BHNewsletter

Behavioral Health Nutrition
a dietetic practice group of the Academy of Nutrition and Dietetics

*BHN: Fuel Your Brain, Feel Your Best!*

# Hormones and Addiction

CPE

**David A. Wiss, MS, RDN, CPT**

## Introduction

It has been known for some time that individuals with substance use disorders (SUDs) have significant vitamin and mineral deficiencies.[1-6] In the past decade, investigators have begun to explore alterations in both neuro-circuitry and nutrition-related hormones (i.e. leptin, ghrelin, insulin) in the SUD population to better understand eating behavior during drug use, recovery, and long-term abstinence. The connection between nutrition behavior and addiction recovery have important implications that are not frequently addressed in clinical practice. According to Virmani et al,[7] drug abuse appears to be a risk factor for metabolic syndrome, which is a cluster of risk factors for cardiovascular disease. Given that weight gain following abstinence from drugs is a source of major personal suffering, there is a pressing need for a more detailed understanding of the effects of drug addiction on dietary intake.[8]

It is well established that drug addicts share many of the same brain imaging[9] and behavioral[10] characteristics as compulsive overeaters. However, since the "reward" or hedonic value associated with food in humans is tied to memory, emotions, and individual characteristics, food perception is difficult to assess at a group level.[11] Meanwhile, evidence supporting the concept of food addiction has become increasingly accepted. The purpose of this review is to examine both drugs of abuse and contemporary palatable food to determine if there is a link addiction between hormones acting on reward-related dopamine pathways that stimulate or inhibit feeding. The majority of neuro-hormonal research in this area has been conducted in animal models, so unless specified as human research, it can be assumed that the physiological observations of the brain are extrapolated from rodents.

The mesolimbic dopamine system is considered a primitive part of the brain that consists of the ventral tegmental area (VTA) which contains dopamine neurons that project to cortico-limbic structures such as the nucleus accumbens (pleasure center), medial prefrontal cortex (cognition), hippocampus (memory), and amygdala (emotional reactivity). The VTA receives direct and indirect input from the hypothalamus, which governs several endocrine processes through communication with various hormone-producing glands. The VTA is involved in somatic processes including body temperature, sleep, and appetite, and influences neurological mechanisms that underlie mood and motivational states. Direct evidence that leptin and ghrelin modulate the hypothalamic pathway has begun to emerge, implying reward-related information that drives feeding behavior at the level of the VTA.

Coll et al[12] have suggested the presence of an integrated system in which adipocyte-derived signals (i.e. leptin) provide long-term information to the brain about the state of nutrient stores, whereas a variety of signals (many not addressed in this article) triggered by eating have important roles in influencing meal initiation and termination. Adam and Epel[13] have highlighted the role of chronic stress and elevated cortisol (a glucocorticoid controlled by the hypothalamus) in the dysregulation of this finely balanced system. This may cause impairment of the satiety signals leading to increased food intake and subsequent visceral fat accumulation. Corticotrophin-releasing factor is released during stress, which stimulates the reward system.[14] Stress has been linked to drug relapse and is a significant cause of failure in dieters, and can actually become a conditioned incentive for food, possibly explaining the phenomenon of "comfort

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# From the Chair

**Sharon Lemons, MS, RDN, CSP, LD**

***Dear Behavioral Health Nutrition members,***

Behavioral Health Nutrition has an exciting year planned for our members. Just a few of the things we have planned are a new website, webinars on all four of our practice areas plus a free webinar, a spotlight session at the Food Nutrition Conference and Expo, and several opportunities for you to be involved.

The website has been redesigned to meet more of our member's needs. This is a work in progress that provides all of you an opportunity to participate. In the members section will eventually be a long list of fact sheets that will provide information on all of our practice areas. To accomplish this we will be needing writers and members who can help research appropriate studies. We encourage our student members to get involved in helping with the research portion of this project. Just image how useful it will be to go to your computer or smart phone and access quick information on that syndrome or condition that just became a part of your caseload. We hope this will become an extremely valuable resource to all of our members.

Those of you who participated in our member survey very clearly told the executive committee you wanted more webinars. As we developed the five year strategic plan in May a plan was put in place to increase webinars gradually. E-blast will be sent out asking for proposals to present a webinar. This will give us a greater breadth of knowledge to be able to offer to you our members. In addition, it will give all of you a chance to send in a proposal to share your expertise.

October brings the Food Nutrition Conference and Expo in Atlanta, Georgia. Behavioral Health Nutrition's spotlight session, *Dysphagia, Mealtime, and Intellectual and Developmental Disabilities,* will be October 20th at 8 a.m. featuring BHN's own, Joan Medlen, RD with Jennifer Meyer, SLP. This will be a session that addresses dysphagia issues across the lifespan of those with IDD and will include information that anyone who works with dysphagia will find useful.

I am glad to report that this June BHN has started with a full slate of officers. We have an amazing group of professional and student officers. Of special note I'd like to thank Diane Spear for her service as Newsletter Editor. Diane has decided to pass the baton to a new newsletter editor. Those of you who have enjoyed the newsletter for the past several years have benefitted from Diane's work. Diane will be working to provide a smooth transition to our new newsletter editor, Hanna Kelley. I can't say enough about what a fabulous job Diane has done. On behalf of BHN thanks for being such a great editor!

I hope that each of you find BHN member benefits to be especially useful for you this year.

Sharon Lemons, MS, RDN, CSP, LD
*Chair of Behavioral Health Nutrition*

# Thank You

BHN Newsletter Team wishes to thank the following volunteers for contributing to the success of *BHNewsletter* as CPE Expert Reviewers and Newsletter Review Board 2013-2014:

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food."[15] Wiss found that individuals with a history of substance abuse reported more difficulty controlling overeating when depressed.[16]

## Food Addiction

Highly palatable food can stimulate endogenous opioid release[17] and trigger dopamine activity in the brain.[18] Palatable food is processed food that typically contains added sugars, salt, and fat. Recent evidence depicts dopamine circuits as a major site of convergence where metabolic/hormonal and visceral sensory cues interact to regulate eating behavior by way of a "gut-brain dopamine axis."[19] Food addiction has been associated with binge eating disorder as well as obesity.[20] According to Heber and Carpenter,[21] obesity-associated inflammation modulated by leptin in the brain may promote addictive behaviors leading to a self-perpetuating cycle of addiction to food, as well as drugs/alcohol and process addictions such as gambling.

Other researchers have challenged the notion that drug addiction and food addiction are near-identical processes, since there are unique evolutionary contexts across species with different environmental pressures, resulting in significant differences between rodents and humans.[22] Such differences can include food availability, visual appeal, economics and incentives, social routines for eating, alternative reinforcement, and the impact of advertising.[23] Other differences in the prefrontal cortex (which weighs pros and cons) are not sufficiently integrated into the current animal models of food intake. The full behavioral consequences of metabolic hormones acting on dopamine neurons will require further investigation. Meanwhile, several authors have identified leptin as a possible link between overeating and addiction, suggesting that many leptin-deficient individuals meet criteria for food addiction.[14]

## Leptin

Leptin is produced and secreted by adipose tissue to increase metabolic rate. In human populations plasma leptin is positively associated with fat mass.[24] Leptin is described as anorexigenic because it is responsible for initiating the starvation response (decreases food intake). Evidence suggests that leptin-mediated modulation of central dopamine circuits provides a neural pathway by which changes in leptin levels lead to adaptive behavioral responses in feeding.[25] De Araujo et al showed that the reward value of sucrose was increased by fasting, yet decreased by leptin via a reduction in dopamine signaling.[26] In other words, a hungry individual with lower leptin levels will assign a higher reward value of food compared to an individual who is satiated (dopamine release and firing is inhibited in the nucleus accumbens when satiated). Interestingly, high circulating levels of leptin have no pronounced effect on metabolism and feeding, whereas low levels may trigger a physiological condition where the body perceives a hungry state and simultaneously enhances motivation for obtaining food.[27]

The failure of elevated leptin levels to control or reverse obesity suggests the possibility of a leptin-resistant state. High-fat diets can induce leptin resistance and is emerging as a cause and consequence of weight gain.[28] Leptin resistance is considered analogous to the concept of insulin resistance, both of which can correlate to obesity.[28] According to Coll et al,[12] leptin resistance occurs when circulating leptin fails to reach its target receptors in the brain, when leptin receptors have decreased expression, when there is attenuation of the intracellular leptin signaling cascade, or when enzymatic dysfunction exists. Additionally, altered leptin signaling due to genetic mutations have been implicated in obesity.[29] Meanwhile, the concept of leptin resistance remains controversial.

As evidence that leptin regulates the activity of the mesolimbic dopamine system by its actions on VTA dopamine neurons continues to grow, recent data indicates that leptin not only regulates the homeostatic center of the hypothalamus but also the hedonic system by affecting subjective desires for food.[30] Food deprivation decreases circulating leptin levels, which has been used to study the impact of leptin on brain reward centers. While still not fully understood, it is known that leptin has action extending to the brain reward circuits thereby contributing to preference for highly palatable foods. Recent research suggests that the leptin-dopamine interaction appears to be bi-directional, as dopamine has been shown to negatively influence leptin action in the lateral hypothalamus.[31] Data collected by Davis et al indicate that leptin signaling within the lateral hypothalamus regulates energy homeostasis and metabolism, whereas midbrain leptin modulates effort-based responding for food via mesolimbic dopamine.[32] Hormone-influenced neuroplasticity infers behavioral changes that include an elevated preference for high-fat and high-sugar diets commonly associated with the phenomenon of food addiction.[33]

Human research using functional magnetic resonance imaging (fMRI) measured responses to visual food stimuli in obese subjects compared to controls.[34] As expected, obese participants had significantly higher plasma leptin concentrations, possibly experiencing a state of leptin resistance. Meanwhile, Kalra states that hypothalamic leptin insufficiency rather than "leptin resistance" leads to decreased energy expenditure, increased energy intake, and consequential obesity.[35] Grosshans et al found a significant positive correlation between plasma leptin concentration and brain activation in the ventral striatum (area which includes the nucleus accumbens) during the presentation of visual food cues,[34] strongly suggesting that the homeostatic feedback mechanism between leptin and mesolimbic reward function is impaired in obese subjects.

## Ghrelin

Ghrelin has opposing effects with leptin, stimulating appetite by activating orexigenic neurons in the hypothalamus. Additionally, ghrelin receptors have also been identified in the VTA, hippocampus, and amygdala.[36] Both ghrelin and leptin play a central role in the neuroendocrine regulation of food intake and energy homeostasis.[30] Ghrelin is stomach-derived and will decrease after eating thus contributing to satiety. Leptin counters the effects of ghrelin thereby decreasing relentless hyperphagia. The sight of food significantly elevates ghrelin levels in non-obese healthy volunteers.[37] In obese human subjects, ghrelin levels are lowered, whereas post-meal ghrelin levels remain higher than in lean

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individuals.[38] Direct injection of ghrelin in the VTA and nucleus accumbens increases feeding behavior.[39] Recent findings suggest that the VTA but not the nucleus accumbens is the direct target site for ghrelin's action on sweet food motivation.[40] Ghrelin also enhances intake of artificially sweetened food (saccharin), implying a role in feeding behavior regardless of caloric content.[41]

Dickson et al suggest that the ghrelin system alters the set point of the dopaminergic neurons in the VTA, thereby enhancing the ability of rewarding substances to activate the midbrain dopamine system.[42] Anticipatory physiological responses to scheduled meals can be learned through ghrelin's interaction with central nervous system (CNS) reward pathways that stimulate motivation to eat[28] via increased release and activity of VTA dopamine.[23] While regular or palatable food by itself activates the mu opioid receptor pathway in the VTA, systemic ghrelin switches the dominant opioid receptor pathway from mu to kappa only for highly rewarding food.[43] These findings suggest a pivotal role of ghrelin in regulation of food incentives and hedonics. Meanwhile, other investigators in contra-distinction have concluded that ghrelin primarily exerts motivational effects on feeding, rather than hedonic or opioid-related effects.[44]

The central ghrelin signaling system interfaces neurobiological circuits involved in reward from both food and chemical drugs including alcohol. Increased ghrelin signaling could contribute to the overconsumption and preference for high-calorie food[45] and alcohol, a high-calorie beverage.[46] Whether or not ghrelin plays a significant role in losing control over drug-taking behavior is yet to be determined. Humans subjected acutely to psychosocial stress displayed increased plasma ghrelin, particularly "emotional eaters" where ghrelin did not decline acutely following food consumption.[47] Since stress has been linked to drug relapse and is a significant cause of failure in dieters, it is possible that stress-related increases in ghrelin are a risk factor for substance-seeking behavior. Some authors have suggested that ghrelin antagonists have therapeutic potential for the treatment of obesity by suppressing overconsumption of sweet food.[48] Similarly, ghrelin agonists might increase the motivation to eat, which could be helpful in cases where the drive to eat is insufficient.[44] Kawahara et al recommends further study on the role of ghrelin in regulating the mesolimbic dopamine system in response to drugs of abuse and alcohol.[43]

## Insulin

While widely studied in connection with the regulation of blood glucose, the CNS effects of insulin are not fully understood. Much like leptin, insulin is an adiposity signal, is anorexigenic, and attenuates food reward. Leptin gene therapy represses insulin secretion and can potentially ameliorate diabetes.[35] In metabolic circumstances in which plasma insulin or leptin levels are low (starvation and reduced adiposity), signaling would be decreased and drive for food intake increased. Insulin and dopamine work together to orchestrate both the motivation to engage in consumptive behavior and to calibrate the associated reward, particularly related to hedonic feeding.[49] More specifically, insulin depresses dopamine concentration in the VTA, which may suppress salience of food once satiety is reached.

Similar to ghrelin, there are insulin receptors in the hypothalamus, VTA, hippocampus, and amygdala.[36] In humans, insulin secretion is decreased by ghrelin, and vice versa.[50,51] There is also evidence that the insulin receptor signaling pathway interferes with leptin signaling, indicating that hyperinsulinemia contributes to the pathogenesis of leptin resistance.[52] Chronic hyperinsulinemia promotes obesity by interfering with leptin extinguishing of dopamine clearance in the nucleus accumbens, which is a hallmark of addiction.[53] Insulin resistance may directly or indirectly impact neural pathways driving desires to consume highly caloric foods and ultimately influence further adiposity.[54] These authors also reported that during exposure to stress, desire to eat is exacerbated in obese humans but not lean individuals. In obese individuals, evidence of insulin resistance can lead to alterations in food craving even in a relaxed state.

Daws et al reviewed the potential impact of impaired insulin signaling in obesity and stimulant abuse suggesting that insulin-influenced dopamine transmission can affect the ability of drugs to exert their neurochemical and behavioral effects.[55] According to these authors, insulin receptors are present in brain and are found on midbrain dopamine neurons, and the interplay between insulin signaling and drug-induced increases in extracellular dopamine may contribute to the high comorbidity of eating disorders and drug abuse. Improvements in brain dopamine function by normalizing or bypassing disruptions in insulin signaling might be effective in treating addictions.

## Alcohol

**Leptin.** The course of alcoholism is associated with suppressed secretion of ghrelin and leptin, both of which influence the hypothalamic-pituitary-adrenal system.[56] Increasing leptin concentrations have been correlated with the course of alcohol withdrawal in human females.[57] While alcohol attenuates the secretion of leptin in the short run (6-8 hours) in non-alcoholic, non-obese human subjects,[58] chronic alcoholism has been linked to elevated leptin.[24] This may indicate that fat tissue of alcoholic patients is sensitized to release more leptin than controls, however levels do normalize after six months of abstinence.[24] Lenz et al reported that lower levels of leptin were correlated with lower levels of alcohol craving in males,[59] while the opposite is true for women.[57] There is epidemiological support for a link between familial alcoholism and risk for obesity in women, and possibly for men.[60] Some authors have suggested that alcoholic men outnumber women four to one, highlighting neurological and hormonal differences that may account for these observed differences.[61] Further investigations into the relationship between leptin and alcohol craving accounting for differences in gender as well as the nutritional status of subjects are clearly warranted.

**Ghrelin.** The rewarding properties of alcohol require ghrelin.[62] Alcohol-dependent patients have increased ghrelin levels when intoxicated and during early abstinence, increasing during the first week of alcohol withdrawal.[57] It is likely that elevated ghrelin can account for measurable changes in hunger and appetite during alcohol withdrawal. The common practice of healthy individuals drinking an alcoholic

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beverage before a meal suggests that alcohol stimulates appetite. Jerlhag et al suggest that by increasing the incentive value of rewards such as alcohol, hyperghrelinemia may play a patho-physiological role in the disease process that leads to addiction.[62] The authors conclude that modulation of ghrelin signaling constitutes a potential target for treatment of alcohol-related disorders. Other human research led by Leggio and colleagues have confirmed the findings that ghrelin plays a key role in alcohol-seeking behavior,[46] highlighting the key role of dopamine in the neurobiology of alcohol craving. The authors conclude that antagonizing ghrelin via homeostatic stabilization might lead to new and innovative ways to provide effective treatment for alcohol use disorders.

**Insulin.** Abstinent alcoholic human subjects have exhibited significantly blunted responses in blood glucose when exposed to intravenous 2-Deoxy-D-glucose.[63] Subjects exhibited trends towards both blunted responses in glucagon and insulin. Authors speculate that that nervous system damage attributable to the effects of alcohol exposure is responsible for the insufficient hormonal response, particularly neurons in the hypothalamus, as well as the adrenal medulla. It has been established that alcohol-dependent subjects during the first month of abstinence report maximal pleasure response to the sweetness significantly more frequently than control subjects,[64] consistent with the concept of "reward deficiency syndrome."[65] The percentage of alcohol-dependent subjects preferring the maximum concentration of sucrose decreased over time. Those alcohol-dependent subjects who reported abstinence at six months were significantly less likely to prefer the maximum sweetness than were the subjects who did not maintain abstinence. Krahn et al propose that sweet preferences should be tested as a predictor of future abstinence.[64] Positive associations between the consumption of any type of alcoholic beverage and anthropometric markers of adiposity have been reported.[66]

Taken together, the apparent link between alcohol abuse and sugar abuse and the subsequent blunted hormonal responses highlight the negative impact of substance use on the endocrine system, providing support for the need for dietary intervention in supporting long-term abstinence and recovery. Manipulation of the insulin signaling system should not rely solely on pharmacological intervention but rather should focus on normalizing the altered dopamine-glucose link via interventions in nutrition behavior, which has proven to be challenging in the light of the science on food addiction.

## Stimulants

**Methamphetamine.** Crystal methamphetamine (meth) use may be associated with the onset of disordered eating or used as an efficient weight loss mechanism for those with established eating disorders.[67] Food restriction has been shown to enhance the central rewarding effect of amphetamine.[68] Research by Jerlhag et al demonstrates that the ghrelin signaling system is required for indirect measures of the rewarding properties of amphetamine, as well as cocaine.[69] The authors highlight the fact that food restriction leading to elevated ghrelin facilitates the acquisition of drug-seeking behavior in rats, attributable to the dopaminergic regions of the nucleus accumbens and VTA. Hyperghrelinemia observable in SUD patients raises important questions regarding the physiological role of ghrelin influencing not only food intake and appetite, but also a broader role in reward induced by addictive drugs such as alcohol, amphetamine, and cocaine.[69] The potential for a gradual normalization of ghrelin levels through medical nutrition therapy appears indicated for patients with methamphetamine use disorders.

**Cocaine.** Research using positron emission tomography (PET) brain imaging has suggested that deficits in dopamine signaling are similar for cocaine-addicted and obese rats.[11] The authors suggest that dopamine binding ability in the D2R/D3R sites can be used to predict future body weight and cocaine preference. A small sample of human cocaine addicts in an inpatient setting reported preference for the highest concentration of sweet solutions, which is in agreement with sweet-preference expressed by alcoholics.[64,72] Clearly sugar reinforces depleted reward pathways in the brain resulting from cocaine abuse. Meanwhile, research conducted on rats has shown that antagonism of ghrelin receptor function has reduced the development of cocaine sensitization, strongly supporting the view that ghrelin receptors are partially responsible for modulating reinforcement/reward function.[70]

A sample of female crack cocaine users presented with lower levels of plasma leptin during early abstinence in comparison with healthy controls, consistently increasing during detoxification.[71] The authors speculate that leptin levels may increase in abstinence only as a consequence of improved diet or weight gain. Consistent with the link between leptin and inflammation reported by Heber and Carpenter[21] and Levandowski et al,[71] it should be acknowledged that disruption of energy homeostasis could interfere with clinical responses to cocaine treatment, since cocaine addicts demonstrate increased immune response inflammation both at the baseline and in response to stress and cue imagery conditions.[73]

According to recent research conducted by Ersche et al,[8] cocaine-dependent men (not abstinent) reported increased food intake, specifically foods high in fat and carbohydrate. There was an expected trend towards lower levels of circulating leptin in the cocaine group, directly interfering with metabolic processes (impaired energy balance). In other words, the higher fat intake was associated with less fat storage, suggesting an inhibition of leptin production that facilitates overeating. The overeating in cocaine-dependent individuals often pre-dates recovery, with the effect masked by lack of weight gain. Taken together, investigators found that cocaine abuse results in an imbalance between fat intake and storage, leading to excessive weight gain during recovery.[8] For many individuals, it is likely that dysfunctional eating pre-dates the initiation of drug use as well. According to Wiss and Waterhous,[74] patients with SUDs often develop disordered and dysfunctional eating patterns during abstinence, and eating disorder patients can similarly progress into substance abuse. Traditionally addiction has been addressed first, however delaying eating disorder treatment can hinder recovery, therefore it is important to alert treatment providers who treat patients

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with dual diagnoses how to assess and address both disorders simultaneously.

**Ecstasy.** Ecstasy is a popular club drug classified as an empathogen or entactogen often mixed with stimulants. In humans, abuse of this drug reduces eating, although there is research to suggest that women who use ecstasy are not necessarily taking it as deliberate means of weight control.[75] In rats, ecstasy was shown to cause significant decreases in serum leptin and increases in serum ghrelin, both of which recovered to baseline after 24 hours.[76] Long-term effects of altered hormonal levels related to ecstasy and stimulant abuse require further evaluation, with more emphasis on behavioral parameters such as food intake.

## Opiates

Acute food deprivation (24 hours) reinstated heroin-seeking in rats, and this effect was attenuated by leptin infusions.[77] Similar to other substances of abuse, decreased activity in the mesolimbic dopaminergic reward system (VTA and nucleus accumbens) following leptin administration is likely to account for this effect. Not surprisingly, Nolan and Scagnelli found that methadone-treated human patients had a higher consumption of sweets, a higher eagerness to consume sweet foods, and a willingness to consume larger quantities desired by controls.[78] In another sample of human patients on methadone maintenance, basal serum leptin concentrations were significantly decreased compared to controls.[79] These findings were independent of BMI, body fat, and insulin sensitivity.

Recent research has also shown that chronic food restriction (14 days of mild restriction) led to robust heroin-seeking behavior in rats.[80] The authors acknowledge the "stress aspect" of food restriction on reward-seeking behavior, meanwhile recognizing that the state of hunger by itself was not sufficient to induce augmentation of heroin-seeking. D'Cunha et al concluded that ghrelin is likely more responsible than leptin for mediating the effect of food restriction on heroin-seeking following prolonged abstinence.[80] These findings are in agreement with Maric et al who provided evidence that activation of ghrelin receptors is sufficient to induce increases in drug-taking and drug-seeking behaviors.[81] Meanwhile, these authors point out that ghrelin is not required for this mechanism since treatment with a ghrelin receptor antagonist had no effect on drug-taking or food deprivation-induced reinstatement of extinguished heroin-seeking.

## Nutrition and Addiction Treatment

**Disordered Eating.** Drug abuse is a risk factor for eating disorders[82] and has been shown to have both genetic and environmental influences.[83] Even a remote history of SUD can negatively impact weight loss in adults[84] and adolescents.[85] Sobriety time has been positively associated with increased sugar use.[86] Substance abuse linked to low distress tolerance can lead to excessive consumption of food.[87] Fischer et al found that problems of alcohol use were associated with binge eating and purging, and that a tendency to act rashly when distressed was associated with both behaviors.[88] In one study, nearly 40% of women in SUD treatment met criteria for an eating disorder most commonly binge eating disorder followed by bulimia nervosa.[89] Men in SUD treatment reported bingeing and the use of food to satisfy drug cravings during the first six months, with weight concerns and distress about efforts to lose weight during months 7-36.[90]

**Interventions and Outcomes.** Positive associations between nutrition interventions and substance abuse outcomes have been reported, where nutrition education was the differentiating factor.[91] An educational intervention on the nutrition behavior of alcohol-dependent patients led to 80% of participants reporting continual abstinence after six months.[92] A six-week environmental/educational intervention to improve dietary intake and reduce excessive weight gain among men in residential treatment reported greater reductions in total energy, percentage of energy from sweets, daily servings of fats, oils, and sweets, and BMI over the intervention period.[90] The findings provide evidence that such interventions can be successful despite challenges met in residential substance abuse facilities. A series of nutrition workshops in a substance abuse program (SAP) in the US prison system led to significant improvements in nutrition and general health, with a trend towards improvements in social ties.[93] A review article on the drug-addicted prison population in the UK builds a convincing argument for the inclusion of more nutritious options in prisons, concluding that such changes are overall likely to make sound economic sense in terms of prisoner health, mood, behavior, and recidivism rates.[94]

**Upcoming Trends.** Betty Ford is a world-renowned treatment center in California that has recently merged with Hazelden in Minnesota. Betty Ford utilizes a treatment model that includes measures to prevent post-detoxification overeating. Patients are provided with access to dietitians and exercise is emphasized, helping patients to plan for expected changes in eating and the reinforcing effects of food. At Breathe Life Healing Center in Los Angeles, a registered dietitian nutritionist is an integrated member of the treatment team, approving all food and beverages that enter the campus, planning nutritionally balanced meals and snacks, teaching educational courses, and working with patients individually to mediate dysfunctional eating behavior and disrupt addictive tendencies. Exercise is also a mandatory component of treatment. It is possible that data supporting the effectiveness of improved nutrition and exercise behavior during the course of treatment will eventually be reported.

## Discussion

The most substantial health burden arising from addiction lies not in the direct effects of intoxication but in the secondary effects on physical health.[8] There is strong evidence to support that food and drugs are competing for overlapping reward mechanisms. When the immediate crisis of substance abuse has been resolved, there is a likely compensatory increased drive for food intake to achieve weight recovery and a likely overshoot, leading to increased adiposity.[13] Ravenous food consumption may be due to "rebound appetite" in the wake of the hypothalamic suppression from drug use. Making healthful food choices after abstinence has been achieved may be very challenging. Sobriety is associated with new emotions, anxiety, and uncertainty. It is easy to seek a predictable and comforting response from food. This may lead to

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overeating, relapse, compromised quality of life, and the development of chronic disease. Caffeine and nicotine abuse should also be addressed since they are highly addictive substances that can perpetuate substance-seeking behavior. Additionally, the impact of stress and adequate sleep should not be ignored, as they too can have profound effects on the endocrine and reward systems.

The modern epidemic of obesity may be in part related to reward and hedonic mechanisms, and that failure of regulatory systems might be related to dysregulation of reward systems. Normalizing the disrupted leptin signaling cascade in the obese brain may be sufficient to decrease motivation for food reward, and interventions targeting the central leptin system and/or other hypothalamic hormones regulated by leptin should be considered for the treatment of drug addicts with comorbid eating disorders. Kalra has proposed trials involving gene therapy aimed at reinstating leptin circuitry in drug addicts.[35] More realistically, weight gain during substance abuse recovery should be monitored and controlled (gradual rather than drastic) in order to counter the associated adaptions in nutrition-related hormones. In order to accomplish this, exposure to highly palatable foods with addictive potential should be minimized.

Educational efforts alone have not reduced use of drugs of dependence. Successful efforts have required both individuals and societal intervention including taxation, regulation, and/or interdiction. Similar efforts targeting the food industry may be required to combat the rising epidemics of food addiction, obesity, and binge eating disorder. Given that individuals with a history of SUD are at higher risk for developing food-intake-related dysfunction, there is a substantial need for nutrition interventions in addiction recovery, and registered dietitian nutritionists should become vital members of the treatment team. Currently, there is no requirement for nutrition education and counseling in substance abuse treatment. Anecdotal reports suggest that most treatment centers allow unlimited or excessive amounts of highly palatable foods to patients. While food restriction can lead to relapse, over-indulgence can perpetuate the cycle of addictive behavior and contribute significantly to healthcare burden. The best intervention appears to lie somewhere in between these extremes, which will require additional clinical expertise in treatment settings. The need for firm commitment to intervention protocols as well as ongoing supervision and consultation is warranted for successful program implementation in residential drug-treatment facilities.[90]

## Conclusions and Further Implications for Nutritionists

Restoration of nutritional status in SUD recovery should look beyond correction of vitamin/mineral status and body weight, but should also account for recovery of dysfunctional neural circuitry and altered hormones. Before a successful nutrition intervention can occur, it is of paramount importance to heal gut function to promote optimal nutrient absorption throughout the gastrointestinal tract. Next, preventing over-exposure to highly palatable foods is critical in repairing addictive processes in the brain. Finally, restoration of hormone levels should occur through gradual yet progressive changes in eating behavior, although there is limited data to support this approach given the presence of confounding variables over extended periods of time. From my own experience in clinical practice, utilizing several small feedings throughout the day is an effective approach towards preventing spikes and subsequent drops in insulin. Given that insulin can block leptin, this technique may be effective in gradually normalizing leptin levels, although to my knowledge there is no data in the SUD population to support this claim. Similarly, stable insulin levels achieved through regular and consistent feeding patterns may prevent ghrelin from increasing to abnormal levels. The mantra I encourage with my SUD patients is "never hungry, never full" and the evidence reviewed herein provides strong support for this approach. Gradual increases in fiber intake throughout the recovery process can improve gut function, minimize undesirable insulin spikes, and promote satiety. In our recent publication, we provide recommendations on nutrition therapy during SUD recovery for specific substances of abuse.[74]

The current trend towards over-medicating SUD patients while failing to address and improve nutrition behavior should be aggressively challenged. Consider this is a call to order for data collection linking drug addiction to reward-related hormones, specifically demonstrating the importance of medical nutrition therapy in SUD recovery over short periods (1-6 months) and longer periods (6-36 months). Without this data, it will be difficult to substantiate the need for nutrition interventions in addiction recovery at the policy level. Nutrition interventions during recovery may prevent or minimize the onset of chronic illness, improving resource allocation. Public health measures should be considered critical.

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# Practice Examples Reflecting the Standards of Practice and Professional Performance for Registered Dietitians in Intellectual and Developmental Disabilities

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Rosenzweig, L., Wittenbrook, Practice Examples Reflecting the Standards of Practice and Professional Performance for Registered Dietitians in Intellectual and Developmental Disabilities. *ICAN: Infant, Child, & Adolescent Nutrition* Vol. 5 (5) pp. 306-310, 

**Abstract:** *The Behavioral Health Nutrition Dietetic Practice Group of the Academy of Nutrition and Dietetics has developed Standards of Practice (SOP) and Standards of Professional Performance (SOPP) of Registered Dietitians in Intellectual and Developmental Disabilities (IDD). These guidelines are recommended for use by dietitians in developing and advancing their professional practice in serving individuals with IDD in various settings and circumstances. Two examples of how some of these specific standards are followed in real-life settings are presented. The first example is Carol, a young woman with a learning disability who has recently been diagnosed with celiac disease and is now following a gluten-free diet. Targeted interventions to Carol, her group home, program, and supportive staff, as well as family members and educators are described in addition to other activities related to selected standards to ensure diet understanding and compliance. The second example is about Laura, a 12-year-old girl with spina bifida, hydrocephalus, and scoliosis. Laura will have surgical correction of her scoliosis in 6 months. The registered dietitian will work with Laura, her mom, and the medical team to help Laura optimize her nutritional intake in preparation for the surgery, with activities related to selected standards.*

**Keywords:** developmental disabilities; growth and development; special health care needs; medical nutrition therapy

The Behavioral Health Nutrition Dietetic Practice Group of the Academy of Nutrition and Dietetics has recently published the revised Standards of Practice (SOP) and Standards of Professional Performance (SOPP) for Registered Dietitians (RD) in Intellectual and Developmental Disabilities.[1] The SOP is based on the 4 steps of the Nutrition Care Process: (1) Nutrition Assessment, (2) Nutrition Diagnosis, (3) Nutrition Intervention, and (4) Nutrition Monitoring and Evaluation.[2] The SOPP is divided into 6 different standards to provide a framework for RDs to use in order to assess and advance their level of practice in working with individuals with intellectual and developmental disabilities (IDDs). Three levels of practice (Competent, Proficient, and Expert) are identified to assist RDs in determining

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their individual competencies and need for additional training.[1]

Individuals with IDDs have unique nutritional needs to be addressed in ways that consider not only the individual but also their circle of support (COS), which goes beyond the immediate family and may include extended family, group home staff, teachers, and medical staff, among others. In addition, there can be communication and learning barriers as well as physical limitations that have to be considered when providing education and services to individuals with IDDs. Since each state does not have legislation to define the RD's scope of practice, RDs may use the published standards to help guide them when conducting assessments and providing interventions with individuals with IDD and their caregivers. Two examples of how selected standards of practice from the SOP/SOPP for RD in IDD article (as they are numbered) may be used in practice are presented here.

### Example 1

Carol is an 18-year-old group home resident with a learning disability who is following a gluten-free diet because of a recent diagnosis of celiac disease confirmed by a celiac blood panel and intestinal biopsy. The RD will need to provide individual counseling for implementation of a gluten-free diet to Carol in addition to her multiple care providers in her COS. Carol lives with 5 other young adults, some of whom are finishing school and/or attending a day habilitation "Life Prep" program. Carol is completing her high school education part-time at the local public school in a special education program. At the "Life Prep" program, Carol learns skills, such as using a computer and the Internet, handling money, and cooking. All residential services are provided to Carol, including nursing, medication administration, transportation, and meals. Carol is able to purchase food and beverages with her own money. Sometimes Carol goes out for a day to eat lunch with her aunt who lives in the area.

Carol was recently diagnosed with celiac disease after experiencing constipation and intermittent diarrhea during the previous year and trying different diet therapies, such as high-fiber and lactose-free diets. The nutrition goal that requires intervention in this example is to ensure that Carol is able to make appropriate food choices within the restrictions of a gluten-free diet to reduce the complications of celiac disease. The following examples illustrate some of the SOP and SOPP indicators used by the consulting RD for the group home when working with Carol.

(SOP 1.2D1) Reviews evidence-based nutrition indicators of complications arising from various treatment modalities (eg, vitamin and mineral deficiencies related to a gluten-free diet).

The RD determined the need for Carol, group home staff, school staff, and others to read food product labels so food products containing gluten could be identified and avoided. In addition, the RD recommended choosing enriched food products when possible, especially those that provide B vitamins, iron, vitamin D, and fiber. Individuals with celiac disease can have low levels of these nutrients secondary to their disease.[3,4]

(SOP 3.4) Confers with individual, family, and care providers—considers knowledge, skills, and willingness to implement nutrition intervention to achieve goals.

The RD identified key players within Carol's COS that needed to be included in the nutrition care plan: consumers in the group home, group home management and direct care staff, residential nurse, "Life Prep" staff, school staff/administration/students, and family members.

(SOP 3.6) Details the nutrition intervention plan and strategies to achieve goal

(SOPP 1.5) Informs and involves individuals and their family/care providers in decision making.

The RD used a "person-centered"[5] approach, focusing on Carol the individual rather than her disabilities. After meeting with Carol and some members of her COS, the RD developed the nutrition care plan to include (1) review of celiac disease and how it is treated; (2) review of signs and symptoms of gluten ingestion; (3) identification of wheat, grain products, and other potential sources of gluten to avoid; (4) identification of gluten-free products in the local grocery and specialty food stores and bakeries; (5) identification of gluten-free products preferred by Carol; (6) incorporation of gluten-free foods into the group home menu; (7) proper food handling techniques to avoid gluten contamination; and (8) strategies to help Carol manage her gluten-free diet away from her group home setting.

(SOP 3.10) Collaborates with colleagues.

The group home staff was responsible for purchasing gluten-free foods, incorporating them into Carol's menu plan, and reinforcing the instruction the RD provided on a gluten-free diet. The consulting RD provided 2 training sessions for all group home staff to review the gluten-free diet and its implementation, including the avoidance of cross-contamination. It was imperative that all members of Carol's COS were familiar with her gluten-free diet.

(SOP 3.14) Individualizes nutrition intervention.

(SOPP 1.9) Applies knowledge and skills to determine appropriate interventions.

Carol enjoyed eating and would frequently refuse to eat lower calorie foods typically offered in the group home. Although Carol was able to read product labels and identify "gluten-free" and "contains wheat" labeling on a food product, she also needed to understand the importance of compliance with the gluten-free diet within the limits of her learning disability.

The RD determined the level of Carol's understanding through informal review of the information presented to her, her history of learning disability, and level of support needed. In addition to the training, a handout with photos of gluten-free food product labels and labeling information was developed. As many gluten-free products do not provide a significant amount of dietary fiber, a list of high-fiber foods was also developed based on Carol's food preferences. These were shared and explained to all those identified previously during the first and subsequent training sessions. The RD brought in gluten-free food products for Carol, group home consumers, and staff to taste test. The RD accompanied Carol and several group home staff members on a grocery store tour to identify and select gluten-free foods and food products with the assistance of the in-store dietitian.

(SOP 4.1) Monitors progress.

During the training phase for the implementation of the gluten-free diet,

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the RD visited the group home several times to check Carol's and staff adherence to the diet. Food products and meal choices were reviewed with group home staff, particularly the gluten-free choices. Food handling methods were reviewed to prevent cross-contamination with gluten. The RD initially met with Carol every few weeks to assess her knowledge of the diet, proficiency in reading labels, and her satisfaction with the food and meals provided to her. The RD also followed up with available COS members on regular visits to assess her understanding of the gluten-free diet.

The RD recommended Carol's medical team obtain biomedical data when she has her annual physical exam to check for nutritional anemia, and vitamin, mineral, lipid, electrolyte, and renal profiles, which may be affected by the disease and/or gluten-free diet.[6]

(SOP 4.4H) Documents future plans which may include recommendations for continuation of nutrition care, nutrition monitoring and follow up referral and discharge.

Carol's nutrition intervention presented here is at the start of her lifelong need to follow a gluten-free diet. The nutrition plan of care will need to be monitored, particularly diet compliance by Carol and her COS. Interventions would be provided based on the needs of her COS, and changes in Carol's life situation and health status.

## Example 2

Laura is a 12-year-old girl with spina bifida, hydrocephalus, a ventriculoperitoneal shunt (to treat hydrocephalus), neurogenic bowel and bladder, and scoliosis. She has been referred to the RD by the orthopedic surgeon for a preoperative nutrition evaluation to prepare for surgical correction of her scoliosis.

Over the past 6 months, Laura has lost 1.5 kg and was hospitalized twice with pneumonia. In addition to weight loss, her triceps skin fold measurements have dropped from the 10th to 25th percentile to the 5th percentile. Laura's bowel management program for neurogenic bowel had been relatively predictable on a daily basis with a good consistency. However; during the past three to four months, her bowel movements have become more ball-shaped, larger, and firmer. She has had one symptomatic urinary tract infection. Laura's mother reported that Laura had decreased the amount of fluids she was drinking. Laura had also begun to cough and choke while drinking water and juice. After the RD discussed the case with the developmental pediatrician in the interdisciplinary clinic, he requested a modified barium swallow study (to rule out aspiration), lab studies (to assess if iron, zinc, or vitamin D supplementation was needed before surgery), and a KUB (the kidney–ureter–bladder x-ray can be used to rule out constipation).

(SOP 1.1A5) Evaluates dietary intake for existing/potential factors that may impact intake of a nutritionally adequate diet related to eating ability, digestion, and absorption.

The RD first evaluated Laura's diet history for fluid and fiber intake, and found Laura was only meeting 70% of her fluid needs based on the Halliday-Segar equation,[7] and 90% of her fiber needs, based on the "age +5 recommendation" commonly used in pediatrics.[8] Laura was only meeting 80% of her estimated energy needs based on the formula for children with spina bifida.[9] Her protein needs were assessed at 2 g/kg[10] to assist with post-operative wound healing. In addition, the RD evaluated Laura's food preferences to determine if they were symptomatic of dysphagia.

(SOP 1.1B2) Recommends changes in diet prescription as necessary to improve nutritional status

(SOP 1.2B6) Reviews physical findings that may indicate structural or functional abnormalities (swallow study, x-rays).

The dietitian made the following recommendations for Laura and her mother to implement at home: (1) increase fluid intake by 2 cups daily to assist with bowel management, following the recommendations from the modified barium swallow study; (2) increase energy intake to promote weight gain by adding high-calorie snacks after school and at bedtime; (3) increase protein intake to meet goal on a daily basis; and (4) begin taking a children's daily multivitamin with iron.

(SOP 1.2B3) Uses specialized techniques and/or equipment and trained personnel to obtain measurements, such as skin fold calipers, as appropriate.

Laura's triceps skin fold measurements were compared to previous measurements one year earlier, and showed Laura had a reduction in her energy stores in the form of fat based on the changes in the measurements. The additional information provided by serial skin fold measurements provides the RD with valuable information to help interpret growth trends when used in combination with growth charts.[10] Since an accurate height measurement is difficult to obtain in individuals with scoliosis, recumbent length was obtained to measure Laura's height, as she was unable to stand. The RD reviewed Laura's growth chart to assess her growth trends, which showed a plateau in her linear growth velocity with the worsening of her scoliosis and the challenge in obtaining reliable length measurements on a consistent basis.

(SOP 1.2B6) Reviews physical findings that may indicate structural or functional abnormalities (swallow study, x-rays).

Laura's modified barium swallow study showed Laura had a delayed swallow with aspiration of thin liquids. The interdisciplinary dysphagia team (physician, speech-language pathologist, RD, RN, occupational therapist) recommended Laura thicken her liquids to nectar consistency or drink naturally thick liquids, such as smoothies, fruit nectars, to reduce aspiration risk. Laura's KUB showed a moderate amount of constipation determined to be related to her reduction in fluid intake, as Laura had consistently taken her medications for management of neurogenic bowel.

(SOP 1.2E) Evaluates diagnostic tests, procedures, and evaluations.

Laura's lab results showed her hemoglobin, serum iron and ferritin levels were low. The dietitian evaluated the labs and recommended the physician prescribe ferrous sulfate in addition to the patient taking a children's chewable multivitamin with iron in preparation for surgery. Her other labs were within normal limits.

(SOP 3.10) Collaborates with colleagues

(SOPP 1.8A) Participates as a member of the interdisciplinary team to meet the needs of the individual with IDD.

The dietitian collaborated with the developmental pediatrician, orthopedic surgeon, and feeding team to make

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recommendations for Laura's preoperative plan of care, along with nutritional management during her inpatient stay for scoliosis surgery.

(SOP 3.11) Initiates plan of care

(SOP 3.14) Individualizes nutrition interventions

(SOPP 3.3A) Determines the most appropriate information and best method/format for presenting based on level of understanding and cognition of the individual and/or target audience.

When the RD met with Laura and her mother, she discussed the recommendations and reviewed the relationship between good nutrient intake (energy, protein, and micronutrients) and wound healing. In addition, good bowel management can help with wound healing and reduce infection risk in individuals that have spina bifida. Children with spina bifida have neurogenic bowel and bladder and require comprehensive management via a bowel management protocol and cathing to prevent incontinence. Because of the reduced sensation from spina bifida, children are also at increased risk of pressure ulcers, which significantly increases infection risk when combined with incontinence.[11] Managing incontinence can help keep the postoperative wound clean and reduce infection risk.

The RD also recommended Laura drink nectar thick or naturally thick liquids to prevent aspiration and reduce the risk of postoperative respiratory complications. The RD demonstrated how to use commercial thickeners to thicken liquids and reviewed a list of liquids that are naturally thick. A Spanish translator was used for the session with the RD, as the mother's primary language is Spanish. Written educational materials were provided in English and Spanish.

(SOP 4.1B) Determines whether the intervention is being implemented as prescribed.

One month later, the RD met with Laura and her mother for a follow-up visit. Laura's weight was up by 1 kg, and she was able to eat a high-calorie snack twice daily as recommended. Although her protein intake had increased, Laura was sleeping in on the weekends and typically skipped breakfast. Laura preferred to drink naturally thick liquids instead of liquids thickened with a commercial thickener. The increase in Laura's fluid intake resolved the problems she had with constipation. Laura had been consistent in taking her children's chewable multivitamin with iron and her ferrous sulfate supplements.

(SOP 4.1E) Gathers information to indicate progress or reasons for lack of progress.

(SOPP 1.9B) Utilizes knowledge, demonstrated by an understanding and use of the general principles, theories, and practices related to individuals with IDD to develop an intervention plan that is flexible, and to accommodate unforeseen barriers or unplanned consequences.

The RD collaborated with Laura and her mother to find ways to increase her protein intake on the weekends. After discussion with Laura and her mother about several options to increase Laura's protein intake, including high-protein shakes or bars, Laura and her mother chose to wake Laura up earlier on the weekends so that she would be able to eat a high protein breakfast. Since Laura preferred to drink naturally thick liquids instead of using commercial thickener, the family and school would continue with this option to reduce aspiration risk.

(SOP 4.4) Documents.

After the initial and follow-up visits, the RD dictated her clinic notes for the medical record according to the policies and procedures of the medical facility so the information will be available to the RD following Laura when she is an inpatient for her scoliosis surgery to ensure continuity of care.

## Summary

In summary, RDs are encouraged to use these published standards to help guide them in their practice and skills development when working with individuals with IDD and their caregivers. The IDD practice setting is an area that requires RDs to be proactive team players and identify creative nutrition intervention approaches that are addressed in the SOP and SOPP. Increasing IDD-related knowledge and skills (as noted in the SOP and SOPP) through education, training, and experience is needed by RDs at all levels of practice. The RD can educate medical and health professionals, caregivers, and other support personnel (also referred to as the individual's COS) on the complex nutritional, health, and behavioral needs of individuals with IDD. RDs have the opportunity to increase the health literacy of individuals with IDD by encouraging their participation in health promotion, screening, and wellness programs, and by providing nutrition education. RDs working in the field of IDD are encouraged to be an advocate for appropriate support systems (including funding for nutrition services) in order to address the unique needs of this vulnerable population and improve their quality of life.

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## Author Note

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# Cognitive Impairment in Iron Deficiency: Biological and Socioeconomic Relationships

Sarah L Thomsen-Ferreira, MS, RD, LDN, CNSC

## Prevalence of Iron Deficiency

The mineral Iron is an integral part of oxygen-carrying hemoglobin and many enzymes, deficiency of which is commonly associated with fatigue, difficulty maintaining body temperature, and decreased immune function.[1] Perhaps less well recognized is the detrimental effect of iron deficiency on cognitive function, specifically its detrimental role in impairing cognitive development and function.[2] Populations at highest risk of iron-deficiency anemia are infants and pregnant women due to increased iron needs, adolescent girls and women of child-bearing age due to menstrual blood loss, those with limited sources of heme iron in their diets, and those with renal failure or gastrointestinal disorders who do not absorb iron properly.[2] Recent estimates from NHANES data suggest that approximately 14% of children ages 1-2 years, and 9% of females age 12-49 suffer from iron deficiency anemia,[3] prevention of which is often dictated by availability of dietary sources of iron or iron supplements. Iron, in it's best form for optimum digestion and utilization, is found in animal foods that originally contained heme, such as chicken and beef liver, oysters, dark meat turkey, and beef.[2] Although not as efficiently utilized, non-heme iron can be found in high quantities in enriched breakfast cereals, cooked beans, tofu, and seeds.[2]

The risk for iron deficiency is heightened for those who suffer from food insecurity, which has been estimated to affect 14.5% of U.S. households.[4] Economic constraints often lend themselves to the selection of lower-cost cereals, sweets, and added fats, with a lower intake of more expensive fruits, vegetables, meats, and dairy products.[5] Calorically-rich, micronutrient-poor foods, too often more affordable than nutrient-rich fare, often play a definitive role in the failure to consume sufficient bioavailable dietary iron, in addition to other micronutrient containing foods important to cognitive function such as zinc, iodine, and B12.[6]

## Role of Iron in Cognitive Function

Iron plays an operative role in neurodevelopment, which begins during gestation.[7] The hippocampus, which has roles in memory, learning, attention, motor control, stress responsivity, emotional affect, and the ability to recall facts and events,[8] is especially vulnerable to iron-deficiency. Deficiency is responsible for reductions in hippocampal size, activity, and metabolism, as well as a reduction in neuronal connectivity.[8] As might be anticipated from those biological effects, early iron deficiency has been linked to attention and memory deficits, including slower response time of deficient infants to familiar stimuli as measured by brain activity.[9]

Within the neurotransmitter dopamine system, which is important in regulating cognition and emotion, motivation, positive affect, reward and pleasure, movement, motor function and hormone release,[10] iron deficiency results in a decrease in the number and density of dopamine transporters in the areas of the brain where iron concentrations fall.[11] It is suspected that alterations in dopamine status explain diminishing degrees of positive affect noted with iron-deficiency.[12] Iron supplementation of premature infants may reduce behavioral problems in early childhood,[13] while multiple studies have demonstrated that iron-deficient infants are more wary, solemn, and demonstrate less social interaction.[12] Additionally, Iron has a role in lipid biosynthesis (which is a part of myelin and cell membranes), and it's absence impairs neurotransmitter synthesis, myelin formation, and synapse stability.[11] Hence, early iron-deficiency anemia does not allow for the complete formation of myelination. Within a group of Chilean children, those with iron deficiency anemia underwent electrophysiological measurements to determine the speed of nerve potentials, which reflects the degree of myelination. Repeated measurements over a period of four years showed consistently slower conduction times, as would be expected with diminished myelination, as compared to controls.[14]

Unfortunately, remedial supplementation seems unable to reclaim hippocampal size and function later in life when deficiency has occurred during development,[15] and a chronic or persistent deficiency has the ability to negatively affect memory and executive function over the long-term.[16] Young adults who had experienced severe iron deficiency as infants demonstrate poorer performance

on information processing and memory tasks tied to the hippocampus than do non-deficient controls.[17]

Research suggests that when iron deficiency occurs outside of critical development periods, cognitive recovery is more consistently realized. Among women between 18-35 years of age, supplementation of iron sulfate resulted in faster completion of learning, memory, and attention tasks.[18] In iron-deficient adolescent young women, iron supplementation for eight weeks with ferrous sulfate has allowed for improved performance on multiple verbal learning and memory tests.[19] Within post-partum mothers, who are especially vulnerable to iron deficiency, iron status is positively correlated with cognitive performance, as well as with measures of anxiety, stress, and depression.[20] In older children and women with anemia, iron supplementation has been shown in some cases to improve attention and concentration, as well as increases in IQ by 2.5 points after iron supplementation.[21] Although supplementation seems to produce greater increases in serum ferritin in adult women when compared to a high-iron diet, either high-iron diets or supplementation tend to improve an anemic state.[22]

## Recommendations for Iron Intake

In summary, the role of iron in cognitive development and function is vitally important, and socioeconomic circumstances which discourage access to a micronutrient-rich diet can have long-term detrimental effects on cognitive capacity and quality of life. Public health and clinical interventions to prevent cognitive impairment from iron deficiency should be especially targeted toward developmental periods where iron is being utilized for brain development, such as with pregnant and breastfeeding mothers and their infants. The adequate intake level (AI) for infants up to 6 months is 0.27 mg/day, while the RDA increases up to 11 mg/day by 7-12 months and needs double during pregnancy.[1] Sufficient intake through diet and supplementation, as appropriate, plays an essential role in

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realizing full neurological and functional capacity at any age.

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## In the BHN Spotlight!

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### Describe your professional role.

I am the dietitian in a small private practice that also includes 2 mental health therapists and a registered nurse who is our lactation consultant. Our practice focuses on all feeding and eating concerns from infancy and throughout the lifespan.

### What was your career path? Looking back, would you have done anything differently if you had the opportunity?

I began my career as a bilingual dietitian with a WIC (Women Infants & Children) program in Seattle, Washington. I submitted my resume to my "dream job" for a position as a pediatric dietitian at Seattle Children's Hospital on the small chance that I would hear something back. Six months later, I received a telephone call asking if I was interested in a position at the hospital, it was a per diem position with no guaranteed hours and no benefits! I took the position and also applied for a Nutrition Traineeship through the LEND Program (Leadership Education in Neurodevelopmental & Related Disabilities) at the Center on Human Development & Disabilities at the University of Washington (UW)

### Looking back, would you have done anything differently if you had the opportunity?

I would NOT change a thing about that path in my career! I stayed at Seattle Children's Hospital for 13 years. In the fall of 2010, I decided to open a small private practice, while working part-time at Children's Hospital. In spring of 2013, I joined private practices with a clinical psychologist to become North Sound Nourishment & Recovery.

### What are some challenges you have faced in your career?

1) Standing for and supporting messages about childhood feeding and growth that are counter-culture in a culture that is weight-obsessed and food phobic. I continue to support that children are able to internally regulate their intake when provided a reliable and predictable structure of meals and snacks. 2) Starting a private practice has been challenging, but so rewarding to see how my hard work pays off in benefiting an underserved community.


### What are key lessons you've learned in your career?

Learn from those who have gone before us – I am continually grateful for the work of veteran dietitians at Seattle Children's who took time to train me in pediatric nutrition, dietitians at the LEND program at UW, and at a broader, national level – clinical work and research related to childhood feeding dynamics.

### What advice would you give to dietetic students or other dietitians in the BHN DPG looking to follow a similar career path as you?

Look to those who have "paved the way" before you. I was first interested in pediatric nutrition, then as I learned and developed through learning from the veteran dietitians at Seattle Children's Hospital, and the leaders of the Nutrition Traineeship; I started to realize my passion for pediatric growth and development and feeding issues throughout childhood and adolescence. Be forever curious about the wisdom of the veterans and how we all might learn from them. Thank you to all who have taught me about nutrition and helped make my career that it is today!

Heather Paves was randomly selected for a member spotlight in BHN Newsletter for her participation in the 2014 BHN member survey. Student member, Valerie Della Longa, kindly volunteered to interview and compose this member interest article.

# Student Corner:
# Putting the Eating Back in Eating Disorders

**Kelsey N. Wallour, BSFCS**

## Introduction

Eating is a complicated process that involves interactions between the homeostatic system, internal and external motivators, and self-regulatory control processes.[1] The self-regulatory system is where an individual's goals, values, and meaning are integrated, and from there decisions about eating can be made.[1] But, if eating disorders (EDs) are biopsychosocial disorders, then every one of those three areas that already makes "normal" eating complicated becomes more scrambled. Thus, symptoms are maintained intrapersonally and interpersonally by the positive and negative responses the eating disorder elicits.[2] Particularly when one considers that EDs, especially anorexia nervosa (AN), are often *egosyntonic,*[3] meaning that the behaviors, values, and feelings are in harmony with or acceptable to the needs and goals of the individual. This is classically exhibited in patients with AN who deny that they have a problem, even when markedly underweight and experiencing health complications.

It is estimated that approximately 50% of people with EDs suffer from obsessive-compulsive spectrum disorders,[4] which includes obsessive-compulsive disorder (OCD) and obsessive-compulsive personality disorder (OCPD). OCD is an anxiety disorder characterized by intrusive obsessions that lead an individual to engage in repetitive behaviors, even though he/she may realize that the obsessions are unreasonable.[5] OCPD is a personality disorder characterized by extreme perfectionism, desire to be in control of situations, inflexibility, and preoccupation with orderliness and rules.[6] Up to 60% of patients with anorexia are diagnosed with comorbid anxiety disorders,[7] under which OCD falls, and these factors may be both risk and maintenance factors for EDs.[2] Therefore it is worth examining OCD concepts to see if any will benefit treatment providers' understanding of patients with eating EDs.

## The Intersection of ED and OCD

Treasure et al. propose that EDs develop, "from an abnormality in emotional learning and memory processing related to food or cognitive representations of food in the form of weight and shape."[1] Negative food associations could be related to any of the following: traumatic experiences that involved food, weight, or shape; threatening information about food, weight, health; learning about food fears from others; and developmental factors. Schmidt and Treasure suggest that ED behaviors (especially restrictive AN) are a mechanism with complex defensive functions, which act to reduce external and internal threats.[2] Often the negative food experiences lead to *experiential avoidance,* which occurs "when the person is unwilling to remain in contact with particular private experiences (e.g. bodily sensations, emotions, thoughts, memories…) and takes steps to alter the form or frequency of these events and the contexts that occasion them."[2]

The glaring similarity between EDs and OCD is that patients report engaging in behaviors that are organized around an irrational belief, which result in avoidance of sufficient caloric intake.[7] The symptoms that manifest to cope with adverse experiences include several components that are similar to OCD and/or OCPD, such as: perfectionism and rigidity, meticulous attention to details, body checking, reassurance seeking, ordering, magic ritualizing, and terror of making mistakes.[2,8] For the individual with an ED this is the typical "black and white" thinking or other cognitive distortions that they may exhibit. A cognitive distortion that was originally associated with OCD but may be specific to eating pathology is *thought-shape fusion* (TSF),[8,9] which can be described as "a belief that merely thinking about eating a high-caloric food leads to perception of weight gain and moral transgression, as well as increased body dissatisfaction."[9] Specifically there is some research showing that patients with anorexia-restrictive subtype are less capable of diffusing TSF because of reduced ability to re-label pathological mental events as unusual.[9]

The longer an individual stays in a chronically starved state, the more unpleasant eating will be, as he/she encounters feeling bloated, nauseous, and overfull, thus eating further threatens the physical and emotional equilibrium.[2] When taken to an extreme, these often result in rituals regarding food, exercise, and weight (to name a few):[1]

- Precise planning: e.g. *"If I eat this, then I will run this long"*
- Magical/superstitious thinking: e.g. *"I need to carry, store and prepare my food separately from the food of other people in order to prevent calorie contagion."*
- Ritualized counting applied to cutting, biting, chewing; and of course calorie counting
- Food rituals: such as eating foods in a certain order, picking things apart, etc.
- Certain exercises before and after meals

Obviously there are several areas where the similarities between EDs and OCD can be seen. Over time these neural patterns set in and become the default steps to reduce anxiety and emotions. The factors that maintain the illness are often intensified by starvation and, conversely, begin to relent when the patient moves to a healthier weight range.[2] As Treasure et al.[1] eloquently put it: "Recovery therefore entails the unlearning of these conditioned responses, an extinction process which involves the production of new non-fear extinction pathways, as opposed to the erasure of old pathways."

## Exposure with Response Prevention

Exposure with Response Prevention (ERP) is a strategy that Treasure et al.[1] suggest which is commonly used in OCD treatment and occasionally in social anxiety treatment. Steinglass et al.[7,10] also found that using the ERP model helped decrease anxiety and increase caloric intake in

*continued on page 16*

## Putting the Eating Back...

*continued from page 15*

individuals with AN. ERP begins with identification of anxiety and feared consequences, and then moves on to exposing the client to the triggering experience that kicks off the obsessions (anxiety about calories, strong urges to engage in unhealthy compensation behaviors, etc.). The session is in a supportive setting where compulsions (running, purging, etc.) are not allowed, or at least put off for as long as possible. Instead, the intervention focuses on enhancing the client's awareness of physical and emotional symptoms, and articulation of fears. Over time and repeated exposure, the patient experiences habituation of anxiety and confirmation that the feared outcomes did not occur[7] (e.g. immediate obesity, loss of control, etc.). Often the client is encouraged to make a hierarchy list of foods or situations to tackle and start from the bottom and work their way up. These exposures could happen in a variety of ways, to name a few:

- Meal support
- Bringing challenge foods to sessions, eating it, and then sitting through the resulting emotions
- Not engaging in disordered eating behaviors or self-harm for a certain amount of days
- Self-care "vacations"
- Grocery store trips
- Cooking and eating meals

## Conclusions

A client's exposure can be as small as adding a teaspoon of margarine to dinner every day, or eating a taste of a binge food without eating the entire amount or purging afterwards. It is impossible for anxiety to continue at that initial sky-high level, but it means putting off the short-term gratification of yielding to the obsession-compulsive tug, and instead working for the long-term gratification of recovery. Easier said than done, but it can be done. Encourage a client to choose one challenge food for the week, or even a month if that is what it takes. Working toward recovery involves tackling those eating challenges, and they cannot be tackled without exposure. Practice makes progress, and progress will eventually lead to whatever the client's recovery will look like.

## About the Author

Kelsey N. Wallour is a member of BHN's PR team, and is a second year MS/DI student at the University of Tennessee, in Knoxville.

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# HOD Supports Member Research Participation

**Harriet Holt Cloud, MS, RD, FAND, DPG Delegate**

*Harriet H. Cloud, MS, RD*

BHN members received a Survey Monkey from our practice group in April asking for responses to a number of questions dealing with the Research Issue to be discussed at the House of Delegates (HOD) virtual meeting in May. As your delegate, I appreciated the 127 members who took the time to respond to the survey which was very important in my representation of you at this Virtual Meeting. The dialogue session this year was entitled "Engaging Members in Research".

In preparation for the Virtual Meeting, all delegates were asked to read the background papers concerning research and why there is so much interest in motivating all dietitians to get involved in some way. You may be wondering "why now" the need for research discussion.

During the HOD meeting at FNCE where we discussed "Nutrition Services Delivery and Payment: The Business of Every Academy Member", the need for outcome data to support increased reimbursement for nutrition services of the registered dietitian nutritionist and dietetic technician, emerged as an issue. There was great agreement that we must use research to demonstrate how RDNs and DTRs improve the nation's health through food and nutrition in order to advocate for increased reimbursement of nutrition services.

As the HOD moved toward the Spring Virtual Meeting the steps to evaluating what has been done in research efforts and what is needed for a viable program in the area of research were reviewed. Actually the process began 12 years ago when the HOD had a dialogue session and approved a motion for the Academy Research Committee to refine a research philosophy.

Here are a few significant dates on the Research Timeline:

- 2000 - Esther Myers, PhD RD appointed as the first Nutrition Scientist for the Academy
- 2001 - First HOD meeting dialogue session on research
- 2002 - RDs granted Medical Nutrition Therapy status in the treatment of Diabetes types 1,2, and gestational diabetes mellitus and non-dialysis kidney disease
- 2003 - Adoption of the Nutrition Care Process (NCP) and International Dietetics and Nutrition Terminology (IDNT)
- 2004 - Evidence Analysis Library (EAL)
- 2006 - HOD meeting on Nutrition Care Process
- 2009 - First HOD session on Evidence Based Practice, Esther Myers, PhD, RD named First Chief Science Officer.
- 2011 - Academy name change
- 2012 - Council on Future Practice, ACEND, CDR, NDEP, work individually and collaboratively envisioning a new model of entry level nutrition and dietetics practice with a greater emphasis on research
- 2013 - Academy of Nutrition and Dietetics Health Informatics Infrastructure (ANDHII) introduced to members in Houston at FNCE
- 2014 - ANDHII to be launched as a free member benefit; DPBRN marks 10th anniversary with 9 papers and 9 completed projects; EAL website revamp underway; and NCP working to be incorporated into the Electronic Medical Record.

Reviewing this history brings us up to the Virtual Meeting Dialogue Session. One of the major take home messages for me was the necessity to understand and recognize all of the initialized tools that have been developed. Of course the newest is ANDHII (pronounced Andy), a tool that promotes efficient and accurate use of the NCP and the corresponding IDNT, helps members track and report on patient outcomes, and collects RDN impact data for use in public policy and quality improvement research. ANDHII is a new website which you can access through the Academy website.

During the HOD meeting it became clear that delegates accept the concept that member involvement in research is an opportunity to support advocacy for the profession. Delegates were enthusiastic in encouraging more learning opportunities during educational preparation and entry into the profession along with professional development for the Academy members to build confidence in research participation. The HOD also identified the need to promote inter-professional collaboration in research to advance best practice and improve the nation's health through food and nutrition.

The survey results from participating BHN members indicated that reading journal articles is the main source of research for 80% of DPG members and over half of the respondents plan to contribute to research in the future; there was great agreement that research is needed in all four practice areas of BHN DPG; and from 10-20% of BHN members are currently participating in research projects and publishing.

This Virtual Meeting was a challenging session for this delegate and many other members of the HOD. I greatly appreciate the input you provide me through the surveys and e-blasts that we send. The more information you can provide related to the topics addressed by the HOD, the greater the impact this DPG can make. BHN DPG Delegate, Harriet Cloud, can be reached at harriet.h.cloud@gmail.com.

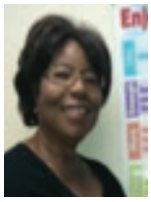
## Check out the *NEW* BHN Website!

Thanks to Website Coordinator/ Editor, Jacqueline Larson, MS, RD, BHN's web site has many new and exciting resources for members. Coming soon is a *Member Store* for BHN members to market their products. Also available soon, downloadable Fact Sheets addressing a wide variety of topics in BHN practice. Access the web site at **www.bhndpg.org**.

# Meet the BHN Resource Professionals!

**These highly experienced RDNs are available to serve you with resources and suggestions for the challenges faced in behavioral health nutrition.**

## Addictions Resource Professional



Jo Kannike Martins PhD RD
addictionsresourceprofessional@bhndpg.org

Josephine Kannike-Martins is the CEO/ Executive Director of Medi-Cure Health Services, Inc., a community based healthcare service provider working with low socio-economic population of South Los Angeles, treating patients with substance use disorders. She received her BS in Nutrition & Dietetics at California State University and MS in Food Science & Nutrition at Chapman University, Orange, California, followed by a Doctoral degree in Public Administration at the University of La Vern, California. Jo has worked as a Clinical Dietitian and Food Service Director at the Care Unit Behavioral Hospital, coordinated the Child Nutrition Program for Charles Drew University Head Start Program developing and building the Lela Hayward Nutrition Center, served as an adjunct professor at the School of Allied Health, teaching Food Science & Nutrition, and worked at the State of California Department of Education, Nutrition Services Division, as Child Nutrition Consultant. She took an early retirement after 13-1/2 years of service with the State of California to become an entrepreneur, with practice focus in Behavioral Health and Nutrition with emphasis in the field of addiction.

## Eating Disorders Resource Professional

Marci Anderson MS CEDRD LDN CPT
eatingdisorderresourceprofessional@bhndpg.org

Marci Anderson is the owner of a group nutrition practice in Cambridge, MA. She specializes in eating disorder recovery and is passionate about helping clients heal their relationship with food, exercise, and their bodies. She is a Certified Eating Disorder Registered Dietitian, a certified Intuitive Eating professional, and an ACSM certified personal trainer. In addition to individual client work and supervising the RDs on her team, Marci is an adjunct professor for Plymouth State University's Eating Disorder Institute. She has spoken nationally on the topics of eating disorder recovery, Intuitive Eating and the role of the RDN on the eating disorder treatment team. She loves social media so tweet her @marciRD. Most recently Marci developed a comprehensive training program for RDNs and dietetic students interested in treating eating disorders. She strongly believes in educating RDNs in the art of integrating therapeutic concepts into nutrition counseling. In the evenings and on weekends Marci happily takes a break from thinking about eating disorders and loves to read, teach exercise classes, eat delicious food, and travel.

## Intellectual / Developmental Disabilities Resource Professional

Patricia Novak MPH RD CLE
intellectualdevelopmentaldisabilities
resourceprofessional@bhndpg.org

Patricia Novak has 30 years experience working with children with autism, developmental disabilities and chronic illness. The common thread throughout has been addressing feeding issues from infancy through adolescence. Patricia received her bachelor's degree in Home Economics (yes, Home Ec!) from Humboldt State University and her Master's in Public Health in Nutrition at UCLA. She completed her dietetic internship at the University Center for Excellence in Developmental Disabilities (UCEDD) at CHLA and a fellowship at UCLA Neuropsychiatric Institute. Her clinical experience includes feeding programs at both Professional Child Development Associates (PCDA) and University Center for Excellence in Developmental Disabilities (UCEDD) at Children's Hospital Los Angeles (CHLA), the Adolescent Eating Disorders Clinic at UCLA Neuropsychiatric Institute, outpatient hospital clinics, school based California Children's Services (CCS) clinics, and Mother-baby/NICU lactation services. In addition to clinical services, Patricia has also been involved with developing nutrition education curricula and parent and professional training at the national, regional and local levels.

## Mental Health Resource Professional

Ruth Leyse Wallace PhD RD
mentalhealthresourceprofessional@bhndpg.org

Dr. Leyse-Wallace received her B.S. degree from the University of California at Davis as a member of Phi Kappa Phi; earned her M.S. degree while completing her dietetic internship at the University of Kansas Medical Center in Kansas City, and in 1998 was awarded her PhD from The University of Arizona in Tucson, Arizona. She began practicing clinical dietetics at Osawatomie State Hospital, followed by practicing at The Menninger Foundation in Topeka, Kansas from 1977 to 1984. She was later employed at Mesa Vista Hospital (now Sharp-Mesa Vista) in San Diego, California and HCA Willow Park Hospital in Plano, Texas. Her practice included providing nutritional care for patients of all ages hospitalized for eating disorders, alcohol and drug abuse, and general psychiatric diagnoses. While attending graduate school in Tucson, Dr. Wallace served at Sierra Tucson and Hospice Family Care in Tucson, as well as Group Health Medical Associates. She has served as an adjunct faculty member at Pima County College in Tucson and Mesa College in San Diego. A long-term member of The American Dietetic Association (ADA), She has been an active contributor to the Behavioral Health Nutrition dietetic practice group in the ADA, (now The Academy of Nutrition and Dietetics). Dr. Leyse-Wallace retired from clinical practice and has published three books: *Linking Nutrition to Mental Health* and *Nutrition and Mental Health* as well as a reader-friendly version of her doctoral dissertation *The Metaparadigm of Clinical Dietetics: Derivation and Applications*. She lives in Alpine, California in San Diego County and has three adult children and five grandchildren.

Email the Resource Professionals directly or even better, share the Q &A with other BHN members on our EML. Subscribe at membershipchair@bhndpg.org, include first and last name, email address, and please title the subject as BHN LIST SUBSCRIBE.

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**Contribute an article or topic for future BHN*newsletter* issues!**

Contact **newslettereditor1@bhndpg.org** or one of the BHN leaders listed in this newsletter.

## BHN: Fuel Your Brain, Feel Your Best!

**Mission:** Empowering BHN members to excel in the areas of Addictions, Eating Disorders, Intellectual and Developmental Disabilities and Mental Health by providing resources and support.

**Vision:** Optimizing the physical and cognitive health of those we serve through nutrition education and behavioral health counseling.

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