Nutrition and Pain Management: Putting Pain Relief on the Menu

Learning Objectives:
After reading this article, the nutrition professional will be able to:
1. Discuss three mechanisms that explain the relationship between nutrition and pain.
2. Describe the basic components of an anti-inflammatory diet and the contribution of each component to balancing inflammation levels in the body.
3. List three chronic pain conditions which have been shown to be directly related to nutrient insufficiencies.
4. Recognize the potential role of the microbiome in modulation of inflammation and pain.

Introduction and Context
In the current opioid epidemic, nonpharmacologic pain management is receiving unprecedented attention. In a widely reported clinical practice guideline, the American College of Physicians recommends trials of evidence-based nonpharmacologic modalities – acupuncture, exercise, cognitive behavioral therapies – before pharmacologic treatments for chronic pain.\(^1,2\)

Dietary interventions are noticeably absent from this list of evidence-based approaches to pain management.

Nutritional therapy for pain has been used in nonwestern systems of healing, such as Chinese Medicine and Ayurveda, for thousands of years. In modern pain management, we think of pain diagnosis in terms of identifying pain generators, such as an arthritic hip, a...
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damaged nerve, or a sprained muscle. We seek ways to repair structural damage, block discrete receptors, or inhibit specific pathways. A nutritional approach to pain is fundamentally different: instead of isolating one cause and correcting the corresponding culprit, the nutritional approach is global, in support of the whole organism’s innate ability to heal, and often works preventively.

There are economic factors that influence the lack of awareness of the benefits of nutrition for pain management. The financial return on investment for studying nutritional remedies for pain is low. An anti-inflammatory diet cannot be patented, and well-designed nutritional studies can be lengthy, laborious, and do not lead to the development of a new product. In addition, nutrition professionals are not always included as key members of the medical team. However, understanding the science and using current evidence to support greater awareness and advocacy will help put nutrition on the map for treatment of chronic pain.

Nutrient Insufficiencies

If we look for a nutrient insufficiency model to link pain and nutrition, there are a few micronutrients that stand out.

Magnesium is necessary for over 300 physiologic reactions, and magnesium deficiency is estimated to be widespread and rising in the US and Canada. Magnesium deficiency is associated with migraine headaches, and repletion is associated with prevention of headaches as well as treatment of acute migraine. Magnesium administration has been associated with relief of premenstrual syndrome (PMS) and fibromyalgia. Recommended oral dosages are 400-600 mg per day.

Vitamin C is important for bone healing and collagen crosslinking, as well as acting as an antioxidant.

In order to investigate whether Vitamin C may prevent the development of complex regional pain syndrome (CRPS), Zollinger et al followed a group of 317 patients with wrist fractures who were randomized to receive either 200, 500, or 1500 mg of vitamin C, and 99 patients who were randomized to receive a placebo. The prevalence of CRPS was 2.4% in the vitamin C group and 10.1% in the placebo group; all the affected patients were elderly women who may have been nutrient deficient. The prevalence of CRPS was 4.2%, 1.8%, and 1.7% in the 200, 500, and 1500 mg groups respectively. The optimal dosage of 500 mg has been confirmed by subsequent studies.

A group of Canadian researchers examined the associations between serum vitamin C concentration and the prevalence of spinal pain and functional limitations in over 4000 adults in the general US population using data from the US National Health and Nutrition Examination Survey (NHANES) 2003-2004. Suboptimal serum vitamin C concentrations were associated with the prevalence of neck pain and low back pain in the previous three months and related functional limitation score.

Vitamin C decreases the requirement for opioid analgesics, especially post surgically, and may be effective for cancer pain and for pain associated with chronic pancreatitis.

Good evidence exists for the use of intravenous alpha lipoic acid for diabetic neuropathy, and some studies show a benefit of an oral dose of 600 mg with and without vitamin B12. Preliminary evidence exists for a preventative effect of Vitamin D and B vitamins on pain of various etiologies, but there is not enough evidence to make clinical recommendations at this time.

Nutrient groups such as phytonutrients and essential fatty acids are best discussed within the context of dietary eating patterns (below).

Pain and Inflammatory Load

Nearly all pain has a component of inflammation. This can be profoundly influenced by diet. A pro-inflammatory diet, such as the Standard American Diet (SAD), contains several elements that promote systemic inflammation, and there are a number of diets that are considered anti-inflammatory. Below is a table comparing the dietary components that determine the degree of inflammation contributed by diet.

Fat balance. Dietary fat is one of the most important determinants of the inflammatory load. The content of dietary fat will determine the essential fatty acid (EFA) balance that is metabolized by the EFA pathway. The essential fats linoleic acid (LA) and alpha linolenic acid (ALA) are called essential because they are not manufactured in the body and must come from diet.

The pro-inflammatory omega-6 pathway begins with LA and produces arachidonic acid, the precursor to pro-inflammatory cytokines, prostaglandins, thromboxane, and leukotrienes. Prostaglandins sensitize peripheral nociceptors (pain receptors), while leukotrienes increase vascular permeability. The cytokines produced by the pro-inflammatory pathway are responsible for multiple aspects of the inflammatory process, including the initiation and persistence of pain. These proteins can sensitize nociceptive neurons, contribute to central sensitization, and influence the development of hyperalgesia and allodynia.

The anti-inflammatory omega-3 (ω-3) pathway begins with ALA and produces eicosapentaenoic acid and docosahexaenoic acid, which give rise to anti-inflammatory prostaglandins and resolvins, which balance the effects of the inflammatory response.

Table 1: Pro-inflammatory and anti-inflammatory dietary components

<table>
<thead>
<tr>
<th>Pro-inflammatory Diet</th>
<th>Anti-inflammatory Diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pro-inflammatory balance of fats; trans fats</td>
<td>Anti-inflammatory balance of fats</td>
</tr>
<tr>
<td>Low in phytonutrients</td>
<td>High in phytonutrients/spices</td>
</tr>
<tr>
<td>Low fiber</td>
<td>High fiber</td>
</tr>
<tr>
<td>High glycemic load</td>
<td>Low glycemic load</td>
</tr>
<tr>
<td>High in animal protein</td>
<td>Low to moderate animal protein</td>
</tr>
<tr>
<td>Highly processed</td>
<td>Basic preparation</td>
</tr>
</tbody>
</table>

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Since both pathways use the same enzymes, the balance of pro- vs anti-inflammatory fat intake determines the character of the inflammatory response. In the prospective INCHIANTI study, blood levels of ω-3 fatty acids were associated with decreased incidence of peripheral neuropathy in the elderly, which can be extremely painful.22

Antioxidant Phytonutrients.

Chronic inflammation produces free radicals, which can deplete stores of antioxidants and lead to a state of oxidative stress, or “inflammaging.” Certain nutrients, such as magnesium and zinc, are cofactors in key enzymatic reactions that dismantle free radicals. Others, such as vitamin C and phytonutrients, are dietary antioxidants that quench oxidation. When co-factors and co-enzymes are deficient, oxidation increases.21

We know that fruits and vegetables, herbs and spices, and green tea contain bioactive compounds such as flavonoids, carotenoids and proanthocyanidins. These compounds reduce the oxidative stress that results from chronic inflammation. There is evidence that soy foods may contribute to pain reduction.23 Data from the British Cohort study, which follows children from birth, revealed an association between a decreased intake of fruits and vegetables and Chronic Widespread Pain (CWP).24 Pain scores of patients with knee pain decreased significantly with increased intake of fruits and vegetables,25 and patients with osteoarthritis experienced decreased pain with a whole foods, plant-based diet.26 Fibromyalgia has been shown to be responsive to vegetarian diets27,28 and a higher consumption of fruits and vegetables has a protective effect for gout.29 Isolated phytonutrients have been associated with decreased post-surgical pain,30 reduced lower extremity pain,31 and less exercise-induced muscle soreness.32 There is a robust literature devoted to the anti-inflammatory properties of green and black tea as well as many herbs.

Figure 1: Essential Fatty Acid Pathway

With permission, courtesy Adam Rindfleisch MD/ University of Wisconsin Integrative Health/ Veteran’s Health Administration Office of Patient Centered Care and Cultural Transformation

Fiber and Glycemic Load.

Carbohydrate foods influence the inflammatory process. There is a correlation between post-prandial glucose concentration, oxidant stress, and inflammation.33 Fiber in the diet slows absorption time of sugars and helps to optimize glucose metabolism, which avoids a high glycemic load and an insulin-driven rise in C-reactive protein (CRP).34,35 Some types of fiber act as prebiotics and support growth of symbionts (helpful bacteria) in the intestine, promoting a diverse microbiome and balanced immune response. The degree of “westernization” of a diet is associated with a decrease in microbial diversity, which includes organisms that process fiber-rich dietary components.36 Reviews of the data of two prospective studies – the Osteoarthritis Initiative and the Framingham Offspring Osteoarthritis Study – show that dietary fiber was significantly and inversely associated with worsening of knee pain in people with osteoarthritis.37 An analysis of more than 15,000 NHANES participants showed significant associations between systemic inflammation,
nutritional approach to chronic pain. Although the evidence base is not complete, there is data that shows relationships at the level of single nutrients as well as dietary patterns.

Our success in establishing dietary intervention as fundamental to pain management depends in part on our ability to guide health care practitioners and patients to recognize its role and to understand the nature of its contribution. In this author’s experience, it is helpful to guide patients and their referring clinicians about the theoretical basis for this approach, as well as the scope, timeline, and potential limitations of using nutrition for pain management. In general, this author recommends that nutrition be recommended as part of a long-term strategy for managing chronic pain, instituted gradually while shorter-acting therapies (e.g., acupuncture, medication, manual therapies) are used to provide pain relief in the near term. As patients experience pain relief with other modalities, they are more able to make changes in dietary habits.

It is helpful to provide some guidance for patient expectations. For an anti-inflammatory diet, improvement (increased function or decreased need for medication) will often be noticed in 3-6 weeks. For patients who may be reacting to specific foods, partial pain relief may be evident in a shorter time period after such foods are removed. Although dietary change is potent, it is rarely the only tool needed for relief from chronic pain, and is best used as part of a multi-modality approach. In this author’s experience, it potentiates the success of other modalities.

Nutrition intervention for chronic pain care is not yet as widespread as it is for other chronic conditions, such as cardiovascular disease and diabetes. Reasons for this include lack of training on the part of providers, market forces, and time pressure in the office. As we better understand the relationship between dietary patterns, the microbiome, immune modulation, and pain, the contribution of nutrition to pain prevention and treatment will become more evident. By educating both patients and providers about the powerful role nutrition can play, registered

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dietitian nutritionists can take the lead in bringing dietary interventions to the forefront of chronic pain management.

References


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**Small Intestinal Bacterial Overgrowth: Impact on Mental Health and Implications for Nutrition Care**

by Joanna R. Pustilnik, MS, RDN, LDN, CDE, CLT, ACE-CPT, ACE-HC

Small intestinal bacterial overgrowth (SIBO) is an increase in the number or type of bacteria in the small intestine to a degree that resembles the colonization of the colon. Symptoms range from subtle to severe and can be unappreciated in the clinical setting. Estimates are that it may affect up to 22% of the general population. SIBO also commonly co-occurs with irritable bowel syndrome (IBS) and, in 60% of cases, is thought to be a causal factor in IBS development. Risk factors for SIBO include low stomach acid; deficient pancreatic enzymes; nutrient deficiencies or disease affecting the immune response; inflammation caused by food poisoning, food allergies or intolerances; a high inflammatory diet which can destroy the gut mucosa; hepatic disease and gut dysmotility. Of these, gut dysmotility- prolonged small intestinal transit time (ITT) or dysfunction of the migrating motor complex (MMC)- is typically considered the primary cause of SIBO.

The MCC can be described as the street sweeper of the gut. When in a fasted state, the MCC sends waves of contractions every 90-120 minutes which clean out food debris and bacteria lingering in the small intestine. Following a meal, normal ITT is about 2.5 to 3 hours. Longer transit times may...
The small intestine typically houses small numbers of gram-positive bacteria and the large intestine houses gram-negative bacteria, but in SIBO, the bacterial composition of the typically more sterile small intestine closely resembles that of the bacteria-laden colon. This can cause leaky gut as gram-negative bacteria release an endotoxin called lipopolysaccharide (LPS) which impacts intestinal brush border function causing bacteria to leak into systemic circulation, causing peripheral and neuronal inflammation.

Neuroinflammation can reduce brain-derived neurotrophic factor (BDNF). BDNF is an important determinant of neuronal functioning with reduced levels being associated with greater risk of depression and anxiety and decreased neuroplasticity. Bacterial overgrowth in SIBO may also affect the brain through alterations in GABA, a main central nervous system inhibitory neurotransmitter. Changes in GABA expression have been implicated in both depression and anxiety etiology.12,13

A 2016 study evaluated whether a fecal transplant from a group of 34 patients with major depression could transfer depression-like behaviors to rats with a reduced microbiota due to antibiotics. Matched healthy control transplants were used for comparison. Inflammatory and stress markers (cytokines, C-reactive protein, salivary cortisol), diversity and abundance of microbiota, and intestinal transit time were measured in both humans and rats before and after transplant. Following the transplant, the depression animal model exhibited anhedonia and increased anxiety behaviors as well as altered tryptophan metabolism (a precursor to serotonin), a decrease in bacterial diversity and richness, and a statistically significant increase in ITT.14 Studies such as this further suggest a connection between depression, the microbiome, and gut motility, but the overall picture is still emerging and more research is needed.

Inflammation and oxidative stress may also independently influence the development of SIBO. One study investigating the relationship between oxidative stress, SIBO, ITT, and type 2 diabetes melitus found positive correlations between oxidation and elevated A1c and oxidation and SIBO and a negative correlation between oxidation and ITT, indicating the more oxidation, the longer the ITT. There was also a prevalence of SIBO of 14.8% among type 2 diabetes patients vs only 2.8% in the control group. The increased prevalence of SIBO in diabetes may not merely be a result of gastroparesis and slowed transit, but inflammation and oxidation could also play a causal role in bacterial overgrowth and slowed digestion.

**Criteria for SIBO Diagnosis**

**Pre-test considerations:**
- Antibiotics to be avoided x 4 weeks prior
- Promotility agents and laxatives avoided x 1 week prior
- Fermentable foods such as complex carbohydrates avoided x 1 day
- 8-12 hour fast needed
- No smoking the day of test

**Consenus doses for breath test substrates:**
- Lactulose: 10 gm
- Glucose: 75 gm

**Breath test:**
- Administer substrate dose with 1 cup of water
- Collect breath every 15 minutes for at least 3 hours

**Positive Diagnosis if:**
- Rise in hydrogen is > 20 ppm over 90 minutes
- Rise in methane > 10 ppm over 90 minutes

**Diagnosis of SIBO**

There has been a lack of standardized testing and interpretation of results for the diagnosis of SIBO. A recent consensus paper published in the American Journal of Gastroenterology evaluated 13 case-controlled studies and found 13 different methodologies. Through collaborative expert consensus, however, they determined doses for breath test substrates and criteria for positive results, as well as pre-test considerations (see Figure 1). Current consensus favors the diagnostic breath test as less invasive and costly than retrieving duodenal aspirates. If duodenal aspirates are tested, however, the threshold should be 103 cfu/mL (colony forming units) for diagnosis.

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Of the intestinal gases, hydrogen and methane are exclusively produced by microbial fermentation of carbohydrates and are therefore the focus of breath testing for SIBO.18 For greater accuracy, measuring both hydrogen and methane is recommended because certain bacteria are methane-producing while others produce more hydrogen. The hydrogen-producers induce a more rapid ITT and thus are more likely to cause diarrhea. Alternatively, overgrowth of methaneproducing bacteria, which occurs in approximately 35% of patients, typically induces constipation due to longer ITT.19

If SIBO is suspected in a patient due to bloating, constipation, depression, or other SIBO-related symptoms, it is important to question about previous testing and encourage a gastroenterology appointment where a breath test can help determine the presence of overgrowth.

Probiotic and Antibiotic Treatment

Another area of SIBO-focused research involves the potential role of probiotics in both competing with pathogenic, inflammatory bacteria as well as possibly decreasing ITT. One small study of 14 patients with IBS administered Lactobacillus casei for 6 weeks, found 64% no longer met criteria for SIBO.20 In a recent meta-analysis and systematic review assessing the role of probiotics in the prevention and treatment of SIBO, 18 studies were analyzed, and a pooled SIBO decontamination rate of 62.8% was found in the probiotic treatment group.21 Significant reductions in hydrogen levels and symptoms such as abdominal pain were also found, but stool frequency was unchanged indicating probiotics may not impact ITT as much as influencing a favorable gut microbiome composition.

New evidence suggests probiotics may also be more beneficial in patients with IBS and co-occurring SIBO than in patients with IBS only. In a prospective study, researchers compared patients with IBS and SIBO to those with IBS without SIBO using a probiotic formula of 4 strains for 30 days- [Saccharomyces boulardii, bifidobacterium lactis, Lactobacillus acidophilus, and Lactobacillus plantarum].22 Compared to the IBS without SIBO group, which only noted a 10.6% reduction in IBS symptoms, the SIBO group had a 71.3% reduction in severity of IBS-related symptoms. Patients with higher inflammation, as determined by cytokine activity in duodenal fluid, had greater decreases in symptoms indicating probiotics may decrease inflammation that leads to SIBO. More research is needed to determine optimal probiotic strains and doses, but evidence is promising.

Antibiotics are often the first line of treatment for SIBO and have been associated with improved depressive symptoms and restoration of gut lining, but complete resolution of SIBO is not always achieved.1 In a 2017 systematic review and meta-analysis examining the effectiveness of rifaximin in SIBO, 67.7% of patients had symptom resolution, but authors noted the studies were not well designed.23 Yet, in a 2019 well-designed, double-blind, placebo-controlled trial of Gulf War veterans suffering from IBS and SIBO, no significant improvement above placebo was found after 2 weeks of antibiotic treatment.24 In another trial, average symptom relief only lasted 22 days.25 Prokinetic agents such as Octreotide may be effective in addition to antibiotic treatment as a way to concurrently address slow ITT.26 More research is needed to determine optimal doses and treatment combinations.

Nutritional Consequences and Medical Nutrition Therapy

Fat malabsorption and diarrhea are common in SIBO and can cause deficiencies in fat-soluble vitamins (A,D,E,K).26,27 Overgrowth leads to bacterial competition for vitamin B12. Inefficient activation of B12 due to low stomach acid or decreased absorption resulting from a damaged ileum mucosa is also common. Folate excess may develop due to increased synthesis of folate by gut bacteria. Carbohydrate and protein malabsorption are also common, leading to further deterioration of the gut mucosa and the hallmark bloating and cramping seen in SIBO. Vitamin repletion is necessary and supplementation with nutrients such as glutamine, zinc, and magnesium may also protect gut mucosa. Supplementing with digestive enzymes can replenish brush border enzyme activity and, if achlorhydria is an underlying cause of SIBO, supplementing with betaine HCL can also be beneficial.27 Avoidance of proton pump inhibitors, which are associated with a significant increase in SIBO, is also recommended.1

The optimal diet to prevent or treat SIBO is still unknown, and an individualized approach is warranted. One dietary strategy is to decrease the frequency of eating occasions as snacking every couple of hours inhibits the gastrointestinal cleansing MMC waves that occur during fasting.27 A diet rich in Omega-3 fatty acids protects against gut inflammation, and some studies show a high fiber, plant-based diet can increase beneficial bacteria while a high-fat, meat and dairy-laden diet is associated with an elevation in LPS endotoxin activity and constipation.28 Prebiotics such as fructo-oligosaccharides in garlic, onions, artichokes, bananas, and legumes can be helpful in promoting growth of beneficial bacteria, but some animal research suggests they may instead increase ITT.29

Overall, research points to a whole foods, plant-based diet with high fiber foods to add bulk and promote gut peristalsis. In a study of 126 patients, 66 were found to have SIBO, and functional bacterial pathways were measured. The most symptomatic patients had higher levels of activated simple sugar pathways vs healthy controls who exhibited more prevalent, active complex carbohydrate functional pathways. This suggests, although is not enough evidence to prove causality, that higher sugar intake may encourage SIBO, yet in this study, some patients consuming a high fiber diet were positive, via duodenal aspirate, for SIBO despite reporting no or few symptoms. An interventional study was done as a follow up, and 16 of the SIBO-positive patients who previously ate a high fiber diet (>22 gm/day diet) were provided a low fiber, high

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sugar (<10 gm fiber, >50% carbohydrates) diet for 7 days. On the lower fiber diet, 80% developed new symptoms (bloating, pain, diarrhea). Returning to a higher fiber diet resolved these symptoms within a week.30

Increasing fiber by no more than 2-4 gm per day allows gut bacteria time to adjust to the altered diet and may decrease incidence of bloating and gas. Malabsorption of fructose and lactose may be a direct consequence of SIBO so avoidance of these sugars until resolution of SIBO is achieved via antibiotic treatment may improve symptoms.31

It is wise to evaluate SIBO with a whole-person, functional and individualized approach. Addressing stress may decrease IIT as psychological stress has been shown to negatively impact transit time and intestinal permeability in mouse models.32 Mindful practices, such as mindful eating, may help speed up digestion by optimizing the gut-brain response to feeding. Also, exercise should be encouraged. High intensity exercise may help increase metabolism and blood perfusion to the gut thereby increasing gut peristalsis and speeding transit.33

Future Directions
Further research is needed to fully understand the etiology and best treatments for SIBO as well as the effects of SIBO on depression risk. The gut-brain axis is an exciting area of study involving how gut peptides and bacteria communicate with the brain, potentially causing a myriad of mental health symptoms, but much of the research remains preclinical.34 More translational research in the form of well-designed human studies, specifically evaluating the impact of SIBO on mental health, are necessary to assist in elucidating the complex dynamic between our brain, body, and bacteria.

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References:
Understanding the Nutritional Impact of Psychotropic Drugs on the Gut Microbiota

by Jennifer Messineo, MS, RD, CDN

Introduction
The human microbiome is colonized by thousands of complex strains of bacteria that have both synergistic and antagonistic effects on the host. The role of the microbiome in health and disease is a growing area of interest in research and clinical medicine. The term microbiota refers to the populations of bacteria that inhabit different ecosystems of the body such as the mouth, skin and intestines; while the term microbiome is defined as all organisms living in the body and their genetic material. The microbiome is unique to the individual and changes throughout the lifespan, although early life appears to have the most significant impact on its development. There are several contributing factors to the development of the microbiome, which is thought to be first colonized at birth. The neonate exposed to the skin microbiome during Cesarean delivery compared to the neonate exposed to the skin microbiome during vaginal delivery. Other factors are prematurity, breastfeeding, the presence of pets, parental smoking, maternal age, weight status, and race. Diet is another factor that significantly impacts microbiota composition throughout the host’s lifespan.

The GI tract is inhabited by a complex ecosystem that contains more than 10 times the number of microorganisms than cells in the human body and 150 times more genes than the human genome. The interaction between the brain and the digestive system has been recognized since the nineteenth century with a primary emphasis on the brain’s role in modulating gut function. It is now understood that the gut-brain axis provides direct and indirect, bidirectional communication between the brain and gut through endocrine (cortisol), immune (cytokines) and neural (vagus and enteric nervous system) pathways. This suggests that microbiota influences brain function, and alternatively, the impact of the brain on the microbiota affects the gastrointestinal tract.

Changes in the gut-brain axis are associated with anxiety and depression as well as with alterations in the stress response and behavior. Mental disorders such as depression affect 300 million worldwide and 10% of Americans are on antidepressants. Certain pharmaceuticals, specifically psychotropic drugs, affect the composition of the microbiome. It is unclear whether these changes are a result of the effect of changes in the brain on the gut microbiota or the direct effect of the medication on the gut microbiota.

Alterations in gut microbiota are associated with metabolic dysfunction, weight gain, GI disorders and possibly increased epithelial permeability, although evidence is conflicting. Certain psychotropic drugs, such as antidepressants and antipsychotics, are associated with weight gain, which is associated with alterations in the gut microbiota. Evidence suggests that metabolic dysfunction may be the result of altered gut microbiota composition and not a direct effect of the psychotropic drug. The mechanism has not been established; however, the antimicrobial activity of some psychotropic medications, similar to that of antibiotics, appears to play a role.

Antimicrobial effect of psychotropic drug therapy on gut microbiota
Bactericidal activity has the ability to disrupt the gut microbiota by depleting “good” commensal bacteria resulting in decreased host resistance to opportunistic, pathogenic microbes, which may have long-term effects on host function and health. Psychotropic drugs have an antimicrobial effect on gut microbiota similar to that demonstrated with antibiotic use. Maier et al. (2018) examined the antibacterial effects of 835 human-targeted drugs against 40 bacterial strains found to colonize the GI tract in vitro. The study found that 24% of the human-targeted drugs inhibited the growth of at least one species, and antipsychotics were one of the classes of drugs that exhibited the greatest antibacterial activity. In another study, Cussotto et al. (2018) examined the antimicrobial effects of psychotropic drugs against two strains, L. rhamnosus and E. coli, which reside in the human gut. Selective serotonin reuptake inhibitor (SSRI) antidepressants (Escitalopram and Fluoxetine) inhibited growth of E. coli in vitro.

SCFAs acetate, propionate and butyrate, produced by microbial fermentation of non-digestible fibers in the cecum and colon, provide an additional energy source for the host. SCFAs act as signaling molecules that modulate several physiological processes such as metabolism, behavior and CNS function. Butyrate and propionate activate intestinal gluconeogenesis, which has a beneficial effect on regulating glucose and energy homeostasis. De Vadder et al. (2014) identified the induction of gluconeogenesis by SCFAs as a key mechanistic rationale by which the production of SCFAs, particularly propionate, by microbial fermentation of dietary fibers, may have a beneficial effect on host metabolism. In another animal study, sodium butyrate, a histone deacetylase inhibitor, exerted an antidepressant effect particularly when combined with the SSRI fluoxetine. Taken together these findings suggest that the antibacterial effect of psychotropic drugs is associated with...
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changes in the composition of gut microbiota, and subsequently altered levels of SCFAs (as a result of microbial dysbiosis) may impact weight, appetite, and mental health. (See tables 1 and 2.)

Weight gain and psychotropic drugs

Energy intake and expenditure

The cause of antidepressant-associated weight gain is unclear, although it is correlated with increased energy intake and decreased physical activity. Evidence supports that obesity is associated with microbial changes in the gut and there is recent evidence that gut microbiota may play a role in weight gain associated with psychotropic drugs. In a retrospective cross-sectional analysis, Jensen-Otus and Austin (2015) assessed the energy intake and physical activity of antidepressant users with data from the National Health and Nutrition Examination Survey (NHANES).

‘Obesogenic’ bacterial composition

The most prominent bacteria in the gut are the phyla Firmicutes and Bacteroidetes, which make up 70-75% of the gut microbiota with a Firmicutes/Bacteroidetes ratio of 10.9 in healthy adults. It is thought that the gut microbiota of individuals predisposed to obesity may be more efficient at extraction/storage of energy compared to the gut microbiota of lean individuals. The composition of microbiota associated with obesity is increased Firmicutes and a proportional decrease in Bacteroidetes. A decrease in the Firmicutes/Bacteroidetes ratio is consistent with the ratio observed in humans with weight loss and after bariatric surgery. Treatment with the antipsychotic Olanzapine was associated with weight gain on a high-fat diet in an animal study examining the interaction of gut microbiota with Olanzapine. The rate and amount of weight gained differed between the eight inbred strains of animals indicating a likely genetic component. Obese mice had an increase in Firmicutes and a decrease in Bacteroidetes compared to the lean mice. Evidence of the microbial impact on weight gain is strengthened by a correlation of the relative abundance of bacteria from the phylum Firmicutes with accelerated weight gain. Bahr et al. (2015) found that mice treated with risperidone had a 32.2% increase in relative abundance of Firmicutes and a 22.4% decrease in relative abundance of Bacteroidetes, similar to results observed with Olanzapine. However, these results are inconsistent with recent studies that failed to demonstrate a correlation between the Firmicutes/Bacteroidetes ratio and BMI, suggesting

Table 1. Key Terms

<table>
<thead>
<tr>
<th>Key Terms</th>
<th>Definition</th>
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<tbody>
<tr>
<td>Microbiota</td>
<td>Microbial population present in different ecosystems in the body (ex: skin and gut)</td>
</tr>
<tr>
<td>Microbiome</td>
<td>Total number of microorganisms and their genetic material</td>
</tr>
<tr>
<td>Antimicrobial</td>
<td>Prevent the growth of intestinal pathogens</td>
</tr>
<tr>
<td>Probiotics</td>
<td>Live cultures of beneficial bacteria</td>
</tr>
<tr>
<td>Prebiotics</td>
<td>Dietary fibers fermented by indigenous good bacteria in the large intestine</td>
</tr>
<tr>
<td>Short Chain Fatty Acid (SCFA)</td>
<td>Produced by microbial fermentation of prebiotics in the large intestine</td>
</tr>
</tbody>
</table>

Table 2. Possible Nutritional Impact of Common Psychotropic Medications

<table>
<thead>
<tr>
<th>Medication</th>
<th>Class</th>
<th>Main Medical Use</th>
<th>Possible Nutritional Impact</th>
</tr>
</thead>
<tbody>
<tr>
<td>Olanzapine</td>
<td>Second-generation antipsychotic</td>
<td>Treats positive symptoms of psychosis and prevents future relapse (e.g., schizophrenia)</td>
<td>May increase appetite and weight; dry mouth, dyspepsia, constipation</td>
</tr>
<tr>
<td>Risperidone</td>
<td>Second-generation antipsychotic</td>
<td>Autism, bipolar disorder and schizophrenia</td>
<td>May increase appetite and weight; constipation</td>
</tr>
<tr>
<td>Escitalopram</td>
<td>SSRI antidepressant</td>
<td>Major depressive disorder, generalized anxiety disorder</td>
<td>May increase appetite and weight; dry mouth, nausea</td>
</tr>
<tr>
<td>Fluoxetine</td>
<td>SSRI antidepressant</td>
<td>Major depressive disorder, generalized anxiety disorder</td>
<td>Anorexia, nausea, dyspepsia, diarrhea, constipation</td>
</tr>
<tr>
<td>Venlafaxine</td>
<td>SNRI antidepressant</td>
<td>Major depressive disorder, generalized anxiety disorder</td>
<td>Anorexia, decreased weight, dry mouth, nausea, constipation, increased cholesterol</td>
</tr>
<tr>
<td>Lithium</td>
<td>Mood stabilizer</td>
<td>Bipolar disorder, mood stabilizer, major depressive disorder, schizophrenia</td>
<td>Increased thirst and weight; dry mouth, nausea, vomiting and bloating, diarrhea</td>
</tr>
<tr>
<td>Valproate</td>
<td>Anticonvulsant</td>
<td>Epilepsy, bipolar disorder, schizophrenia</td>
<td>Increased appetite and weight; nausea, vomiting, dyspepsia, diarrhea, decreased vitamin D</td>
</tr>
<tr>
<td>Aripiprazole</td>
<td>Atypical Antipsychotic</td>
<td>Schizophrenia, major depressive disorder, bipolar disorder, obsessive compulsive disorder</td>
<td>Increased weight</td>
</tr>
</tbody>
</table>

SSRI = Selective serotonin reuptake inhibitors; SNRI = Serotonin-norepinephrine reuptake inhibitors

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that the amount of SCFA and Firmicutes and Bacteroidotes subgroups may be more important than the Firmicutes/Bacteroidotes ratio.37,38 Members of both Firmicutes and Bacteroidetes produce SCFAs from indigestible dietary compounds; subsequently, changes in concentration and proportion of individual SCFAs are concurrent with changes in gut bacteria.38 A study examining differences within the human gut microbiota and fecal SCFA concentration found that total amount of SCFA was higher in subjects with a BMI >30kg/m² and the proportion of propionate was significantly higher in those with a BMI >25kg/m².38 Leaner subjects had a higher ratio of acetate to butyrate and propionate.38 In summary, gut microbiota impacts energy intake and expenditure and level of SCFA of the host, and the observed increased ratio of Firmicutes/Bacteroidetes is correlated with obesity with some conflicting evidence.

Dietary modulation of the microbiome

Introduction of prebiotics (non-digestible plant fibers) and probiotics to the gut have been shown to restore microbial composition.39,40 Probiotics are beneficial bacteria that inhabit the gut while prebiotics are food for the probiotics. In a recent study, treatment with a probiotic, BimunoTM galacto-oligosaccharides (B-GOS) significantly attenuated olanzapine-induced weight gain in rats by altering the gut composition.41 The weight modulating effects of Lactobacillus gasseri (LG2055) were examined in a randomized controlled trial with healthy adults with a BMI between 24.2 and 30.7 kg/m² and abdominal visceral fat area between 81.2 and 178.5 cm².39 The participants were given a fermented milk prepared with a mixture of 11% skim milk, lactic acid bacteria starter cultures and LG2055.39 After 12-weeks, they experienced significant reductions in abdominal visceral and subcutaneous fat, body weight, BMI, waist to hip circumference, and body fat mass.39

Dietary prebiotics increase microbial diversity, although they are a less targeted approach to modifying gut composition. While current evidence to support a predominantly plant-based diet as treatment for gut dysbiosis is lacking, a favorable increase in Bacteroidetes and SCFAs levels are correlated with intake of the Mediterranean diet (high in whole grains, legumes, nuts, fruits and vegetables with a moderate amount of fish and poultry and limited red meat).3,43 In contrast, a diet high in fat and sugar is associated with the Firmicutes/Bacteroidetes ratio which is typically associated with obesity.42 Potential mechanisms for the dysbiosis associated with high-fat-diet-induced weight gain include: (1) an improved capacity to extract and store energy and; (2) increased gut permeability and inflammation.42

Clinical Practice

The majority of studies in the literature examining the impact of dietary components on gut microbial diversity focus on diet composition and probiotics and prebiotics. However, studies investigating clinical application in the modulation of gut microbiota are lacking. There is preliminary evidence that some food components may reduce gut permeability and enhance protection against the effects of antimicrobials.44 Flavonoids found in fruits, vegetables, coffee, tea and other sources affect the expression and surface location of tight junctions found on cell surfaces, impacting gut permeability.44 In an animal study, quercetin, which is found in apples and onions, was shown to increase the total population of gut bacteria, although more studies are needed to assess a greater number of bacterial groups and the impact of a high-flavonoid diet on microbiota.45 Dysbiosis influenced by diet pattern, alcohol and psychotropic drugs may result in increased epithelial wall permeability, subsequently causing “leaky gut.”46 Zinc has the potential to modify and enhance epithelial barrier function, although evidence of the impact of zinc on gut permeability has not been established.44 As reviewed by Bischoff et al., vitamin A has been shown to regulate the integrity of intestinal cells, while a vitamin A deficiency is associated with alterations in the microbiota and an impaired intestinal barrier.47 Vitamin D and histone deacetylase inhibitors from dietary intake of sulforaphanes (cruciferous vegetables) and bacterial metabolism (butyrate) enhance barrier protection of the cell.44 In an animal study, Assa et al. found that vitamin D deficiency resulted in altered gut microbiota, which is implicated in increased gut permeability.48 As reviewed by Jayachandran, Xiao, and Xu, mushrooms act as prebiotics, stimulating the growth of gut microbiota.49 White button mushrooms increase microbial diversity and Ganoderma lucidum (GL) variety has been reported to reduce weight in mice by modulation of gut microbiota composition.49 In that study, GL decreased the Firmicutes/Bacteroidetes ratio reversing dysbiosis.49 The composition of microbiota found with GL is consistent with results of human and animal studies, showing a reduced Firmicutes/Bacteroidetes ratio in lean subjects.21,35,36 Taken together, the preliminary findings suggest that these diet components may protect against gut permeability, reverse dysbiosis and increase the diversity of gut microbiota. (See table 3)

Conclusion

The microbial component of weight changes associated with psychotropic drugs has potential therapeutic implications. While treatment with probiotics is promising, diet composition is a significant modifiable factor modulating the composition of gut microbiota throughout the lifespan. One important finding is that microbial changes induced by the psychotropic drug Olanzapine are similar to the effect of a high-fat diet alone.21 There is a strong correlation between changes in the microbiome and weight, although causation has not been established. However, diet, antibiotics (as well as other drugs with antibacterial activity), and effects related to the genetics of animal models may be confounding factors.32 The growing interest in the relationship between the microbiome and the host shifts the clinical focus to a more patient-centered approach of targeting the microbiome.

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Understanding the role of microbiota in metabolism and behavior could enhance our understanding of the RDN’s role in the treatment of behavioral health. However, more studies are needed to examine the interaction between genetics, diet, and the human gut microbiota that could lead to the development of evidence-based practice to modify the gut microbiota with diet, prebiotics or advanced probiotics.

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35. Ley RE, Bäckhed F, Turnbaugh PJ, et al. The gut microbiota as a}
Empowering the Dietitian in Eating Disorders Counseling: Psychological Theory and Counseling Techniques to Support Lasting Behavior Change

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Abstract
Given that the Registered Dietitian Nutritionist (RDN) is at the forefront of promoting behavior change, increasing knowledge of and exposure to behavior change and counseling techniques is vital. Working in the field of eating disorders (ED) requires a counseling skillset that simultaneously addresses the nutritional needs as well as the underlying psychopathology of the client. The following will introduce the RDN to five types of psychological modalities, provide ED specific examples and counseling vignettes to illustrate counseling techniques, and provide information for further study.

Table 1, Psychological Modalities and Resources, summarizes the psychological modalities and corresponding resources that will be discussed.

Introduction
According to The Commission on Dietetic Registration (CDR), a core competency for the RDN includes utilizing “effective counseling and coaching skills and strategies in practice.” This competency is defined by the performance indicator 9.6.1; the RDN “determines and applies counseling theories, psychological methods and strategies that empower customers to make changes.”

The Academy of Nutrition and Dietetics advocates that RDNs integrate behavior change counseling into their dietetics practice. The following counseling modalities and techniques will focus on their use in (ED), however these counseling techniques can be used regardless of client diagnosis and treatment setting. Developing the skills to utilize a variety of therapeutic counseling techniques increases the RDN’s confidence and capacity to join with the client in facilitating lasting behavior change.

Cognitive Behavioral Therapy (CBT)
Traditional CBT asserts that thoughts, feelings, and actions are related to and influence one another. In its simplest form, CBT claims that changing maladaptive thinking leads to a change in affect and in behavior. As individuals challenge problematic ways of thinking, their capacity to replace unhelpful thought patterns with more adaptive ways of coping grows.

Some examples of CBT techniques include but are not limited to:
• Structured journaling assignments – Ask a client to journal about their beliefs or thoughts related to carbohydrates. Ask them to observe times in the week where they have these thoughts and see if they can identify how these beliefs lead to certain feelings or actions. This exercise can drive insight into the interplay between thoughts, feelings, and actions and present opportunities to identify healthier alternatives.
• Thought records – Ask the client to jot down thoughts while eating a food they deem “unsafe” or “bad.” Encourage them to bring this thought record into session to review, identify cognitive distortions, and work together to reframe the thoughts into more helpful or accurate statements. Clients often need support in the beginning and may not be able to reframe their cognitions on their own.
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- **Situation exposure hierarchies** – These are used to identify behaviors or situations that clients avoid due to anxiety. The purpose is to expose the client to the fear-inducing situation (i.e., a feared food) while preventing a coping response. This process is termed exposure and response prevention.\(^2\) An example of a situation exposure hierarchy is asking clients to make a list of feared foods and then rate the intensity of the fear on a scale of 1-10. They then use those foods to develop behavioral experiments.

- **Behavioral experiments** – Select one moderately feared food (think 4/10) and develop a plan to integrate it into their eating plan for the week. Experiments must begin small, be determined in collaboration with the client, and processed together. If clients alleviate their anxiety by utilizing their eating disorder (i.e. restricting before or after eating the feared food, exercising, etc.) it is considered an incomplete exposure. The client's need to work with a client to either repeat or identify a different version of the exposure that can be completed successfully.

- **Imagery based exposure** – This may be a precursor to behavioral exposure and is a useful technique to use in session. Guide clients through an exercise where they visualize eating the feared food. Ask them to vocalize their thoughts and feelings they are having while visualizing this action and work together to identify alternative thoughts and coping skills.

- **Pie charts** – This involves 5 steps:
  1. Identify distorted or unhelpful belief: “If I eat breakfast I’ll just feel hungrier all day and won’t be able to stop eating.”
  2. On a scale from 1-10, rate strength of the belief (e.g., 8/10).
  3. List all other possible alternatives to that belief.

  4. Divide the pie chart into percentages:
     - “If I eat breakfast I will stimulate my metabolism which is actually a healthy thing.” 20%
     - “If I eat breakfast I may be less likely to binge in the afternoon.” 15%
     - “If I eat breakfast I may feel like I’m going to eat more but may in the long run not.” 10%

  5. Re-rate the original belief (e.g., 6.5/10)

**Table 1: Psychological Modalities and Resources**

<table>
<thead>
<tr>
<th>Psychological Modality</th>
<th>Resources</th>
</tr>
</thead>
</table>
  [https://www.beckinstitute.org/about-beck/our-history/history-of-cognitive-therapy/](https://www.beckinstitute.org/about-beck/our-history/history-of-cognitive-therapy/)  
  [https://iocdf.org/about-ocd/treatment/erp/](https://iocdf.org/about-ocd/treatment/erp/)  
  Training and workshops:  
  [https://www.beckinstitute.org](https://www.beckinstitute.org) Accessed 5-25-17 |
  Worksheets and training aids:  
  Accessed 7-23-17 |
  [https://selfleadership.org](https://selfleadership.org) Accessed May 25, 2017 |
  Molly Kellogg’s Counseling Tips for Nutrition Therapists: Practice Workbook Series.  
  Accessed April 1, 2017. |

4. **Dialectical Behavioral Therapy (DBT)**
   DBT, developed by Marsha Linehan for the treatment of Borderline Personality Disorder (BPD), is widely used in the treatment of EDs. DBT is designed to address deficits in interpersonal relationships, affect regulation and impulse control by teaching specific adaptive skills in the areas of Mindfulness, Distress Tolerance, Emotion Regulation, and Interpersonal Effectiveness.\(^3\) In particular, DBT is an effective treatment modality for EDs as it emphasizes the development of specific skills to manage distressing situations or emotions rather than relying on ED symptoms.

   DBT treatment follows a “treatment hierarchy” that dictates which problematic behaviors are addressed first: life-threatening behaviors (i.e. self-injury, suicidal gestures); behaviors that interfere with receiving effective treatment (i.e. missed sessions, incomplete

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homework, “stonewalling”; and quality-of-life interfering behaviors.3

The RDN will want to clarify whether the client is taking part in formalized DBT treatment and work collaboratively with the therapist DBT involves weekly sessions with a DBT-trained therapist, access to the therapist between sessions using paging, and a weekly DBT group. You do not have to be trained in DBT to be effective at integrating aspects of this framework into your nutrition counseling sessions.

Clinical Case Vignette. Amy is a year-old female who works part-time at a local café and is attending a partial hospital program (PHP) for her bulimia nervosa (BN). She is referred to you by her therapist. Through your assessment you learn that Amy has been diagnosed with BN, depression, anxiety, borderline personality disorder (BPD), and Post-Traumatic Stress Disorder (PTSD). She has a history of substance abuse but has been sober for two years as well as a history of self-harm and one suicide attempt four years ago but is not acting on these urges. She is meeting with her therapist twice per week for DBT treatment in addition to attending PHP. You have a very basic working knowledge of DBT and realize it’s critical to integrate these core concepts into your sessions with Amy. You begin laying that groundwork by asking her the following:

• “Amy, you mention that you keep a daily diary card for your therapist. What are the targets on your diary card? What do you think about adding any targets from our nutrition work, like how well you’re following your meal plan, bingeing and purging, or body-checking behaviors?”

• “Amy, I’d like to be more familiar with the big picture of everything you’re working on in therapy and how our work fits into that big picture. Would it be okay if we started our sessions by reviewing your diary card?”

• “I know you have done a lot of work with your therapist and in group to develop alternative coping skills. What DBT skills are most helpful to you? I’m not familiar with Wise Mind, or D.E.A.R. M.A.N. Could you explain these to me?”

• “It’s common for clients to feel pretty anxious when we’re talking about food, body image, etc. What skills do you think will help you while we’re together? Do you feel able to let me know what you’re feeling and the intensity of that feeling?”

“If you find that you start to become flooded with emotion how will you let me know? Or how will I know? What will I notice? What is the most helpful thing for me to do in that moment? Is there anything I should or shouldn’t say? What grounding tools (i.e. frozen orange, silly putty, paper and markers) are most helpful?”

Acceptance and Commitment Therapy (ACT)

ACT uses acceptance and mindfulness approaches in addition to behavior change strategies, intended to increase flexibility of beliefs and actions.4 ACT may sound similar to DBT and CBT but is distinctly different in that the primary goal of ACT is to help individuals notice “what is,” to develop a separation between one’s thoughts, feelings, sensations, and memories then choose actions that are in alignment with personal values.5 Rather than trying to change thoughts, ACT helps individuals develop a different relationship to their thoughts.

Clinical Case Vignette. Valerie is 34 years old and has a 3-year-old at home. She has struggled with AN for 13 years and most often struggles with feeding herself due to a longstanding belief that “she does not deserve it.” Below is a conversation between Valerie and her RDN.

RDN: “Valerie, will you help me understand what keeps you from following your meal plan?”

Valerie: “I’m scared of getting fat but I also don’t believe I am worthy of feeding my body.”

RDN: “Well I know a part of you is feeling and thinks these things. But I also know a part of you wants something different. I think that’s the part that keeps coming to see me. Why do you think you keep coming?”

Valerie: “Well, I’m a Mom now so I really don’t have a choice. I can’t go back into residential care.”

RDN: “I know how much you care about being a Mom.”

Valerie: “It’s the most important thing in my life.”

RDN: “That’s really amazing. I wonder if we can use that? I know you don’t believe you deserve to eat now. And that is a very old belief that has fueled your ED for many years. I’m wondering if you could choose to eat from your values (i.e. being a Mom who is healthy and present) even when your head tells you no. What do you think?”

Internal Family Systems (IFS)

IFS is a psychotherapy model created and developed by Richard Schwartz, PhD. IFS is rooted in family systems theory and parts-based framework psychotherapies. IFS identifies “the self” and “parts” all of which are different aspects of ourselves:

1. Self- who we are at our most connected, calm, curious, and open. Our “self” is always there but we do not always act from “self.”

2. Parts- embodied aspects of our psyche, our “mini-ME’s” that hold emotions, thoughts, feelings, beliefs, and lead us to behave in certain ways.

It’s important to note that everyone has parts and lots of them. For example, there is a part of me that really likes the idea of sticking to a budget. And yet another part of me likes to shop whenever the mood strikes. These opposing desires live inside of me simultaneously. Acting out on the latter part could lead to financial trouble and doesn’t align with my “self” or core values. IFS aims to help individuals develop greater awareness of their parts, how those parts can be distinguished from their core “self” so that they can live life with greater self-leadership rather than being guided by a singular part of themselves.

You do not need to be IFS-trained (although IFS trainings welcome RDNs)
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to begin integrating core concepts into your work. However, gaining a basic idea of IFS descriptions of the various parts is useful. A valuable summary of the parts can be found on The Center for Self-Leadership website.6

Clinical Case Vignette. Brian is 55 years old, a middle school teacher, and has been struggling with Binge Eating Disorder (BED) for nearly 20 years. He also has pre-diabetes and depression, which he describes as well-managed. He works with a therapist whose primary therapeutic orientation is IFS which Brian likes because it helps him “feel less broken.”

- RDN: “Brian, I am not IFS trained but am familiar with some of the basics. If it’s okay with you, I’d like to integrate some IFS language and principles into our work together.”
  Sample questions:
  - “In what ways has parts work helped you? What do you like about it?”
  - “In what ways do you think we can bring that into the realm of nutrition?”
  - “As you were describing last night’s binge, you mentioned that a part of you didn’t care about recovery in that moment. I’d love to know more about that part.
  - How would you describe that part? How has that part been helpful to you? When do you think that part is most in charge and why? What do you think that part needs?”
  - “So, in certain moments one part of you doesn’t care about getting better from your eating disorder, but in other moments you care a lot?”
  - “What will the part of you that wants to binge have to tolerate if you decide to work through an urge?”

Motivational Interviewing (MI)

“MI is a collaborative goal-oriented method of communication with particular attention to the language of change. It is intended to strengthen personal motivation for and commitment to a target behavior change by eliciting and exploring an individual’s own arguments for change”.7

One of the most difficult aspects of working with the ED population is that clients are often highly ambivalent about change. This clinical dilemma makes MI one of the most empowering resources a RDN can utilize, as one of the core goals of MI is to help an individual resolve their own ambivalence about change. MI also provides clinicians with tools to help the individual resolve their own ambivalence in a compassionate and non-judgmental way. One such principle is learning to “roll with resistance” to change. This principle may help decrease clinician frustration and burnout and is exemplified below.

Some of the most common skills include:

1. Targeting interventions based on the client’s “stage of change.” See Table 2. “Stages of Change and Tasks: Eating Disorders.”

2. Normalize your client’s experience through intentional language. This helps the client to feel less pathologized. One of the core goals of MI is to help the client feel equal to the clinician.

- “It makes so much sense that…”

Table 2: Stages of Change and Tasks: Eating Disorders

<table>
<thead>
<tr>
<th>Precontemplation Eating Disorder ego syntonic and/or client pressured by others</th>
<th>Contemplation Aware of a problem, but not ready for action</th>
<th>Preparation Plans to take action soon &amp; taking small steps</th>
<th>Action Puts plan into motion; changes behavior/ environment; commitment &amp; energy</th>
<th>Maintenance Works to prevent relapse &amp; consolidate change; Action successful more than 6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tasks:</td>
<td>Explore both sides of ambivalence in depth</td>
<td>Concretize initial plan for symptom reduction</td>
<td>Mobilize specific tx plan</td>
<td>Prevent relapse</td>
</tr>
<tr>
<td>Make alliance</td>
<td>Generate pros &amp; cons of change for self &amp; others</td>
<td>Define how success &amp; failure will be defined</td>
<td>Work on strategies to cope with feelings, urges, pressures</td>
<td>Encourage coping skills</td>
</tr>
<tr>
<td>Be nonjudgmental</td>
<td>Keep making connections between ED &amp; consequences</td>
<td>Define how to monitor</td>
<td>Review/learn from past relapses</td>
<td>Reinforce bond with self-help &amp; other resources</td>
</tr>
<tr>
<td>Use questionnaire</td>
<td>Stress cons of change</td>
<td>Develop plan B</td>
<td>Attend to creeping signs of denial</td>
<td>Watch for creeping denial</td>
</tr>
<tr>
<td>Connect ED &amp; consequences</td>
<td>Discuss possible treatment plans</td>
<td>Review tools: self-help, supports, coping strategies</td>
<td>Consider family/ SO work</td>
<td>Encourage new healthy interests</td>
</tr>
<tr>
<td>Emphasize pros of change</td>
<td>Explore difficulties if change occurred</td>
<td>Reinforce awareness of consequences</td>
<td>Explore difficulties as change occurs</td>
<td>Discuss other life issues as medical/ nutritional status improves</td>
</tr>
</tbody>
</table>


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- “Most clients with eating disorders experience…”
- “I was just talking with another client yesterday about…”

3. Desire(Conviction)/Confidence Rulers
   - RDN: “Kimmy, on a scale of 1-10 how much do you want to follow through on the lunch plan we just created?”
   - Kimmy: “Probably a 7.”
   - RDN: “Why not a 5?” Then ask, “Why not an 8?”

   Based on the answer, the RDN should then explore ways to increase desire to follow through on the plan either by tweaking the plan or challenging any cognitive distortions.
   - RDN: “Kimmy, on a scale of 1-10 how confident do you feel about following through on the lunch plan we just created?”
   - Kimmy: “Probably a 3.”
   - RDN: “That’s significantly lower than your desire to follow through.

   What made you choose a 3 instead of a 5?” Then ask, “Instead of a 1?”

   RDN will use that information to determine how to increase client’s confidence in following through on her lunch plan.

4. Utilize discrepancies to demonstrate conflict between recovery and ED mindsets and desires. “You know Corey, we have talked a lot about reducing your exercise. When we’re together you are very clear this is something you are willing to do. And yet after 3 weeks, you haven’t been able to make any traction on this. What do you make of that?”

5. Explore ambivalence with pros and cons. See Table 3: “Exploring Ambivalence Utilizing Pros and Cons.”

Summary

While providing only a brief introduction to a few psychological modalities, this lays the groundwork for the RDN to engage in further study and begin to incorporate some of the techniques into clinical practice. Doing so will not only help the RDN become a more effective change agent but also decrease the natural frustrations and roadblocks frequently encountered in this difficult yet rewarding work.

References

1. Final CDR Competence PDF, page 43.

Table 3: Exploring Ambivalence Utilizing Pros and Cons

<table>
<thead>
<tr>
<th>Dieting / Restricting</th>
<th>Rainbow / Middle Path</th>
<th>Bingeing</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pros:</strong> Results, clear cut Rules, feel in control</td>
<td><strong>Pros:</strong> Long-term, not as rigid as dieting</td>
<td><strong>Pros:</strong> Like what I eat, get to do what I want</td>
</tr>
<tr>
<td><strong>Cons:</strong> Can’t stick with it, food isn’t good</td>
<td><strong>Cons:</strong> Unknown, don’t rust it, hard- not black and white</td>
<td><strong>Cons:</strong> Feel out of control, gain weight, feel discouraged</td>
</tr>
</tbody>
</table>

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BHN practice standards: www.bhndpg.org/members/practice-standards/