For the past 7 years, Dr. Van Gaver has held a faculty position at the Boucher Institute of Naturopathic Medicine, and currently teaches Hydrotherapy, and Pharmacology to 2nd and 3rd year students. Dr. Van Gaver currently offers a 10-day outpatient program for patients struggling with Addiction in the Vancouver area. This program consists of IV Amino Acid treatments, NADA Acupuncture, Counselling and Nutritional Support, and works best when done in conjunction with one of the many outpatient programs available in the Lower Mainland. For more additional information about these services, please check out www.draaronvangaver.com, or call 605.629.1120.

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Naturopathic Treatment of Addictions

Dr. Maureen Horne-Paul, B.Comm, ND

It is clear that there is a substantial need in Canada for effective treatment of addictions. According to the Centre for Addictions and Mental Health (CAMH), "one in five people in Ontario in any given year experiences a mental health or addiction problem. About 20 percent of people with a mental disorder have a co-existing substance abuse problem. One in ten Canadians 15 years of age and over report symptoms consistent with alcohol or illicit drug dependence. Over 70 percent of mental health problems have their onset during childhood or adolescence, and young people age 15 to 24 are more likely to experience mental illness and/or substance abuse disorders than other age groups. The economic burden of mental illness and addiction in Ontario (Canada) is estimated at $39 billion annually."[1]

Defined, addiction refers to "the continued use of any psychoactive drug, or the repetition of a behaviour despite adverse consequences, or a neurological impairment leading to such behaviours."[2] "Classic hallmarks of addiction include impaired control over substances or behaviour(s), preoccupation with substance or behaviour(s), use despite consequences, and denial".[3] "Habits and patterns associated with addiction are typically characterized by immediate gratification (short-term reward), coupled with delayed deleterious effects (long-term costs)".[3]

The consequences of addiction impact not only the sufferer, but also extend to family members, the workplace, and our healthcare system. To date, the conventional allopathic medical system has not yet developed a substantially effective, long-term, restorative treatment method to assist individuals in recovering from substance abuse. Current methods include harm reduction, 12-step programs, in-patient and out-patient treatment centres, counselling, and pharmaceutical treatment. However, it is clear that none of these methods alone provides clients with long-term, healthy sobriety, as on average only 25% of the people who use these methods recover and are able to maintain long-term sobriety without relapse.[4]

Additionally, societal misconceptions and myths regarding substance abuse (see below) can significantly hamper treatment and recovery. These myths are perpetuated by counsellors and physicians and have become the cornerstone belief of almost all traditional approaches, often being used to justify the failure of treatment strategies that have been used for over 50 years.[6]

Myth 1: Compulsive substance use is a sign of lack of will power, or of an underlying moral or spiritual problem.

Myth 2: Drugs and alcohol are the causes of substance abuse.

Myth 3: Chronic substance users are "victims" of a disease that can be treated as we treat other diseases with prescription drugs.

Myth 4: Once an individual has successfully stopped using drugs or alcohol, they must engage in a constant struggle not to relapse.

From a naturopathic viewpoint, conventional methods of addiction treatment do not address the root cause of disease. Whereas individuals do recover and remain in recovery using some or all of the conventional methods, there is no doubt that there is a major gap in all of these treatment methods. A naturopathic approach to treating addictions must bear in mind the principle of 'tolle causum' and development of a naturopathic treatment plan for individuals with addiction must adhere to the principle of 'treating the root cause'. As the cause(s) of addiction varies between individuals, as naturopathic doctors, we must strive to treat as many of the underlying causative factors as we can determine and that are amenable to modification.

Naturopathic Approach to Addiction

Undeniably, genetics, lifestyle, upbringing, family life, stress, nutrition, and other factors will influence the health and wellness of a patient. Whereas numerous theories surrounding addiction exist, it is essentially the result of a complex interplay between genetics, life stressors and ultimately, an imbalance in the biochemistry of the brain and neurotransmitter production at a cellular and molecular level. These changes can occur as a result of the influence of several factors including poor nutrition, exposure to toxins, stress, inherited genetic vulnerabilities, and stress in its various forms. These factors can result in imbalances that regulate mood, impulsivity, reward, control, and other emotions and feelings that we experience.

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Neurotransmitter Balance and its Role in Addiction

Under normal, healthy conditions, the brain is able to cope with the usual demands for the production of necessary chemical factors and neurotransmitters. However, stress, fatigue, trauma, or increased demand over prolonged periods of time increases the demand on tissue reserves. Unless cofactors, amino acids, vitamins, minerals, and other nutrients required to maintain and compensate for this increased demand are replaced, a deficiency may eventually develop, and manifest as symptoms of disease.

There are four main neurotransmitters involved in regulating and balancing mood, emotions, feelings, appetite, cravings, and overall feelings of well-being. These are serotonin, endorphins and enkephalins, γ-aminobutyric acid (GABA), and the catecholamines dopamine, epinephrine and norepinephrine (also termed adrenaline and noradrenaline respectively).

Serotonin functions mainly as an inhibitory neurotransmitter, and regulates mood, sleep, emesis, sexuality, and appetite, and has been implicated in neuropsychiatric disorders such as depression, migraine, bipolar disorder, and anxiety.

Endorphins and enkephalins are primarily inhibitory neurotransmitters that are overproduced in the brain, spinal cord, and gastrointestinal tract in response to pain and physical exertion. They inhibit pain signals sent from other areas of the body and allow for the continuation of activities in the presence of inflammation or injury that may otherwise be disabling.

GABA is also an inhibitory neurotransmitter associated with mental relaxation. It is instrumental in calming racing thoughts that can interfere with sleep, and plays a role in preventing seizures, anxiety and panic attacks.[6]

The catecholamines dopamine, epinephrine, and norepinephrine are excitatory in nature. Dopamine regulates short-term bursts of intense concentration, feelings of euphoria, and assists in producing behaviours that need to rapidly shift to accommodate changing circumstances. Epinephrine and norepinephrine help to sustain and maintain alertness, awareness, and vigilance. During situations of prolonged stress, the stores of catecholamines can easily and quickly be depleted, resulting in fatigue, lack of motivation, difficulty concentrating, and trouble experiencing pleasure.

All of these neurotransmitters are manufactured in the neurons in the brain and other areas using certain amino acids as their primary building blocks. Specifically, serotonin is synthesized from tryptophan; dopamine, epinephrine and norepinephrine from tyrosine; GABA from glutamine via glutamate with P5P; and endorphins/enkephalins from DL-(or D-)phenylalanine. A more complete understanding of the biochemistry of neurotransmitter synthesis helps support the inclusion of naturopathic medical treatments that promote their synthesis into therapies for addiction.

Basics of a Naturopathic Treatment protocol for the Addicted Patient

Assessment

1) Adequate full history including a comprehensive mental emotional history specifically including a time line of substance use/abuse including family members, going as far back as the patient can remember; trauma history

2) Physical exam as appropriate

3) Lab work: CBC with differential, FBS, HbA1c, Ca, P, Mg, RBC folate, RBC Zn, ferritin, TSH, fT3, fT4, rT3 if possible, LFT including AST, ALT, uric acid, 25OH Vit D, K, and Na; any other labs indicated

4) Dietary analysis-specific focus on protein intake, sugar, caffeine, additives and preservatives that can interfere with brain function, foods that can affect thyroid function, EFA intake, vit C foods (or lack thereof), refined white flour starches and other refined foods

5) Any clinical reports, rehab history or other related reports from other practitioners

Other helpful Assessment Tools

1) ION profile or Comprehensive Organix Profile (Metametrix) to assess amino acids, organic acids, fatty acids, vitamins, and minerals.

2) Drug taking confidence questionnaire: www.camh.ca has many to choose from and other tools to assist you in determining the nature and extent of the addiction

3) Homeopathic case (not necessary but can be helpful)

Treatment Pillars

Dietary modification, basic supplementation, amino acids, and acupuncture (in the authors' opinion) are the best place to start in treating any addictions as these therapies likely will have the most dramatic effect both short and long-term.

1) Dietary modification:

No less than 3 meals a day containing

- Protein 25-30g/meal: eggs, fish, chicken, dairy products, high quality protein powders (containing at least all of the essential amino acids)
- Low CHO vegetables (at least 4 cups a day, at least 2 cups of greens every day)
- Fats: butter, coconut oil, olive oil, nuts and seeds, avocado
- Other complex CHO foods such as fruits, beans, potatoes, yams, grains (if tolerated)
- Pure water, at least 2 litres a day

2) Basic Supplements:

- High quality multivitamin/mineral
- Vitamin C at least 2g daily divided doses

3) Amino Acids as indicated (based on testing and clinical presentation)

- Refer to the amino acid chart below to assist in determining which amino acids are required, in what order, and at what dosage
- gradually increase the dosing and frequency according to individual patient need and response or lack thereof

4) Acupuncture-suggested points (7) in the acute phase of recovery (as indicated by TCM diagnosis)

a) Alcohol: ear points - brain, Liver, Ki, SP, ST; ST 6/8/36/37, GV20/26, GB 20/34, Bl 10/54, LI 4/11/18, Lu7, SP1/4/6, Ki 1/3/7, CV 4/22, Ht7

b) Tobacco: ear points - lower jaw, Shenmen, Lung, Pharynx, Abdomen, Ki, Internal nose; H7, CV6, GV24, Lu4/5/7, LI4/13, ST36/40, SP8

c) Drugs (non-specific): ear points - brain, lung, liver, abdomen; LI4, ST8/15/25/40, CV10/12/13/14, GV20, GB20/34, Ki1, CV6/7, SP1/6, H7, Lu7, TH5/6

5) Far infrared sauna is helpful for relaxation and initiating detoxification if patient can tolerate it; start at 42C for 10 minutes and work up gradually in temperature and time (max. 55C 1 hour session)

6) Massage therapy can also be helpful for inducing relaxation and a parasympathetic response

7) Hydrotherapy: gentle constitutional treatments can be helpful in detoxification if indicated and tolerated by patient

8) Intravenous therapy: there are several very specific naturopathic IV protocols available that are helpful for patients suffering from acute or chronic withdrawal symptoms. Protocols are specific for alcohol, benzodiazepines, and opiates (see IVT course notes, Dr. V. Osborne 2010, 2011, 2012) and can also help to improve a patient's nutritional status.

9) Counselling, psychological and/or psychiatric care as indicated; Emotional Freedom Technique (EFT), Eye Movement Desensitization and Reprocessing (EMDR), any other technique that will help to reveal and heal the emotional drivers of the addiction; Tai Chi, Reiki, yoga, and meditation techniques can also prove invaluable.

10) Any detoxification strategies or protocols that are indicated using botanicals, nutraceuticals, and homeopathics.

- Be mindful that patients in acute withdrawal can become very ill very quickly, even as a result of minor changes in nutritional supplementation or medication alterations
- Gentle detoxification may be appropriate but it will be a clinical decision based on the patient's presentation, their stage of recovery, and level of toxicity
- Changes in any pharmaceutical medications need to be made in conjunction with the prescribing medical doctor

11) Alcoholics Anonymous (AA), Narcotics Anonymous (NA), Cocaine Anonymous (CA), Adult Children of Alcoholics (ACOA) and other support groups can be invaluable as part of a comprehensive addiction recovery program; support for family members is important as well and Al-Alon can be useful for families and friends of the addicted person.

Cautions and Additional Considerations

Concomitant conditions: Many other conditions can commonly occur in conjunction with addiction and need to be taken into consideration when developing a holistic treatment plan.

1) Thyroid, especially hypothyroidism

2) Adrenal fatigue

3) Hypoglycemia

4) Other mood disorders or psychiatric conditions including anxiety, depression, self-image and self-esteem issues and bipolar disorder.

5) Other accompanying medical conditions needing concurrent treatment[8]

Avoid or be cautious when using amino acids with these conditions:

1) Hypertension: tyrosine, GABA, DLPA

2) Hypotension: GABA

3) Migraine: tyrosine, L-phenylalanine, DLPA

4) Manic depression or bipolar disorder: L-phenylalanine, tyrosine, DLPA, glutamine

5) Severe depression: melatonin

6) Asthma: tryptophan, 5HTP, melatonin

7) Carcinoid tumor: tryptophan, 5HTP

8) Hyperthyroidism: tyrosine, phenylalanine, DLPA

9) Hypercortisolemia: 5HTP

10) Melanoma: tyrosine, L-phenylalanine, DLPA[9]

Relative contraindications for amino acid therapy:

1) Patient reacts to supplements, foods, medications negatively in any way
2) Serious physical illness particularly cancer
3) Severe liver or kidney disease
4) Gastric ulcer (amino acids are slightly acidic)
5) Pregnancy/nursing
6) Schizophrenia or other mental illness
7) PKU (phenylketonuria)
8) MAO inhibitors or SSRIs (more than one)
9) In 50% of bipolar patients, glutamine can trigger mania; in some patients low doses can relieve bipolar depression without triggering mania
10) Additionally, SAMe, St John's Wort, excess amounts of flax or fish oil, and bright therapeutic lights can also trigger mania[10]
    - If patients experience any discomfort with any of the amino acids, they should be stopped immediately
    - A dose of 2g of vitamin C can be given immediately upon any reaction with an amino to help counteract its action

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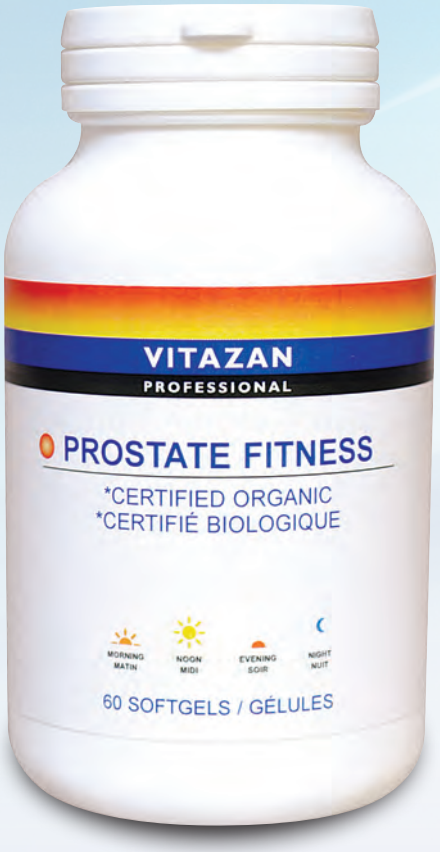

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Manganese (from manganese citrate)	2.79 mg
Copper (from copper citrate)	930 mcg
Vitamin B_1 (thiamin hydrochloride)	4.65 mg
Vitamin K_2 (menaquinone-4)	83 mcg
Vitamin K_2 (menaquinone-7)	10 mcg
Horsetail herb extract (7% silica)	30 mg
Vitamin D_3 (cholecalciferol)	1000 IU
Vitamin C (ascorbic acid)	186 mg
L-Lysine	300 mg
L-Proline	300 mg
Glucosamine sulfate (sodium-free)	252 mg
Vitamin B_{12} (methylcobalamin)	150 mcg
Folic acid (as folate)	500 mcg
Curcumin (95% curcuminoids)	20 mg
Lutein (providing 100% pure value)	2 mg
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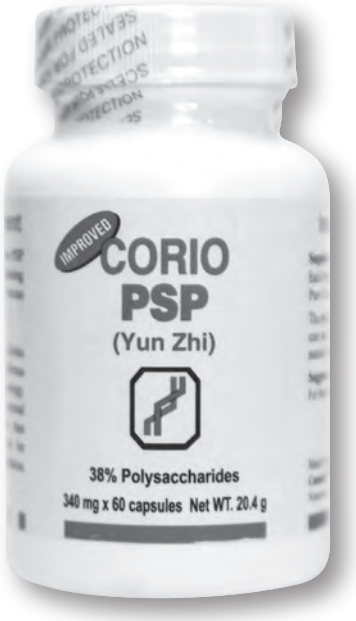

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TABLE 1 – Amino Acid Therapy for Reversing Neurotransmitter Depletion

With thanks to Julia Ross, author of *The Mood Cure* in conjunction with the Alliance for Addiction Solutions.[11]

COLUMN A	COLUMN B	COLUMN C	COLUMN D
Low Serotonin __Afternoon or evening cravings __Negativity, depression __Winter blues, SAD, worry, anxiety __Low self-esteem, guilt, irritability __Perfectionistic __Obsessive thoughts or behaviours __Rage __Panic attacks, phobias, fear of heights, etc. __Hyperactivity __Suicidal thoughts/feelings __Dislike hot weather __Headache, TMJ, FM __Nervous stomach, other GI problems __Insomnia, disturbed sleep __Night owl, hard to get to sleep **TOTAL__**	**Substances That Relieve Sx** Antidepressants__ Pot__ Ecstasy__ Prozac, Zoloft, other SSRIs__ Trazadone__ Sweets and starches__ Other sleep medications__	**Amino Acid solutions** **5HTP** 50-200mg Mid-PM, eve by 10pm, or L-Trypto-phan 500-2000mg mid PM, eve by 10pm. *eve doses needed if sleep is a problem or if symptoms persist into eve or are very severe) **Melatonin** 0.5-5mg or more as tolerated if 5HTP or Trp don't suffice	**Serotonin Sufficiency** Emotional stability Self confidence Positive outlook Emotional flexibility Sense of humour **Melatonin** (which is normally converted from serotonin); 8 hours of deep, restful sleep
Low Catecholamines __Apathetic, depression __Lack of energy __Lack of drive, motivation __Easily bored __Lack of focus/concentration __Thrill seeker __ADD **TOTAL__**	**Stimulants** Cocaine__ Meth__ Caffeine__ Alcohol__ Tobacco__ Opiates__ Wellbutrin__ Ritalin__ Adderall __ Pot__	**L-tyrosine** 500-2000mg AM, mid AM, mid PM by 3pm if sleep is a problem or L-phenylalanine 500-2000mg AM, mid AM, mid PM	**Catecholamine sufficiency:** Alertness Energy Focus Drive Enthusiasm
Low GABA __Stiff tense or painful muscles __Overstressed and burned out __Unable to relax/loosen up/sleep __Often feels easily overwhelmed __Hard to get to sleep **TOTAL__**	**Tranquillizers** Alcohol__ Pot__ Benzodiazepines __ Tobacco __	**Amino Acid Solutions** **GABA:** 100-500mg qid; more during benzodiazepine and ETOH detox as needed. Add Taurine and Glycine 500mg each or more prn as indicated.	**Neurotransmitter sufficiency** **GABA**: Calmness Relaxation Stress tolerance
Low Endorphins __Crave comfort, reward __Numbing from drugs, ETOH, __Foods, or behaviours __Very sensitive to emotional or __Physical pain __Cry/tear up easily __History of chronic pain **TOTAL__**	**Opiates** Oxycontin__ Heroin__ ETOH__ Pot__ Chocolate__ Sweets and starches__ Exercise__ Tobacco__ Caffeine__	**DL-phenylalanine** 500-1500mg AM, mid PM by 3pm (Add free form amino blend 700mg tid before meals)	**Endorphin Sufficiency:** Pain relief (physical And emotional) Pleasure Reward Loving feelings Numbness
Brain Hypoglycemia __Cravings for sugar, starch or Sweets __ETOH __Irritable __Shaky __Headaches, especially if going too long between meals **TOTAL__**	**Blood sugar raisers** Any carbs__ Alcohol__ Caffeine __	**L-glutamine** 500-1500mg AM, mid AM, mid PM. Add Cr 200-300mcg tid prn.	**Fuel sources for brain cells:** Sense of stability Groundedness Blood sugar balance

Patients must be closely monitored for symptoms of withdrawal from their addictive substance, as well as for improvements. Changes in amino acid dosing regimes will be necessary as improvements are seen.

The tables in this article are available for download from the Member's Only site at cand.ca

TABLE 2[12, 13] – Withdrawal Symptoms

Mild to moderate (mental)	Mild to moderate (physical)	Severe
ALCOHOL:		
Feeling of jumpiness or nervousness Feeling of shakiness Anxiety Irritability or easily excited Emotional volatility, rapid emotional changes Depression Fatigue Difficulty with thinking clearly	Headache (general/pulsating) Sweating, especially the palms of the hands or the face Nausea and vomiting Loss of appetite Insomnia, sleeping difficulty Paleness Rapid heart rate (palpitations) Eyes, pupils different size (enlarged, dilated pupils) Skin, clammy Abnormal movements Tremor of the hands Involuntary, abnormal movements of the eyelids	A state of confusion and hallucinations (visual, known as delirium tremens or "the DTs") Agitation Fever Convulsions "Black outs" (when the person forgets what happened during the drinking episode)
OPIATES:		
Dysphoria Malaise Cravings Anxiety/Panic Attacks Paranoia Insomnia Dizziness Nausea Depression	Tremors Cramps Muscle and bone pain Chills Perspiration (sweating) Priapism Tachycardia (rapid heartbeat) Itch Restless legs syndrome Flu-like symptoms Rhinitis (runny, inflamed nose) Yawning Sneezing Vomiting Diarrhea Weakness Akathesia (a profoundly uncomfortable feeling of inner restlessness)	Cardiac arrhythmias Strokes Seizures Dehydration Suicide attempts
BENZODIAZEPINES:		
Aches and pains Agitation and restlessness Anxiety, possible terror and panic attacks Blurred vision Chest pain Depersonalization Depression (can be severe), possible suicidal ideation Derealisation (feelings of unreality) Dilated pupils Dizziness Dry mouth Dysphoria Electric shock sensations Elevation in blood pressure Fatigue and weakness Flu-like symptoms Gastrointestinal problems (irritable bowel syndrome) Hearing impairment Headache Hot and cold flushes Hyperosmia Hypertension Hypnagogia (hallucinations) Hypochondriasis Increased sensitivity to touch Increased sensitivity to sound Increased urinary frequency	Indecision Insomnia Impaired concentration Impaired memory and concentration Loss of appetite and weight loss Metallic taste Mild to moderate Aphasia Mood swings Muscular spasms, cramps or fasciculations Nausea and vomiting Nightmares Numbness and tingling Obsessive compulsive disorder Paraesthesia Paranoia Perception that stationary objects are moving Perspiration Photophobia Postural hypotension REM sleep rebound Restless legs syndrome Sounds louder than usual Stiffness Taste and smell disturbances Tachycardia Tinnitus Tremor Visual disturbances	Attempted suicide Catatonia, which may result in death Confusion Convulsions, which may result in death Coma (rare) Delirium tremens Delusions Hyperthermia Homicide ideations Mania Neuroleptic malignant syndrome-like event (rare) Organic brain syndrome Post-traumatic stress disorder Psychosis Self-harm Suicidal ideation Suicide Urges to shout, throw, break things or harm someone Violence

Post-acute-withdrawal syndrome (PAWS), or the terms **post-withdrawal syndrome**, **protracted withdrawal syndrome**, **prolonged withdrawal syndromes** describe a set of persistent impairments that occur after withdrawal from alcohol, opiates, benzodiazepines, antidepressants and other substances. Infants born to mothers who used substances of dependence during pregnancy may also experience a post acute withdrawal syndrome. PAWS affects many aspects of recovery and everyday life, including the ability to keep a job and interact with family and friends. Symptoms occur in over 90% of people withdrawing from a long-term opioid (such as heroin habit), 75% of persons recovering from long-term use of alcohol, methamphetamine, or benzodiazepines and to a lesser degree other psychotropic drugs.[14] PAWS as a result of GABA-agonist (benzodiazepine, barbiturate, ethanol) dependence and opioid dependence can last from a year to several decades, or indefinitely, with the symptoms entering into periods of relative remission between periods of instability. Symptoms include mood swings resembling an affective disorder, anhedonia (the inability to feel pleasure from anything beyond use of the drug), insomnia, extreme drug craving and obsession, anxiety and panic attacks, depression, suicidal ideation and suicide and general cognitive impairment.

Treatment of PAWS from a naturopathic standpoint involves continuation (often in some patients indefinitely) of supplementation with the appropriate amino acids and other nutraceuticals, continued prudence with dietary modifications (with special attention given to adequate protein intake), as well as appropriate counselling or psychiatric care. Many patients will recover completely over time and not require ongoing use of any of the supplements or aminos. Of course, there will also be those patients who, from time to time, will require short courses of aminos to maintain or increase their levels as they cope with the stresses of everyday life.

As naturopathic doctors, we have great opportunity to positively impact the lives of our patients affected by addictions because we are able to offer them a holistic, patient-centred approach that can be individualized and customized for each person. Many patients using these methods, well supervised by their ND as part of their recovery team, can experience long-term, healthy recovery without relapse, and many will be able to wean off their pharmaceutical medications for good. Nutritional approaches to addiction have been utilized for approximately two decades and statistics show a remarkable success rate; with of 5 out of 6 people recovering, both in North America and the UK.[15] When we remember the principle of "tolle causum" especially with regard to addictions, we can have a significant, positive, and long lasting effect on the lives of our patients suffering from addictions.

About the Author

Dr. Maureen Horne-Paul, B.Comm, ND, graduated from CCNM 1995 and received her PT license in 2003. Her main practice focuses are oncology and IV therapy. Maureen currently practices in Prince Edward County, Ontario where she has a family-oriented practice as well as a naturopathic retreat centre and B&B in the summertime. She writes a biweekly health column for the County Weekly News and is a member of the OAND, CAND, and is vice-chair of the Prince Edward County Chamber of Tourism and Commerce. Contact drmaureennd@gmail.com or visit www.saraswatispa.ca / www.drmaureennd.ca.

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Electronic Media Addiction: A Literature Review

By Marie-Jasmine Parsi, ND (cand.), **Kaeli Sweigard,** ND (cand.) and **Sara Ip,** ND (cand.)

Background

Electronic media addiction is an emerging phenomenon that is characterized by excessive use or exposure to a given media. Current research is scientifically testing media dependence of various outlets including the Internet,[1] video games,[2] and cell phones,[3] amongst others. However, defining media addictions has become increasingly difficult, as technological advancement allows us to access the Internet on our cell phones, watch TV on our computers, and participate in interactive multiplayer online games.

The prevalence of media in our daily lives has led to debate on the legitimization of media addictions[4] and how to define it.[5] While there is disagreement on the validity of electronic media addiction, the concept has gained acceptance as a clinically treatable condition[6] and in 2013, the American Psychiatric Association (APA) considered a subset of media dependency, Internet Gaming Disorder, for inclusion in the DSM-V.[7]

Increasing media usage over the last decade[8] has been met with public concern about its overuse and potential negative outcomes ranging from physical to mental health issues.[9] While prevalence rates range across countries, the cases of media addiction are expected to increase as accessibility to media continues to expand. A recent survey measuring Internet and mobile usage amongst Canadians revealed that 80% of the population have home Internet access and spend an average of 17 hours online per week.[10] Screen time rates are even higher in Canadian youth who average 3 hours per day.[11] This high rate of electronic media utilization, in a population at risk for Internet addiction, provides cause for concern.[12]

The impact of increased media usage on health is two-sided, as it is likely a contributing factor to novel behavioral addictions, while also being an important tool for facilitating patient and doctor interaction. Media can play a valuable role in advancing patient engagement, public awareness[13] and health communication.[14] Therefore, it is important to understand media addictions so as to appropriately label pathological behaviors. The following review highlights how media addictions of Internet, video games, and cell phones are defined and measured. In addition, the most common risk factors, outcomes and treatments are identified. This paper aims to provide an overview of media addictions for naturopathic doctors.

Internet addiction

Definition

Internet addiction (IA) has been variously termed "pathological Internet use", "Internet dependence", "problematic Internet use",[1] and "Internet addiction disorder",[15] with many studies simply interchanging the term "addiction" with any of the previous designations.[16] The lack of a standard definition has lead to the inaccuracy and ambiguity that seems to plague much of the research. Estimates of prevalence vary widely internationally, ranging from 0.7% through 18.3%, depending on diagnostic criteria used.[15-25] Notably, the vast majority of studies of problematic Internet use have focused on investigating students, therefore prevalence of IA outside of children, adolescent and young adult populations is not well understood. Due to student necessity of spending time on a computer for activities related to school, it could be suggested that high rates of Internet usage among this population might be a reflection of the realities of the current schooling experience. Though not officially a DSM diagnosis, reports of Internet addiction have existed since the early 1990s, largely based on a behavioral addiction model using criteria such as dysfunctional use, tolerance and withdrawal.[19,21] More recent studies have corroborated the presence of tolerance and withdrawal in pathological Internet use,[1,21,26-29] and, employing an addiction paradigm, cite dependence,[27] preoccupation,[1,27,28] inability to cease,[1,7,27,28] life consequences,[1,7,19,21,30,31] diminished impulse control,[21] and relapse[1] as characteristics of such use.

Pathological Internet use is associated with high comorbidity with other disorders.[7,16,18,21-24,32-36] It is associated with depression, anxiety, or gambling, although direction of causality or the presence of shared risk factors remains uncertain.[16,19] The Internet may well provide a medium for other disorders to be expressed. What remains unclear is the extent to which using the Internet can cause a psychological dependency.[7,19,37,38] The appropriateness of "Internet addiction" as a blanket term is further complicated when one considers that addictive tendencies are sometimes displayed towards particular Internet applications (e.g., chatting on-line or visiting specific sites repeatedly) but not others.[19]

Measures

Dr. Kimberly Young, creator of the Internet Addiction Test (IAT),[26] contends that the practice of online engagement, regardless of content, is an addictive behavior. The IAT is a 20-question survey validated in the United Kingdom, United States, Finland, and South Korea.[16] Many studies, however, use differing cut-offs from IAT outcomes to classify Internet addiction, resulting in sometimes misleading claims as to prevalence and risk. Other widely used assessment tools include the Internet Addiction Proneness Scale for Youth (K-Scale)[22] developed in South Korea, one of the most wired countries around the globe, and the Chen Internet Addiction Scale (CIAS)[39] commonly used in the Far East from where much of the research on IA is originating.[5] The CIAS alleges a specificity of 92.6%,[39] although this cannot be confirmed due to variability amongst screening tool diagnostic criteria. Shapira et al.[40] have proposed the following as appropriate diagnostic criteria for IA based on impulse control disorder assessment: maladaptive preoccupation with Internet use and Internet use that causes clinically significant distress or impairment.

Risk factors

Psychosocial comorbidities have been associated with Internet addiction. In a study of 20 individuals exhibiting pathological Internet use, all patients had at least one lifetime DSM-IV Axis I diagnosis, with bipolar disorder and substance use disorder occurring most frequently.[36]

Duration of time spent online has also been associated with pathological Internet usage. There are conflicting results whether frequency or duration of time spent online is an appropriate measurement in diagnosing IA.[7,21,23,24,26,41] However, one study specifically correlated increased hours spent on online social networking sites with increased psychopathology,[15] and some have associated more hours spent online with higher rates of IA.[7,25]

Some researchers have studied the impact of biological markers on behavioral addictions, drawing from links made with substance abuse.[42-48] fMRI scans of pathological online users versus healthy controls revealed that cue-induced gaming urges in the former group resembled that of substance dependence.[49] In another case-control study, 15 male adolescents diagnosed as Internet-addicted were shown to have significantly reduced lateral orbitofrontal cortex thickness, an area that may play an important role in addiction behavior.[50]

Outcomes

There exists a high comorbidity of pathological Internet use with psychiatric disorders, the most notable of which appear to be substance abuse,[7,16,18,21,32,33] ADHD, [16,21,34,35] anxiety,[16,17,21] and depression.[17,21-23,35] One study from Hong Kong determined that 58.9% of Internet addicts surveyed met the cut-off for depression using the General Health Questionnaire-12 ($p<0.001$).[23] The same study found significantly more pathological Internet users with concomitant insomnia as compared to non-problematic users ($p<0.001$). Health risks related to sleep issues amongst Internet addicts have been noted elsewhere, including increased use of sleep medication, higher prevalence of apnea, bruxism, and lethargy.[16,51-54] Prospective studies would be helpful in determining direction of causality with regard to the relationship between sleep troubles and problematic Internet use.

In a report on Internet addiction, Dr. Kim Young asserts that 29% of patients reported physical symptoms as a result of extensive inactivity associated with increased computer use. Consequences of such behaviour included carpal tunnel syndrome, strained vision, back pain, headaches and weight gain or loss.[54] Irregular eating patterns have been suggested as a contributing factor to said weight changes,[55] with one study finding the likelihood of obesity of students with IA to be 1.840 times that of non-addicted counterparts ($P<0.001$).[56] Other serious, frequently occurring difficulties described include marital/sexual problems resulting from a preference for online sex-related activity such as chat rooms and viewing of pornography; financial troubles due to debt incurred from online gambling, subscription fees and shopping; and work-related issues such as diminished productivity, including dismissal.[53]

Video game addiction

Definition

Video game addiction is a relatively new concept that has gained increasing attention over the last couple of decades. There has been growing concern over the problematic or "addictive" nature of video games since the mid-nineties.[57]

Game play is regarded pathological when it begins to harm the individual's social, occupational, family, school and/or psychological function. However, there is no standardized clinical diagnosis to date that has been validated for measuring video game addiction. It is often categorized as a "behavioral addiction",[58] in which there is no chemical dependence yet it can stimulate emotional responses and dopamine release.[59,60] While there is still much debate on how best to define behavioral addictions,[32,61,62] many theorists employ the term when referring to problem video game use.

Some researchers have criticized the notion of video game addiction, suggesting the condition has been overestimated due to a lack of standardized definition.[31] Furthermore, it has been argued that it is a 'secondary addiction' (a symptom of a pre-existing problem) resultant of an individual's poor coping abilities and time management skills.[63-66] However, this school of thought does not consider that game play can produce flow states where the player is focused, has a sense of control, and may lose sense of time and place.[67] Certain video game characteristics (promotion of interactivity, creation of alternative realities, feeling of anonymity in socially rewarding situations) can also contribute to addictive behavior.[57,68] Therefore addiction is a complex condition influenced by individual biopsychosocial factors, their actions, and culture,[2,69] as well as the structural characteristics of video games.

Measures

The majority of studies measuring video game addiction used screening tools adapted from the DSM-IV criteria for the assessment of pathological gambling.[70-73] The measure defines video game addiction based on 6 core components of addictive behaviors:

- Salience (the most important activity in the person's life),
- Mood modification (using games to alter mood as a coping strategy),
- Tolerance (increasing amounts of time playing video games to achieve former mood-altering effects),
- Withdrawal (unpleasant physical and psychological states caused by reduced patterns of video game playing),
- Relapse (person's failed attempts to control or reduce patterns of video game playing), and
- Conflict (conflicts between the use of video games and other activities/interpersonal relationships/negative personal emotions like guilt).[74,75]

Questions were scored on Likert-type scales with acceptable reliability and validity (Cronbach's alpha = 0.7). Questionnaires consisted of self-report surveys, suggesting possible social desirability bias and subsequent underestimation of prevalence of addiction rates. Some studies also surveyed children who might not have fully understood the concept of addiction.[76]

The Engagement-Addiction scale, another measure used, was developed by Charlton to differentiate high engagement from video game addiction.[29] Charlton argues that some of the 6 behavioral addiction items (tolerance, euphoria, and cognitive salience) do not apply to video game addiction but rather to non-pathological 'high engagement' game play.[29] He concludes that assessing gaming addiction based on Brown's criterion can lead to overestimation of video game-related addiction.

Prevalence

Rates of addiction varied across studies. Research mainly focused on pre teens and adolescents (ages 8-18 years) observing 2.7%-9% of the studied populations presenting with pathological patterns of playing.[60,72,73,77,78] In studies including older age groups, the rates were similar, ranging from 8-11.9% for pathological playing.[79,80] Firstly, variability in rates of prevalence might be related to the variety of screening tools used to assess addiction across studies. Secondly, national and cultural differences between participant study cohorts could also explain variance in prevalence observed. Differences in norms surrounding video game play and addiction may vary with culture. For example, local area networks for Internet gaming are popular in Singapore.[81] Lastly, age ranges were not consistent between studies and different video game playing habits may be dependent on age.

Risk factors

Several significant risk factors for video game addiction have been observed: younger age,[72,82] male sex,[70,72,77] greater number of hours playing video games,[72] lower social competence, greater impulsivity, poorer emotional regulation,[78] lower self-worth and esteem,[82] high level of escapism during video game play,[83] dysfunctional coping, higher levels of violence acceptance,[77] and the playing of role playing games.[84] There is little evidence for a genetic predisposition to gaming addiction.

Outcomes

The literature has found deleterious outcomes to physical and psychosocial health associated with excessive video game playing. Youths characterized as "pathological" video gamers had significantly higher rates of depression, anxiety, social phobia and poor family relations.[78] Greater psychological distress was also observed,[85] along with increased thoughts of suicide.[77] School performance has been shown to suffer in individuals showing pathological video gaming habits.[73,74,86] Typical physical symptoms associated with high levels of video game play included eyestrain, headaches, epileptic fits, muscle soreness,[70] and pain syndromes associated with repetitive strain injuries.[87,88] It has been shown in some studies that video game playing is also strongly associated with weight gain,[89,90] although this may not be true of activity-promoting video games such as some games using the Wii console.[91]

Video game addiction is also described as a comorbidity in certain mental health conditions. Pathological gamers were found to be twice as likely as non-pathological gamers to have been diagnosed with an attention problem (ADHD).[73] Addiction has been correlated with attention problems.[92]

Cell-phone addiction

Definition

Since their debut in 1983, mobile phones have transformed into multi-functional devices integrated into daily social and work environments.[3] In addition to calling, mobile phones allow access to various media platforms including text messaging, Internet browsing and game playing.[93] The APA has yet to acknowledge a disorder associated with the addictive use of cell phones making problematic mobile use difficult to define and diagnose.[3] The consensus amongst the studies on cell-phone use reviewed suggests mobile phone addiction is a form of technological addiction and subset of behavioral addictions.[3] Prevalence rates of cell phone addiction were limited; however, one review noted rates that varied from 0-38%, depending on the scales used and the populations studied.[94]

Measures

Various measurements have been used to assess the risk factors associated with problematic mobile use.[93,95,96] The most commonly used screening tool is the Mobile Phone Problem Use Scale developed by Bianchi & Phillips.[3] It has been translated across

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different languages (Cronbach ~0.8).[3,93] Vagueness in defining cell phone addiction makes it difficult to generalize the results of the above studies listed.[89]

Risk factors

The strongest risk factors for cell-phone addiction identified included being of a younger age,[3,97] a high self-monitor,[93] extroverts,[93,94] persons with low self-esteem[3,93,94] and depression.[94,95] As expected with utilization of newer technologies, younger cohorts were more likely to own a cell-phone and engage in problematic cell-phone use.[3,97] High self-monitors are described as persons being overly concerned with their public image, making efforts to regulate their behaviors to meet the demands and expectations of their peers.[93] High self-monitors were found to converse more on mobile phones and have more symptoms of problematic mobile use.[93] Persons with low self-esteem were also likely to indulge in problematic mobile use among other addictive behaviors to avoid problematic situations.[3,93] These individuals were more likely to have alcohol, smoking, Internet, gambling and sexual behavior addictions although the etiology of such behaviors remains unclear.[3,93] Individuals who reported feeling lonely were found to converse more on mobile phones although it was not significantly associated with problematic mobile use.[93]

Depression is the most commonly associated symptom of problematic mobile use and is significantly correlated with individuals who have low self-esteem.[94] In addition, adolescents with severe depression were more likely to have 4 or more symptoms of problematic cell phone use (CPU).[95]

Although some studies have observed differences in the rates of calls made between men and women, the relationship between gender and addictive cell-phone use is inconclusive.[3,93,94] Neither culture nor ethnicity conclusively found to be risk factors for problematic use despite different cultural norms concerning mobile phone use, such as increased utilization in Eastern versus Western cultures.[94,97]

Outcomes

There have been many associated health risks due to problematic mobile use. Some countries have banned cell phone use when driving (both texting and the use of handheld devices), though individuals continue to participate in these risky behaviors.[3,95] Additional problems associated with problematic mobile use include: incurring financial debt, mobile bullying, social harassment, disruption to social life and work, depression, anxiety, radiation exposure, oxidative lens stress and shoulder/neck/back pain.[3,93,95] Some problematic mobile users suffer from anxiety when deprived of use but overall mobile use has not been shown to influence anxiety.[95]

Treatment

As of yet, there is no standardized treatment for media addictions due to lack of evidence-based research. Some studies have based psychosocial interventions on those used in substance use disorders with relative success, however unreliable reporting on validity, reliability and statistical significance limit generalizability of findings.[6,16,98] Internet abstinence and counseling programs specifically dealing with IA do exist,[99,100] including the Center for Internet Addiction in Pennsylvania, recently founded by Dr. Young.[101] Individual cognitive-behavioral therapy seems to be the most frequently employed psychosocial intervention thus far.[6,98]

Pharmacological studies are relatively lacking, though there have been a couple of studies examining the effects of methylphenidate in children (mean dose 30.5 +/- 13.3 mg/d, range 18-54 mg/d),[102] and Escitalopram (a selective serotonin reuptake inhibitor) on adults (20mg/d),[103] with IA. In both studies, measures of Internet usage decreased significantly.

Acupuncture treatments have been used for decades for substance use addictions, but results from randomized controlled trials have proved inconsistent.[104] A study by Zhu et al. looked at the effect of electro-acupuncture (EA) on cognitive function in Internet-addicted patients as compared to psycho-intervention (PI) or comprehensive therapy (CT; EA + PI) and found that scores on the IA self-rating scale decreased more significantly in the CT group than with EA or PI alone.[28]

Conclusion

Electronic media have changed our lives fundamentally – from how we socialize to how we work. Despite varying definitions, measures, study designs and populations, the review shows that overuse of electronic media can have multiple negative psychosocial and physiological influences. The most commonly recurring health outcomes of media addictions included depression, anxiety, sleep disturbances, an increased disruption of one's social life, and various musculoskeletal pains. Pathological behaviors were also associated with several mental health disorders such as autism, ADHD and substance abuse. Subjects more likely to develop a media addiction were young (adolescent), male, having a comorbid mental illness, spending more hours playing and having low self-esteem.

There are many challenges facing future research in this field. Without a standardized definition for specific media addictions and criteria for diagnosis, assessing the validity of results across studies is difficult. Definitions of media addiction must take into account demographic and cultural norms on acceptable media use. While problematic use mainly affects younger cohorts, development and persistence of addiction over time remains unknown since there are few longitudinal studies completed on the subject to date. Future research would benefit by including follow-up measures to assess the behaviors of "addicts" after adolescence. It is also important to elucidate whether addictive behaviors can translate into different behavioral or psychosocial problems later in life and whether negative health outcomes are reversible. Media outlets contain many different types of activities that can fuel specific addictions or compulsions (i.e., emailing, socializing, information browsing, gaming), making it difficult to clearly define specific media-related pathologies. In addition, with the growing prevalence of mass media in our lives,

we are surrounded by these technologies, making media pathologies all the more likely and difficult to avoid.

As media addictions become more common, it is essential for naturopathic doctors to be aware of the effects of excessive media use on physical and psychosocial health outcomes. While currently there is no gold standard screening tool for media addictions, NDs would benefit from asking questions during the patient assessment derived from the abovementioned diagnostic measurements based on the components of addictive behavior adapted from the DSM-IV. Severity should be determined mainly by how negatively the activity has affected the patient's daily life. Specifically for IA, NDs can ask patients to complete the IAT's online questionnaire for a baseline measure of addictive Internet use. Efforts to mitigate these pathological behaviors must go beyond individual cases and target community and societal norms regarding media use.

About the Authors

Marie-Jasmine Parsi is currently a 2nd year student of naturopathic medicine at the Canadian College of Naturopathic Medicine (CCNM). Prior to attending CCNM, Marie-Jasmine studied Biomedical Sciences and completed a Master's degree in Physical Rehabilitation at McGill University. Her interests lie in promoting patient education and furthering the development of integrated health care.

Kaeli Sweigard is currently a naturopathic student at the Canadian College of Naturopathic Medicine (CCNM). Prior to attending CCNM, she studied anthropology, cultivating a love of people and culture. With a special interest in mental health and nutrition, she is passionate about the health and wellbeing of Guelph and surrounding area.

Sara Ip is currently a 2nd year student enrolled at the Canadian College of Naturopathic Medicine and is in the process becoming a Naturopathic Doctor. Sara completed her undegraduate studies at McMaster University in Business, and soon thereafter discovered a love for naturopathic medicine. Her interests include: oncology, epigenetics, neurology and nutrition. On her spare time, she enjoys travelling, fitness, yoga, and meeting new people.

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Current Concepts in Food Addiction

Dr. Penny Kendall-Reed, BSc, ND
and **Dr. Stephen C. Reed,** BM, BCh, MA(Oxford), MA(Toronto), FRCSC

We all experience cravings once in a while and we all overeat on occasion. We are generally aware of what we are doing, sometimes regret it afterwards, but generally recognize it as atypical behaviour, something that is not likely to be repeated too often. What would happen, however, if this behaviour became more frequent, if cravings became harder to ignore and overeating became the norm rather than the exception? What would happen if eating became the focus of most activity to the detriment of social functioning and began to cause harm to your health? And what if withdrawal from food caused anxiety, irritability and altered mood? When does craving become addiction?

The past decade has seen the diagnosis of "food addiction" (FA) propelled from lay literature conjecture to the front page of esteemed scientific publications, a move fuelled by research into the alarming rise in obesity. It fell just short of recognition in the DSM-V released earlier this year, but not without substantial lobbying and controversy.

While it is apparent that not all overweight or obese individuals suffer from food addiction, and similarly, not all those with food addiction are obese, an understanding of the mechanisms controlling appetite, craving and addiction is essential for those treating weight-related health issues.

The aim of this article is to give an overview of the current theories behind appetite control, food cravings and food addiction. By detailing the neurophysiology of these conditions, groundwork is laid for an approach to diagnosis and treatment.

Appetite, satiety and craving

The innumerable interactions within the brain and between the brain and the body systems associated with nutrition and metabolism continue to unfold, creating an ever more complex web of feedback loops and neuroendocrine control mechanisms. However, it would appear that there are two separate but interlinked systems that contribute to the control of food intake. The first is a ***homeostatic*** system that integrates metabolic needs with food-related satiety messages. The second is a ***reward*** pathway that mediates the hedonistic value of food along with motivational behaviour and neocortical rationality.

The ***homeostatic system*** within the hypothalamus (arcuate nucleus, lateral hypothalamus and paraventricular nuclei) incorporates hunger and satiety centres that direct us toward feeding or resting behaviour. The predominant primitive drive is to eat, a message mediated by neuropeptide-Y (NPY) and agouti-related peptide (AGRP) from the arcuate nucleus (ARC). This message is turned off by a number of satiety messengers that provide peripheral feedback from ingested food and metabolic state plus neural control within the brain. The most important peripheral endocrine messengers are insulin and leptin, which provide satiety signals based on both the consumption of food and the status of the body's energy supplies, most notably, fat stores. Other messengers include glucose, ghrelin (promoting hunger), PYY, CCK and GLP, which all act on the hypothalamus to control the balance between hunger and satiety.[1,2]

The ***reward system*** is more complex and incorporates a larger number of brain areas and pathways. Humans (and other higher functioning animals) can eat or starve independently of basic hunger-satiety messaging.[3] We act on situational, emotional and hedonistic cues, allowing such activity as ordering chocolate dessert despite feeling full from previous courses. The ventral tegmental area is the initial site of activation through opioid, nicotinic and cannabinoid receptors, releasing dopamine through the mesolimbic dopamine system (MDS) to the nucleus accumbens (NA) and limbic system. This is the initial key pathway involved in reward (and addiction), its primary role being the promotion of motivational behaviour in response to reward-predicting stimuli. While essential in mediating the acutely rewarding value of drugs or food, it has a secondary, longer-term effect to promote need or craving. These so-called "wanting incentives" or "incentive salience" are inherent to most animals.[4-7]

The nucleus accumbens (NA) receives input from the MDS, limbic system, amygdala (AMY), pre-frontal cortex (PFC) and hippocampus, allowing it a central role in the mediation of reward-based behaviour. In humans, the ability of certain drugs (such as cocaine, alcohol and nicotine) to release dopamine (DA) in the nucleus accumbens leads to "artificial reward" and hence addiction.

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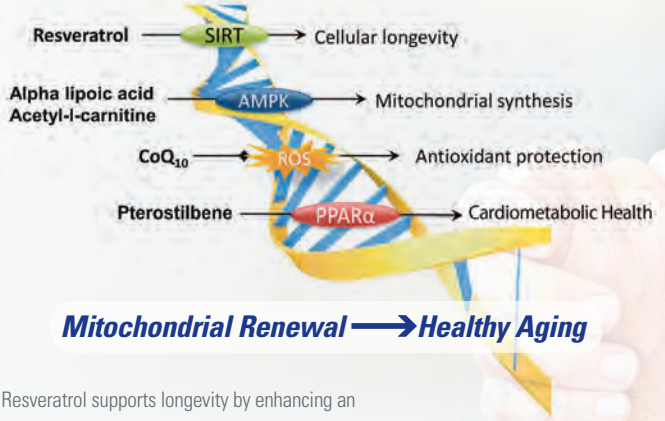

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Similar neurochemical stimulation is seen with highly processed sugary or fatty foods and provides the basis for some aspects of food addiction.

The amygdala (AMY) is a primary processing area and receives information about the perceived metabolic "value" of food as well as its hedonistic or pleasurable value such as taste.[4] It mediates the addictive properties of cocaine via dopamine D-1 receptors, and alcohol via GABA and opiate receptors.

Thus, while the MDS appears central to addiction, other dopamine-independent pathways act via numerous circuits to provide reinforcement and reward-seeking behaviour.

Craving was originally thought to be simply one end of the homeostatic spectrum, an extreme version of hunger resulting from food deprivation. This "restrictive theory" has empirical support[5] yet fails to explain craving triggers in humans such as emotion, boredom and stress. It is this strong emotional element of craving that makes it a far more complex aspect of hunger and satiety than the neuro-hormonal feedback implicated by simple physiologic homeostasis.[8] It is now believed that the mesolimbic dopamine system, which is intricately involved in addiction, is the one of the key subconscious triggers for craving and its attendant behavioural sequelae. Serotonin also exerts a strong influence on craving although this relates primarily to the emotional control of eating rather than the reward system. Certain foods, including chocolate and other sweets, increase serotonin in the brain and become desirable in low-serotonin states such as depressed mood, pre-period and during the initial stages of dieting.

Dopamine and addiction

The past ten years have seen a tremendous amount of research into the role DA plays in addiction. While certain drugs (such as cocaine) have the ability to acutely and dramatically raise dopamine activity in the limbic system with resultant euphoric psychomotor effects, this effect is not noted with all addictive substances (such as alcohol). It now appears that the MDS has a central and profound influence over the way we react and respond to stimuli that we consider ***potentially rewarding***. Under the influence of numerous factors, including the intake of "addictive" or rewarding substances, or indeed rewarding activity (gambling for example), the MDS undergoes neurochemical remodelling. This reorganization of reward and memory circuits leads to a heightening of what is termed "incentive salience".[9] This is the emotional "wanting" attribute given by the brain to stimuli that predict reward. So, for someone addicted to alcohol, the mere visual stimulus of a bottle or glass will increase DA activity, create craving and drive behaviour toward consumption. Similarly, it would be the sight or smell of food, or even a restaurant sign, that would lead to craving and eating. It is this incentive salience that makes withdrawal so difficult as neurochemical pathways are activated by remote stimuli even when trying to avoid the substance itself.[10] The altered MDS makes these cravings impossible to ignore as they are promoted in importance above all other activity. To further exacerbate this response, prolonged MDS dopaminergic activity impairs the normal rational and cognitive control over impulsive behaviour exerted by the PFC. This dissociation of cortical and subcortical systems with resultant impairment of decision-making and avoidance of impulse is a feature that addiction shares with chronic stress, as we shall see later.

The "reward deficiency" theory[11] suggests that in obesity, altered Type-2 dopamine receptor (DA-D2) availability or sensitivity leads to increased demand for dopaminergic stimulation and thus increased intake of rewarding substances. Relative DA deficiency would therefore induce eating behaviour that promises to raise dopaminergic signalling. This would also explain the tendency of individuals to eat palatable food on withdrawal of nicotine (a dopaminergic stimulator) or while taking antipsychotic medication that reduces DA levels.[12] Although intuitive, the picture is likely more complex. One interesting paradox is the fact that DA is known to have two separate effects on the nucleus accumbens (NA) and hypothalamus[13] While increased DA activity in the MDS promotes food-seeking behaviour, it simultaneously exerts a controlling affect on hunger and food consumption by inhibiting NPY, an effect augmented by satiety messengers such as leptin. In addition, the NA receives other dopaminergic inputs from the *dorsal striatum*, a mechanism thought to be involved with survival and the maintenance of adequate calorie intake. Drugs that block DA-D2 receptors cause increased appetite and weight gain while agonists cause anorexia. Although there is argument as to whether this DA-D2 abnormality is primary or secondary, there is certainly a clear association and in some cases a genetic predisposition. A plausible argument is that the MDS exerts its motivational effect on the NA via Type-1 dopamine receptors (DA-D1), while DA-D2 receptors mediate feedback regulation of hunger, eating behaviour and compulsion in order to limit over-consumption.[14,15] Thus, food addiction in obesity would also be considered a failure of adequate feedback in common with other aspects of the condition including leptin and insulin resistance.

Factors promoting food addiction

Stress – Acute and chronic stress have a profound effect on the neurophysiology of subcortical structures. At its simplest, the hypothalamus-pituitary-adrenal (HPA) axis provides a primitive protective response to threatening stimuli, the classic sabre-toothed tiger. However, our modern lifestyles provide an excess of predominantly "perceived" and non-resolving threats, which over-stimulate this system and result in harmful neural and endocrine dysfunction. Adrenal fatigue is well recognized and leads to an impaired response to true physical stressors such as inflammation or infection. Less known but more profound is the development of cortisol resistance leading to disruption of feedback within the HPA system. Subsequent increased corticotropin releasing hormone (CRH) production in the hypothalamus and associated changes within subcortical structures including the amygdala have significant effects on a number of systems including the hunger-satiety centre and the reward pathway. Acute stress and cortisol increase DA release in the NA,[16,17] which as we have seen, leads to reward-driven behaviour. Stress and anxiety further sensitize the MDS and

amygdala to promote this behaviour while simultaneously causing atrophy in the hippocampus and PFC resulting in even less control over impulsive behaviour and craving.[16]

Thus, acute stress will promote craving, seeking and eating of highly palatable sweet or fatty foods in order to satisfy and reset the pathway. Chronic stress will enhance this pathway and reduce cognitive control leading to ever-escalating desire and ingestion of calorific foods, weight gain and metabolic syndrome.

Further interesting research has found that chronic stress reduces DA activity[18] and alters the DA-D2 receptor making it less sensitive to DA. This receptor co-exists with the ghrelin receptor (GHSR-1), which, as we know, is responsible for intense feelings of hunger. As the DA-D2 receptor modulates the effect of ghrelin, any decreased efficacy will only heighten craving in food addiction, obesity and stress.

Food content – Processed, highly calorific foods rich in fats and sugars affect both the homeostatic mechanisms that normally control food intake and the hedonistic reward pathways that influence whether we eat for pleasure. As a result of an effect called "epigenetics", environmental factors can profoundly influence gene expression and thus physiologic function.[19] Diets high in saturated fats increase the production of and sensitivity to NPY, AGRP and the orexins, our neural hunger messengers while inhibiting the satiety messenger alpha-MSH. In addition they reduce sensitivity to peripheral messengers such as insulin, leptin and CCK. Palatable foods stimulate the reward system through a number of pathways by increasing levels of endogenous opioids and stimulation of "pleasure" receptors such as GABA and benzodiazepine. Increased activity in cannabinoid receptors also occurs and promotes hunger. Through these various mechanisms palatable foods increase dopamine production and adjust the neural "set-point" such that any reduction in DA levels leads to anxiety and craving. The role of our food environment cannot be understated. The heavy marketing of "fast" and processed foods, the inclusion of high-fructose corn syrup and the relative cheapness of poor quality "addictive" foods is considered by some as dangerous as having cocaine samples available at your local supermarket.

Emotion and Serotonin – Adequate serotonin promotes a sense of calm, security, relaxation and confidence, while low levels are associated with depressed mood, anxiety and irritability. Serotonin is widely considered to be the "anti-dopamine", diminishing reward-seeking behaviour and craving thus explaining why reduced levels in PMS, depression and dieting lead to a desire for sweet or starchy foods that temporarily increase serotonin levels.[20,21] This in itself can lead to overeating and weight gain. The interaction with obesity and food addiction is obviously multifaceted but it is clear that factors resulting in impaired serotonin production or sensitivity will serve to promote activity in the dopaminergic reward pathway and exacerbate craving along with food-directed behaviour.

Genetics – There appears to be some evidence of a genetic susceptibility to obesity and food addiction that may predispose an individual to these conditions, but in the absence of other emotional and environmental factors is not sufficient in most cases to be a cause. These include reduced DA-D2 receptor numbers and sensitivity along with enhanced activation of brain areas involved with processing food palatability.[19,22]

Diagnosis of Food Addiction

Understanding the neurophysiologic basis of food addiction helps direct us towards an accurate diagnosis, something of utmost importance when planning treatment. However, we need to distinguish food addiction from other conditions, particularly eating disorders such as binge eating and bulimia and simple overeating.[23-25,27]

The American Society of Addiction Medicine (ASAM) gives the following short definition of addiction:

"Addiction is a primary, chronic disease of brain reward, motivation, memory and related circuitry. Dysfunction in these circuits leads to characteristic biological, psychological, social and spiritual manifestations. This is reflected in an individual pathologically pursuing reward and/or relief by substance use and other behaviors.

Addiction is characterized by inability to consistently abstain, impairment in behavioral control, craving, diminished recognition of significant problems with one's behaviors and interpersonal relationships, and a dysfunctional emotional response. Like other chronic diseases, addiction often involves cycles of relapse and remission. Without treatment or engagement in recovery activities, addiction is progressive and can result in disability or premature death."

Our understanding of food addiction certainly fits this picture yet there is on-going discussion as to whether physiologic and diagnostic criteria for addiction to recognized substances of abuse can be applied to food. Although perhaps less acutely toxic than, for example, cocaine, the neurochemical pathways involved and dysfunctional outcomes resulting from the ingestion of sweet, fatty and processed foods are identical (Table 1).

TABLE 1
Neurochemical changes during the 3 stages of addiction

1. **Binge/Intoxication**	• MDS/Opioid pathway stimulation • Stimulus-response pathways engaged
2. **Withdrawal/ Negative Affect**	• Decreased MDA stimulation, decreased serotonin. • Decreased mood, activity and motivation for other tasks • Increased activity of HPA axis – anxiety
3. **Preoccupation/ Anticipation (Craving)**	• Reorganization of reward/memory circuits • Impaired PFC + Amygdala dysfunction – loss of control • HPA/CRF sensitivity

Binge eating disorder is classified in the DSM-V and includes a number of diagnostic criteria that show significant overlap with the features of food addiction.

Gearhardt et al[23] argue convincingly that food addiction fits into the parameters of the DSM-IV Substance Dependence category, arguing that the legality and availability of food makes some of the social aspects of the condition less obvious than might be found with drugs. For the practising clinician, however, there are some important differences, which may allow us to differentiate between binge eating disorder (BED) and food addiction. Firstly, in BED, there is little craving or compulsion to eat on a regular basis and individuals frequently have negative feelings toward food. This contrasts to the craving and overeating associated with FA and the need for high fat or sugary foods to feel normal. BED is considered an "expressive disorder" associated with underlying emotional or psychological dysfunction, something not found in FA. In FA, triggers to eat are primarily food based rather than emotional, although, as we have seen, emotional input can influence the development or progression of food addiction. Withdrawal occurs in some, but not all subjects with FA, leading to heightened HPA activity when unable to satisfy their food triggers. This is not seen in BED. Finally, although counterintuitive, the incentive salience associated with FA results in far less control of food intake than BED. Another useful and validated diagnostic tool, which incorporates a number of physical and emotional factors along with a food diary, is the ***Yale Food Addiction Scale.***[26]

Treatment for food addiction

The National Institute on Drug Abuse (NIDA) advises a four-step program for addiction; detoxification, medication, behavioural therapy and relapse prevention. The aim is obviously to remove intake and dependence on a substance entirely. While the steps involved in the treatment of food addiction may be similar, the goal is the avoidance of *certain* foods rather than *all* foods. In addition, the prevention of relapse may be more difficult due to the omnipresence of triggering stimuli, a factor easier to control in drug or alcohol therapy.

My approach changes the order of the above steps. Initial detoxification, which, in drug/alcohol abuse is often completed under controlled and monitored conditions, is impractical and likely to cause undue stress making success less likely. I focus on resetting hormonal and neurochemical imbalance first, which allows a less traumatic transition to detoxification.

Step 1 – Rebalancing

Akin to stage 2, "medication", the rebalancing of hormonal and neurochemical systems is multi-modal, including changes to diet, behavioural modification and supplements. There are four primary aims; restoring satiety feedback, controlling HPA overactivity, modifying dopamine reactivity and increasing serotonin.

Satiety feedback incorporates a number of messengers but the predominant dysfunction involves leptin and insulin resistance. Depending on the individual and degree of addiction I may not enforce a strict diet with withdrawal of all addictive foods initially. However, I will recommend incorporating adequate protein at each meal and encouraging only three meals per day without snacking in order to reset insulin and leptin satiety signalling. I use L-Carnosine as a supplement to improve leptin sensitivity.

Due to its adverse effects on metabolism, satiety feedback and reward system dysfunction, correction of HPA axis overactivity is a key factor in the management of obesity and food addiction. I have found Serenitin-Plus, a casein decapeptide from milk known as *lactium*, to reliably reduce CRH/cortisol levels and re-establish feedback within the HPA system. I also address sleep disturbance and recommend lifestyle therapies such as meditation, massage therapy, exercise and music or laughter therapy.

Increasing dopamine levels through diet or supplementation is popular in the management of food addiction but is simplistic and ignores much of the newer research into functioning of the reward pathway. As such this approach may backfire and actually increase craving and food-seeking behaviour. My approach is to concentrate on promoting satiety feedback, rebalancing the HPA axis and increasing serotonin while cautiously increasing dopamine. In addition I would add omega-3 DHA supplementation to *increase* D2 receptor activity and *reduce* D1 activity.[28] The omegas will also help to increase serotonin and control cortisol.

As noted previously, the relative inactivity of the DA-D2 receptor in the D2-GHSR1 complex enhances the hunger-inducing effects of Ghrelin.[29,30] Therefore, minimizing ghrelin production is key. I use protein, fibre and fluids in the diet along with omega-3 supplements, which have been shown to promote D2 function.

Serotonin levels can be increased through a diet rich in tryptophan and with supplementation with 5-HTP. Sun exposure, exercise and laughter are behavioural therapies that can also increase levels.

Step 2 – Detoxification

Detoxification involves primarily the withdrawal of trigger foods and restoration of a more normal healthy diet. In individuals with significant addiction I will delay this stage until rebalancing is complete, while in others I may commence it simultaneously. Detoxification involves both phase 1 and phase 2 naturopathic processes to help the body eliminate the inflammatory products associated with processed foods and environmental toxins. It will also help clear the excess hormones and neurochemical messengers, which have altered brain pathways and contributed to both food addiction and ill health.

Step 3 – Behavioural Therapy

Although food addiction appears to have a relatively well-defined neurochemical basis, there are numerous personal, emotional and environmental factors that make the condition highly individual. As such, the behavioural aspect of treatment can be quite difficult and the involvement of other health professionals is recommended.

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Firstly it is important to create a support system. Enlist people you admire and trust to provide encouragement and support your healthy lifestyle. Exercise is a key component of treatment, providing reward, reducing stress and improving metabolism and satiety. Bodywork including yoga, massage therapy, meditation and music or laughter therapy increases serotonin while reducing cortisol.

Cognitive behavioural therapy (CBT) is a type of psychotherapy that addresses dysfunctional emotions, maladaptive behaviours and cognitive processes through goal-oriented, systematic procedures. Under the care of a qualified practitioner this treatment modality can be central to the management of the psychological and emotional aspects of FA.

Step 4 – Relapse Prevention

Perhaps one of the most difficult aspects in the management of any addictive disorder is the prevention of relapse or the development of "addiction transfer", the shifting from one addiction to another to maintain reward. Success in steps one through three minimizes this likelihood but given the perpetual exposure to triggers in daily life, the risk is substantial.

Removing all triggers within controlled environments such as home and workplace is key. Avoiding situations where triggers might exist is more difficult and needs to be balanced with the value of friendship, social interaction and healthy activity such as exercise. Having a supportive social group is essential and the "buddy system" can be extremely valuable when faced with a trigger crisis. Using techniques learned in CBT or meditation will also provide a defence against relapse.

Food Addiction is rapidly being recognized as playing a major role in the seemingly unrelenting rise in obesity seen in our population. Driven by a combination of factors including, most importantly, chronic stress and the availablity of processed high fat and sugar foods, this compex disorder may represent one of the most significant health issues of the twenty-first century. Understanding its causes and pathology is essential for naturopathic doctors given their pivotal role in the prevention and treatment of nutrition-related disease. By formulating a cohesive approach to diagnosis and treatment they are uniquely positioned to enable a significant and vital impact on the proliferation of food addiction in the coming years.

About the Authors

Dr. Penny Kendall-Reed, BSc, ND is a naturopathic doctor in Toronto. After graduating from McGill University with a B.Sc. in Neurobiology, she Earned a degree in naturopathic medicine from the Canadian College of Naturopathic Medicine, where she received the Dr. Allen Tyler Award for Most Outstanding Clinician. Penny Kendall Reed is the co-author of five national bestselling books including The New Naturopathic Diet, The No Crave Diet, and The Complete Doctor's Stress Solution. Penny Kendall-Reed travels throughout Canada and the United States lecturing on neuro-endocrine related diseases and holds health retreats at various resorts worldwide. She appears regularly on television, magazine and radio across Canada and the United States addressing various health issues, and is a monthly health expert for Health and Wellness Magazine in Toronto. Penny Kendall-Reed has also designed an all natural oral and topical anti-aging skin care line called Age Aware Skin Care sold throughout Europe. Presently, Penny Kendall-Reed is the director of natural therapies at the Urban Wellness Clinic in Toronto.

Dr. Stephen C. Reed, BM, BCh, MA(Oxford), MA(Toronto), FRCSC is a Toronto-trained orthopaedic surgeon and a graduate of Oxford University Medical school. Currently on active staff at the Humber River Hospital in Toronto, he received specialty fellowship surgical training in England, Australia and at the Orthopaedic and Arthritic Hospital. He has Master's degrees from Oxford University and the University of Toronto and has published extensively in the orthopaedic literature. Dr.Reed has co-authored five best-selling books with his wife, naturopathic doctor Penny Kendall-Reed. In addition to an active surgical practise he focuses on health education through articles and lectures.

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APPENDIX 1:
Impact of Food Addiction on Health

Metabolic: obesity, type-2 diabetes, hypercholesterolaemia, hypertension, heart disease.

GI: cholecystitis/cholelithiasis, gastritis, constipation

Emotional: depression, anxiety, sleep disturbance, social dysfunction, poor self-esteem

Nutritive: vitamin/mineral deficiency.

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