Introduction
Sixty-five percent of Americans are overweight or obese, and obesity causes or contributes to increased risk of numerous chronic diseases such as diabetes, obesity, hypertension and cardiovascular disease. This paper offers potential insight into the problem by describing a new approach to understanding it, i.e. that some overeating is an addiction.

The phrase ‘food addiction’ is coming into popular use. However, no paper has attempted to define food addiction in classic drug addiction terms according to current addiction diagnostic criteria. The goal of this paper is to fill that gap by defining food addiction through the behaviors described in current American Psychiatric Association (APA) addiction diagnostic criteria, as well as addictive brain dysfunctions found in overeaters.

In addition to describing a definition of food addiction, the paper briefly discusses the specific foods used addictively based on research and clinical observations. The Conclusion covers the implications of food addiction as an explanation for the overeating epidemic, and the need for understanding addiction in managing obesity and some eating disorders.

The limitations of this paper are several. This paper does not cover assessment of the severity of food addiction, treatment, causes of food addiction, vulnerable populations, genetics, scope of the field of research, nor research techniques. Nor does the paper offer evidence that food addiction exists as that has been done elsewhere. Readers can also find an extensive description of consequences elsewhere.

Background
Before the advent of brain imaging research techniques, definitions of addiction were made through descriptions of patterns of behaviors. Starting in the 1950’s, the APA played a key role in the development of definitions of addictive behaviors through publication of addiction diagnostic criteria in its Diagnostic and Statistical Manual of Mental Disorders (DSM). The drug and alcohol addiction diagnostic criteria are developed by committees of experts in lengthy discussions over years. The criteria are periodically revised.

Can some food use fit these drug addiction criteria? Let’s look at the DSM V, and its criteria for substance dependence. In 2013, the DSM V revised the criteria for substance use disorders; there are now eleven symptoms. This paper uses the DSM-V criteria to define food addiction, as they are considered to be the ‘gold standard’ for diagnosing addictions.

Brain imaging technology introduced in the 1990’s gave insight into the origins of addictive behaviors. With scanning technology such as Magnetic Resonance Imaging (MRI) and Positron Emission Topography (PET), addictions could be defined as the presence of specific dysfunctions in the brain. These dysfunctions suggest that over-activation of cravings pathways in concert with deactivation of inhibition and decision-making centers could explain irrational addictive and overeating behavior that is otherwise difficult to understand.

continued on page 3
Dear Colleagues,

Your Executive Committee has been hard at work to provide you some great benefits of membership. I hope you have had the opportunity to enjoy one or all of our recent webinars. If you haven’t and you would like to view those, please check it out on our new and improved website, www.bhndpg.org! We have also been in the beginning stages of developing fact sheets for professionals and the public. When completed, you will be able to access the fact sheets, which provide information on a wide range of topics, right at your fingertips.

Of course, Behavioral Health Nutrition is a great dietetic practice group because we have great volunteers. We hope you will find an opportunity to join in the fun. Our nominating committee is already beginning to look for candidates to run in next year’s election and for volunteers that are willing to hold one of our appointed positions. All of us bring something unique to the table. I hope you consider holding a position or maybe helping write a Fact Sheet.

FNCE in Atlanta is quickly approaching. BHN has a number of things planned for our members. On Monday, October 20th, you are invited to attend our spotlight session, Dysphagia, Mealtime, and Intellectual and Developmental Disabilities at the Georgia World Congress Center. Our own, Joan Medlen, MS, RDN, LDN, and a fantastic SLP, Jennifer Meyer will give their insights on this topic. Our member breakfast will be on Sunday morning, October 19th. Lee Wallace, MS, RDN, LDN, will be presenting on how adults learn. I personally am hoping she can give me some insights into how to make more efficient use of my time when I am studying. BHN will be at the DPG showcase on October 20th starting at 9:30am in the Georgia World Congress Center. Come by and say hello; we will be there to answer any questions. We look forward to meeting some of you face-to-face!

Even if we won’t see you at FNCE, we want to remind you to take advantage of the other member benefits. BHN has Resource Professionals for all four of our practice areas of Addictions, Eating Disorders, IDD, and Mental Health. Our Resource Professionals have an amazing accumulation of knowledge. As a member, you are encouraged to contact them if you need information on those practice areas. You are also encouraged to post any questions you have to our Electronic Mailing List. Frequently, you will find other members have run across a similar situation and have valuable input. Of course, one of our very best resources is this newsletter. Back issues can be found on the website.

I appreciate the privilege of serving as BHN’s Chair this year. The best part of this experience is the great friendships I am making along the way. I look forward to the rest of the year and hope I have the opportunity to meet more of our members. See you at FNCE!

Sharon Lemons, MS, RDN, CSP, LD, FAND
Chair of Behavioral Health Nutrition
From the Editor

All Good Things Must Come to an End . . .

thus ends my stint as the Editor for this wonderful publication. I have enjoyed the last seven years of being blessed to work with an incredible group of volunteers, members, authors, editors and everyone associated with the BHN DPG. I could not have imagined a better or more learned experience. I am pleased to turn the title over to Hanna Kelley, RD, CD, who will continue the task of making this publication a top member-rated benefit.

This issue of BHN Newsletter is devoted to the theme of comorbidities in behavioral health nutrition practice. Medical comorbidities are prevalent in the populations we serve and often difficult to recognize due in part to communication difficulties and ambiguous symptomology, and possibly underdiagnosed as a result of a commonly held belief that symptoms are just a part of the behavioral health condition. To the contrary, behavior may indicate or even mask underlying medical conditions in the populations we serve. This compilation of articles includes the topics of obesity and eating addiction, medical comorbidities in autism, mental health in patients with diabetes, classic galactosemia, and alcoholic hepatitis.

It has been a pleasure to serve you!

Diane M Spear, MS, RD, LD

Obesity Epidemic... continued from page 1

By combining the APA’s work in developing addiction diagnostic criteria and with the neuro-researchers’ work in exposing the underlying brain dysfunctions; we now have a good understanding of the ‘how and why’ of addictions. These large bodies of research make it possible for us to describe food addiction with confidence. As seen below, findings developed to describe addictions apply readily to overeating.

Addictive Foods

Although a thorough discussion of addictive foods is beyond the scope of this paper, a short description of these foods helps to orient the reader to the below discussion. Addictive foods are identified by the presence of psychotropic ingredients, by neuro-responses in brain imaging research, by clinical and 12 Step observations of loss of control in use of these foods, and by animal studies showing addictive behavior in use of these foods.

The addictive properties of sugar are perhaps the most studied. Rats will choose sugar, high fructose corn syrup, and saccharine over cocaine and heroin. Rats have shown a withdrawal syndrome similar to that of morphine. Sugar activates the dopamine pathway [8]. Food addiction recovery groups often recommend abstinence from sugar and sweeteners.

Gluten and flour made from gluten-grains contains a glutemorphine that appears to activate the opiate pathways. Recovery groups often recommend eliminating wheat and flour. Salt has been observed to be used by morphine addicts in withdrawal, presumably as a replacement for morphine. Processed fat appears to activate the opiate pathways in the brain. Dairy contains a casomorphine which has been shown to create a numbing effect in rats. Caffeine has intoxication and withdrawal diagnoses in the DSM-V. These would all be classified as addictive or “trigger foods.”

Further, the Overeaters Anonymous pamphlet, Dignity of Choice, defines trigger foods in this way: “Trigger” or “binge” foods are foods that we eat in large quantities or to the exclusion of other foods; foods that we hoard or hide from others; foods that we eat secretly; foods that we turn to in times of celebration, sorrow, or boredom; or foods that are high in calories and low in nutritional value. In addition, we look to see whether there are any common ingredients among those foods – like sugar or fat – that might exist in other foods we haven’t listed.

Each of us may have problems with different foods or ingredients. If a food has been a binge food in the past, or if it contains ingredients that have been binge foods for us, we remove it from our plan…. Extra servings of a non-trigger food might create cravings. If we are unsure whether or not a food causes problems for us, we leave it out at first. Later, with abstinence, the correct answer becomes clear. Thus we see that there are similarities between the foods avoided in 12 Step eating recovery groups and foods found to have addictive properties in the research literature. Abstinence from these foods has been seen to reduce cravings and help establish control over eating.

The DSM-V Addiction Diagnostic Criteria as Applied to Overeating

In 2013, the APA published 11 addiction diagnostic criteria in its DSM-V. A person needs to meet two to three criteria for a diagnosis of mild addiction; four to five for moderately addicted; and six or more for severely addicted. For the purposes of this article, the words, ‘processed food’ are used as the addictive substance. Citations are provided to support clinical observations by referring to research that describes the behaviors in overweight populations. Here are examples of how these diagnostic criteria are described in paraphrased quotes from overeating clients.

1. “Processed food is often taken in larger amounts or over a longer period than was intended.” This criterion is reported in studies as the tendency to regain lost weight, presumably in a pattern of unintended overeating.
   a. I started by promising myself I’d only have three cookies, but before I knew it, I had eaten the whole bag.
   b. I always feel like I have to have more no matter what I’ve promised myself!

continued on page 4
2. “There is a persistent desire or unsuccessful efforts to cut down or control processed food use.” This behavior is seen in studies as an inability to lose weight.15
   a. I have tried every diet out there – Atkins, Medifast, Jenny Craig, you name it. I do good for a while and then I just can’t stand it and go back to my old ways.
   b. I know I need to change my eating and eat less junk food, but some days I just can’t stop!

3. “A great deal of time is spent in activities necessary to obtain processed food, use processed food, or recover from its effects.” Food addicts report spending much time and effort getting access to their foods, and then feeling too tired to exercise or do anything but watch television. This behavior is found in the overweight research.16
   a. I plan my day so there is plenty of time to stop at a number of fast food outlets on the way home. Then I eat dinner, too. Then I’m too tired to do anything.
   b. I have food hidden everywhere; I am constantly watching to make sure I have enough.

4. “Craving, or a strong desire or urge to use processed food.” Research shows a relationship between cravings responses and BMI.17
   a. I spent the whole day just thinking about the doughnuts. I couldn’t focus on my work. I finally decided to go eat them just to get them out of my head.
   b. As soon as I finish one meal, I start thinking about the next. I think about food all the time.
   c. I was so angry with my spouse, but oddly, all I could think of was potato chips.

5. “Recurrent processed food use resulting in a failure to fulfill major role obligations at work, school, or home.” Lower productivity among the obese is established in the research literature.18
   a. I am too tired from eating processed foods to play with my kids in the evening.
   b. I miss a lot of work going to doctor’s appointments. I wish I could just clean up my diet and improve my health but I always fail.
   c. My performance at work is declining. I spend all day going back and forth to the break-room and vending machines. I never get anything accomplished.

6. “Continued processed food use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of processed food.” Weight gain is documented in non-supportive marriages.19
   a. My husband wants me to get the fattening foods out of the house and away from our overweight kids. He wants me to lose weight. We fight about it but I love sweets and bread and I love giving them to our kids. It may cost me my marriage.

7. “Important social, occupational, or recreational activities are given up or reduced because of processed food use.” Isolation has been documented in the obese20 and human resource managers are seen to discriminate against the obese.21
   a. I’ve given up applying for promotions. I’m too depressed. I would feel better if I could improve my diet but I can’t give up my ‘comfort’ foods.
   b. I don’t like to eat out with friends and family anymore because I’m afraid I’ll lose control and start eating too much. I know I’m isolating but I don’t care.
   c. The only friends I have are characters on TV. They don’t bug me about my weight. Outside of work, I’m spending my life lying on the couch, eating and watching TV.
   d. I don’t exercise because I don’t want to put on the tight workout clothes. I’m so ashamed of myself.

8. “Recurrent processed food use in situations in which it is physically hazardous.” Eating is the most common distraction while driving.22
   a. I got pulled over for missing a stop sign! I was eating the pizza on the way home and I did not notice the stop sign or the police car behind me. I’m oblivious when I’m eating in the car.
   b. Drive-thru fast food means I pull out onto the street driving with no hands because I’m frantically digging out the fries and catsup.

9. “Processed food use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by processed food.” Research shows non-compliance with diet in diabetics, for example.23
   a. My doctor just diagnosed me with diabetes. He says I need to lose weight and start taking care of myself. I know this, but I just can’t seem to do it.
   b. My blood pressure and cholesterol are really high. They wanted me to lower them with diet, but I keep falling off the wagon.

10. “Tolerance, as defined by either of the following: a. A need for markedly increased amounts of processed food to achieve intoxication or desired effect.” The evidence for this phenomenon in overeaters comes from brain imaging research that shows the down-regulation of dopamine receptor fields characteristic of tolerance.24
    i. Once I start with ice cream, I cannot stop! It never feels like enough! I used to be satisfied with a few spoonfuls!
    ii. From one bag of chips and dip to a jar of peanuts, a bag of pretzels, and then a few bars of chocolate…. I can’t stop till I’m so stuffed I can’t move! I never used to do this.

b. “A markedly diminished effect with continued use of the same amount of processed food.”
   i. With my coffee drinks, I never know what will be enough anymore – sometimes it’s only a little; sometimes it takes a lot.
   ii. One soda used to be enough to get me through the afternoon. Now I’m crashing before the afternoon is over.

11. “Withdrawal, as manifested by either of the following: A morphine-like withdrawal from sugar has been observed in rats25 and caffeine withdrawal is described in the DSM-V.3
    a. “The characteristic withdrawal syndrome for the processed food.”
       i. When I’m trying to cut-back on junk food, the cravings seem to intensify.
       ii. After I binge, I wake up groggy and miserable, so the next day is ruined, too.
    b. “Processed food is taken to relieve or avoid withdrawal symptoms.”
Obesity Epidemic...

continued from page 4

i. If I stop eating sweets and bread for a few days, I am anxious and irritable, and sometimes I even feel like I have the shakes. I start eating sweets again to feel better.

ii. It got so when I tried to diet, I would become lightheaded, forgetful, restless, and unable to concentrate. I would fall off the diet because I was so miserable.

As these quotes and citations show, there are observations and studies of loss of control over processed foods that meet the DSM-V criteria for the diagnosis of an addiction. Although processed foods are quite different from drugs and alcohol as they are perceived and used in our culture, nonetheless the eating patterns that result from their use are similar to addictive behavior.

Addictive Neuro-dysfunction as Seen in Overeaters

Scientists have noticed for several decades that the brains of overeaters show altered functioning similar to the brains of drug addicts.26,27 These observations are based on a growing body of research literature generated from brain imagining studies designed to understand addictions as well as overeating. This research is important because it supports defining overeating as an addiction.

The addiction-like changes found in the brains of overeaters are as follows:

1. Over-active addictive pathways. These pathways are also known as the pleasure or craving pathways and include serotonin, dopamine, opiate, endorphin, and endocannabinoid. In overeaters, these pathways over-secrete craving neurotransmitters.28 This ‘flooding’ of addictive neurotransmitters appears to produce intense cravings that are associated with loss of control.

2. Sensitivity to triggers. In overeaters, very slight stimulation can trigger the overproduction of craving neurotransmitters. Researchers have seen that just thinking about a processed food product can produce the flooding of neurotransmitters.29

3. Conditioned, learned, or Pavlovian responses. The craving response of overeaters, like those of addicts, can be subject to conditioning.28 Just as Pavlov conditioned the saliva glands of dogs to activate at the ringing of a bell without the presence of food, addictive neuro-pathways can also be conditioned to activate even when processed foods are not present. This means that a place, person, thing, event or time associated with consumption of processed foods can trigger cravings without the presence of processed food.

4. Non-functioning cognitive centers. During a flooding of addictive craving neurotransmitters, the decision-making, memory and learning centers in the brain cease to function.27 This may ‘explain’ many of the behaviors seen in the APA’s addiction diagnostic criteria. At the moment of flooding, people are not able to remember consequences nor make good decisions.

5. Non-functioning inhibition center. The flooding has also been observed to coincide with ‘shut-down’ of the inhibition center.30 This is interpreted as a loss of control, and may contribute to the unintended use described in the APA’s addiction diagnostic criteria.

6. Down-regulated receptor fields. In order for the pleasure neurotransmitter to complete its circuit, it must ‘hit’ or ‘dock on’ a transmitter receptor. In addicts and overeaters, these receptor fields are down-regulated or ‘shut-down’.31 The theory is that as over-use and overexposure to stimulation bombard these receptors, they down-regulate. The person then increases consumption in an attempt to reestablish the level of pleasurable feeling they once had when the receptors were open. This may explain the phenomenon of tolerance.

7. Activation by stress. The addictive pathways are activated by stress in overeaters.32 This body of research is important evidence for overeating as an addiction to processed foods.

What Food Addiction is Not

Food addiction is not binge eating just as binge drinking is not alcoholism. However, binge eating may be present in food addiction. Food addiction is not emotional eating just as alcoholism is not emotional drinking; rather, it is a substance-based addiction. Even ‘food addiction’ is not well-named. It should be ‘Processed Food Addiction’ as there is little evidence for addiction to non-processed foods.

A very important distinction must be made between a substance-based addiction and a behavioral addiction. Food addiction has been discussed as a behavioral syndrome. However, this is not consistent with neuro-imaging research nor clinical observations of the role of specific processed foods in addictive eating behavior. Approaching food addiction as a problem of behavior leads to a confusing dead end in terms of treatment, because we can’t become abstinent from the behavior of eating.

Framing the problem as loss of control over specific foods opens the door to treatment success because, while we have to eat, we don’t have to eat the foods that trigger addiction. We are now in the familiar territory of recovery from alcoholism. Yes, we have to drink, but we don’t have to drink alcohol. The ‘substance’ approach is well-supported by both research and clinical experience.

Under this approach, food addiction is being defined as a syndrome of addiction to substances in the same category as alcohol and drug addiction; and not as a behavioral addiction such as shopping, sex or gambling.

Conclusion

Understanding addiction to certain foods may be helpful in managing the obesity epidemic. If overeating is, in fact, an addiction to processed foods, that could explain why weight-loss treatment approaches that involve continued use of addictive foods have sometimes been unsuccessful. Although extensive 12 Step Groups use abstinence protocols on a lay basis, health professionals need to become comfortable with assessing and treating food addiction. Given the dire nature of the obesity epidemic, such commitment would seem necessary.

About the Authors:

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Joan Ifland, PhD is a food addiction professional active in both practice and research. She earned her PhD in 2010 at Union Institute in addictive nutrition.
Obesity Epidemic... continued from page 5

She is the chair of the Council on Food Addiction at the American College of Nutrition and co-founded The Society of Food Addiction Professionals.
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References:

CPE credit (1.0 hour) is available from BHN for the full text version of the article, Obesity Epidemic: Understanding Addiction in Managing Overeating. Access the article at http://www.bhnpg.org/moa/cpes.asp

Take your passion for Behavioral Health Nutrition to the next level:
Become an officer or volunteer for BHN!

With one simple commitment, you can grow your skills for communication, advocacy and leadership on a national level. Connect with amazing colleagues while making a real impact in promoting the dietitian’s role in behavioral health.

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- **HOD Delegate**
- **Treasurer**
- **Nominating Committee Chair Elect**

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- **Addictions Resource Professional**
- **Policy and Advocacy Leader**
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**Deadline for officer nominations is October 31, 2014.**
Identifying Medical Comorbidities in Autism Spectrum Disorders
GUIDE FOR HEALTHCARE PROFESSIONALS
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Care providers should be aware that problem behavior in patients with ASDs may be the primary or sole symptom of the underlying medical condition.” Consensus Report, Pediatrics, Buie et al., 2010

Recent studies have shown that many medical conditions are significantly more prevalent in people with autism compared to the typical population, including: eczema, allergies, asthma, ear and respiratory infections, gastrointestinal problems, severe headaches, migraines, and seizures.

Individuals with autism appear to be at increased risk for developing common chronic diseases including: diabetes, coronary heart disease, cancer, and osteoporosis. Mortality is significantly increased in autism, with death rates being more than three times higher than the general population. Premature deaths in autism are mostly the result of co-occuring medical conditions such as epilepsy, respiratory, gastrointestinal and cardiovascular disease. Risk of both epilepsy and premature death increases with the severity of autism.

Autism is increasingly being recognized as a whole body disorder, with the core deficits in communication, social interaction, restrictive and stereotypic behaviours being surface manifestations of a systemic and complex disease process. Immune dysregulation appears to be a key feature.

Sudden and unexplained behavioral change can be the hallmark of underlying pain or discomfort. Behavioral treatment may be initiated as the possible concurrent medical illness is being investigated, diagnosed (or excluded), and treated, but the behavioral treatment should not substitute for medical investigation.”
Consensus Report, Pediatrics, Buie et al., 2010

Accurate diagnosis of co-existing medical conditions is possible by taking account of the following points:

- Problem behavior in patients with autism may be the primary or sole symptom of an underlying medical condition.
- Self-harming, aggression, night-waking, change in appetite, grimming, strange postures and such are not part of the diagnostic criteria of autism. In conflict to current research and accumulating clinical experience, these and other symptoms and behaviours have been erroneously attributed to either a mental health or behavioural problem or as being inherent to autism or some preconceived facet of that diagnosis. There is a substantial body of evidence that these behaviors may have a physical cause (e.g. reflux) and to prevent diagnostic overshadowing, organic causes should be sought in the first instance.
- Parents and carers generally DO give accurate and quality information about symptoms or behavior change.
- Parents and carers may be unaware of the possible implications of the symptomatology, especially if at any point they have been told that behaviours are ‘simply autism’.
- Individuals with autism who are experiencing pain or discomfort may not be able to identify the physical location of that pain/discomfort within their body.
- Individuals with autism may not respond in the typical way to common illnesses.

THE TABLE BELOW IS DESIGNED TO HELP IMPROVE RECOGNITION OF SOME OF THE PROBLEMS ENCOUNTERED WHEN AUTISTIC PATIENTS PRESENT WITH COMORBID HEALTH ISSUES.

<table>
<thead>
<tr>
<th>Behaviours which may indicate an underlying comorbid illness include:</th>
<th>Behaviours which may indicate an underlying comorbid illness include:</th>
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<td>Sudden change in behavior</td>
<td>Sudden change in behavior</td>
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<td>Loss of previously acquired skills</td>
<td>Loss of previously acquired skills</td>
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<td>Irritability and low mood</td>
<td>Irritability and low mood</td>
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<td>Tantrums and oppositional behaviour</td>
<td>Tantrums and oppositional behaviour</td>
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<td>Frequent night-waking or general sleep disturbance</td>
<td>Frequent night-waking or general sleep disturbance</td>
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<td>Teeth grinding</td>
<td>Teeth grinding</td>
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<td>Change to appetite or dietary preferences</td>
<td>Change to appetite or dietary preferences</td>
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<td>Heightened anxiety and/or avoidance behaviours</td>
<td>Heightened anxiety and/or avoidance behaviours</td>
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<td>Repetitive rocking or other new repetitive movement</td>
<td>Repetitive rocking or other new repetitive movement</td>
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<td>Walking on toes</td>
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<tr>
<td>Posturing or seeking pressure to specific area</td>
<td>Facial grimacing, wining, tics</td>
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<td>Sensory hyper-responsitivity: hyperacusis (e.g. covering ears with hands), tactile defensiveness, sensitivity to light</td>
<td>Sensory hyper-responsitivity: hyperacusis (e.g. covering ears with hands), tactile defensiveness, sensitivity to light</td>
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<td>Behaviour around evacuation</td>
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<td>Aggression: onset of, or increase in, aggressive behaviour</td>
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<td>Self-injurious behaviour: bitting, hits/slaps face, head-hanging, unexplained increase in self-injury</td>
<td>Self-injurious behaviour: bitting, hits/slaps face, head-hanging, unexplained increase in self-injury</td>
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<td>Constant eating/drinking/swallowing (‘grazing’ behavior)</td>
<td>Constant eating/drinking/swallowing (‘grazing’ behavior)</td>
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<td>Frequent clearing of throat, swallowing</td>
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<td>Mouthing behaviours: chewing on clothes</td>
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<td>Oesophagitis</td>
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<td>Soft or hard stool constipation (underlying cause will be relevant)</td>
<td>Soft or hard stool constipation (underlying cause will be relevant)</td>
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<td>Small Intestinal Bacterial Overgrowth</td>
<td>Small Intestinal Bacterial Overgrowth</td>
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<td>Musculoskeletal injury or disease</td>
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<td>Seizure Disorder (including subclinical crisis)</td>
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<td>Allergy Disorder</td>
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<tr>
<td>Pain can be acute or chronic, progressive or static.</td>
<td>Pain can be acute or chronic, progressive or static.</td>
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For an electronic version of this guide sheet with full list of references, as well as a full length version of this document visit www.treatingautism.co.uk/resources/research-science

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DATE OF PREPARATION: OCTOBER 2013
Classic Galactosemia: A Disease Incompletely Treated

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Introduction
Classic galactosemia is defined as an autosomal recessive disorder of galactose metabolism. It is among the most common carbohydrate metabolic disorders and is most commonly caused by a deficiency of the enzyme galactose-1-phosphate uridylyltransferase (GALT), first described in 1935 by Mason and Turner and later demonstrated by Isselbacher and colleagues in 1956. GALT deficiency impairs metabolism of galactose derived from dietary lactose and endogenous sources. Presentation manifests in the neonatal period after galactose has been ingested via lactose in breast milk or infant formula, although variant forms can present at a later time. Symptoms include food intolerance and muscle hypotonia; while more definitive clinical signs include hepatosplenomegaly, jaundice, hepatocellular insufficiency, hypoglycemia, renal tubular dysfunction, sepsis, and cataracts. As galactose accumulation progresses, symptoms likewise evolve until death ultimately occurs unless dietary intervention is started. Life-saving treatment involves the severe restriction of dietary galactose and lactose. Without diet intervention, death typically occurs within one month. Even the severest restriction of dietary galactose intake does not eliminate long-term complications such as delayed mental development, verbal dyspraxia, motor abnormalities, and hypergonadotrophic hypogonadism. Adults produce an estimated one gram per day of endogenous galactose that is suggested to significantly contribute to these late complications.

Metabolism
Normal infants rapidly metabolize galactose to glucose through the Leloir pathway. In cases of galactosemia, impaired galactose metabolism causes high concentrations of galactose to accumulate and be excreted in the urine. In normal metabolism, galactose is promptly metabolized to glucose-1-phosphate by the action of four enzymes (Figure 1). Galactose mutarotase (GALM) is responsible for allowing subsequent conversion of β-D-galactose into α-D-galactose; galactokinase (GALK) allows conversion of α-D-galactose into galactose-1-phosphate; GALT permits conversion of galactose-1-phosphate and uridine diphosphate (UDP)-glucose into glucose-1-phosphate and UDP-galactose; UDP-galactose epimerase (GALE) allows interconversion of UDP-glucose and UDP-galactose. Collectively, these enzymes make up the Leloir pathway (Figure 1).

There are three types of galactosemia, characterized by the deficient enzyme within the Leloir pathway. GALT deficiency is known as type I, GALK deficiency is type II, and GALE deficiency is type III. Each type of galactosemia has a primary set of clinical features with crossover amongst them all. GALT deficiency (type I) – the focus of this manuscript – is the most common and is often referred to as classic galactosemia.

The gene for GALT is located on chromosome 9p13; the gene for GALK is located on chromosome 1p36-p35; and the gene for GALE is located on chromosome 17q24. Mutations in genes coding for GALT, GALK, and GALE may cause a major decrease in enzyme activity resulting in variable clinical phenotypes. Galactosemia is associated with common and precise gene expression alterations affecting multiple metabolic pathways as recently demonstrated by Coman et al.

The presence of endogenous production of galactose was first examined in classic galactosemic patients in 1995. This ability of humans to produce galactose is "self-intoxicating" for patients with galactosemia. Infants with galactosemia prior to having ever received dietary galactose still demonstrate elevated levels of erythrocyte galactose-1-phosphate. Studies have shown endogenous galactose production to be age-related, with highest production at birth and decreasing by more than 50% with age. Levels of GALT in the liver rise over the first week of life, and then decrease to low levels in adulthood. One study has concluded that endogenous galactose production is a constant in patients and controls, not being influenced by exogenous galactose supplementation.

Screening
Newborn screening can result in improved health outcomes for many disorders; and for some of these, it can also be life-saving. A single heel stick using a 3mm blood sample is used to test for 30-40 disorders in one vial of blood. While general guidelines exist requiring newborn screening to be conducted on or before the fifth day of life, in the United States, newborns are screened during the first 48 hours of life prior to hospital discharge to prevent long-term complications among those with metabolic disorders. While screening varies from state to state, all 50 states require screening for galactosemia. Screening for galactosemia reveals a negative or positive result based on the Beutler test, also known as the fluorescent spot test. If a positive result is obtained (the fluorescent spot test shows reduced or no galactose-1-phosphate activity), then the GALT enzyme is measured to determine if a patient has classic galactosemia.

Figure 1. Enzymes in the Leloir pathway of galactose metabolism.

<table>
<thead>
<tr>
<th>Enzyme</th>
<th>Reaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>β-D-galactose</td>
<td>GALT</td>
</tr>
<tr>
<td>α-D-galactose</td>
<td>GALM</td>
</tr>
<tr>
<td>Galactose-1-phosphate + UDP-glucose</td>
<td>GALT</td>
</tr>
<tr>
<td>UDP-galactose</td>
<td>GALE</td>
</tr>
</tbody>
</table>

continued on page 9
Classic Galactosemia... continued from page 8

Screening allows for early diagnosis, and therefore early intervention, to prevent mortality from galactosemia.\(^1,5\) As a result of newborn screening over a ten-year period, mortality was reduced more than ten-fold (4.6 to 0.3 children per year) in newborns with galactosemia.\(^5\) Although early interventions as a result of screening do not eliminate long-term complications, screening still has many advantages. It is not only life-saving, but it also demonstrates a significant cost savings in future patient care due to reduced complications and improved patient outcomes.\(^1,5\)

Presentation

Infants with classic galactosemia, once exposed to dietary galactose, experience poor feeding and weight loss followed by deterioration of multiple organ systems including liver dysfunction, coagulopathy, renal tubular dysfunction, cerebral edema, and vitreous hemorrhage.\(^1,8\) Cataracts are also frequently detected fairly early.\(^1\) The first clinical symptoms may include any number of the previously mentioned symptoms and are not necessarily sequential.

Diagnosis

Signs and symptoms of classic galactosemia and other inborn errors of metabolism (IEM) are typically common and nonspecific.\(^1,8\) Gupta suggests the following four parts to the evaluation and diagnosis of any IEM: history (family history), physical examination, initial screening tests, and advanced screening tests.\(^1,8\) Since galactose is reduced and excreted in the urine, urine can be used as a simple screening test for variant forms of galactosemia.\(^1\) If a newborn is maintained on intravenous fluids, as is often the case during neonatal crisis, no galactose will be metabolized and therefore will not be present in the urine.\(^1\) The gold standard for diagnosis of classic galactosemia is the measurement of GALT activity in the erythrocytes.\(^1\) A normal GALT level is >3.5 u/gHb, borderline is a reduced level of 3.2-3.5 u/gHb, and a positive result for diagnosis of galactosemia is defined as ≤3.1 u/gHb.\(^9\)

Prevalence

Frequencies of IEM among the population vary. Most are rare, but collectively they are common.\(^12,13\) IEM in the United States are estimated to be between 1 in 1400 and 1 in 5000 live births; prevalence varies worldwide.\(^12\)

The most common mutation in classic galactosemia is the p.Q188R mutation.\(^1,2\) This is the substitution of glutamine at position 188 with arginine.\(^1\) This mutation accounts for the highest frequency of galactosemia in Caucasian populations.\(^1,2\) Another mutation, p.S135L, replaces serine with leucine and is found nearly exclusively in the African American population.\(^1,2\) Neither of the above mutations are present in Japanese populations, among which galactosemia is very rare (1:1,000,000).\(^1,12\) Overall, the incidence of galactosemia is 1 in 30,000 to 60,000 for classic (type I) and less than 1 in 100,000 for type II, with type III being even more rare.\(^5\) Galactosemia equally affects males and females.\(^2\)

Treatment and Follow-up

The most important step in management of galactosemia is immediate removal of galactose from the diet once the diagnosis is suspected.\(^1,3\) This alone reverses the crisis experienced by neonates with galactosemia and is life-saving.\(^4,9\) In infants, this means switching from breast milk or cow’s milk formula to soy-based formula.\(^1,14\) Soy formulas, or those based on dextrin maltose as a carbohydrate source, contain minimal amounts of galactose.\(^1\) It is recommended that galactose-1-phosphate levels of treated infants be at or below 3 mg/dL by six months of age.\(^14\) To put this in perspective, if an infant ingests a 60 mL bottle of cow’s milk-based formula, a rapid accumulation of 10-20 mM (18-36 mg/dL) galactose in his or her blood and tissue would result.\(^15\) Most infants consuming only soy-based formulas are able to sustain levels within treatment range after consuming a soy-based formula for a few weeks to several months.\(^14\) Dietary restriction of galactose during pregnancy is noted to have no effect on long-term complications for an affected fetus.\(^2\)

As an infant ages, it is inevitable that some galactose will be introduced into the diet through fruits and vegetables, breads, and legumes which contain trace amounts.\(^1\) Removing galactose from the diet requires exclusion of milk and milk products and an understanding of the galactose content of foods, which can be quite difficult.\(^2,3\) During adherence to a galactose-restricted diet, urinary galactose excretion normalizes.\(^1\) While serum concentrations of galactose-1-phosphate detected at diagnosis decline after dietary intervention has begun, concentrations always remain elevated compared to healthy controls.\(^1\) Avoidance of galactose in the diet is recommended to continue throughout life; and due to restriction of dairy foods, a calcium supplement is recommended.\(^3\)

There is much debate over how much exogenous galactose can be tolerated and whether this changes with age. One case study has demonstrated good outcomes with increased galactose tolerance in an adult woman who discontinued diet at age three.\(^1\) This may be due to the natural decrease in endogenous galactose production with age.\(^1\) This concept needs further examination. Although late complications of classic galactosemia seem inevitable, consensus focuses on the importance of detection, evaluation, and early intervention for the prevention and/or delay of developmental problems.\(^1\) This can be achieved when providers adhere to the following recommendations:

1. Evaluate girls between ages 10-12 years for hypergonadotrophic hypogonadism and refer to an endocrinologist or reproductive gynecologist.\(^1,3\)
2. Conduct regular ophthalmological evaluations (within 30 days of diagnosis and annually).\(^1,3\)
3. Conduct regular measurements of blood galactose-1-phosphate and/or urinary galactitol excretion (every three months).\(^1,3\)
4. Refer children to appropriate language and speech centers.\(^1,3\)
5. Refer patients to a dietitian who has experience with metabolic disorders.\(^1,3\)
6. Encourage consultation with a biochemical geneticist for diagnostic laboratory evaluation, monitoring, and clinical care.\(^1,3\)

Though these tests and follow-up parameters monitor compliance, only severe noncompliance is detected through laboratory evaluation.\(^1\)

Ideally, new therapies for galactosemia would result in an increase in GALT enzyme activity, providing at least a basal amount to cover metabolism of endogenous production, or to decrease toxic metabolites; currently

continued on page 10
no treatment is available that addresses endogenous production. Suggested methods for achieving this include administration of exogenous GALT enzyme and liver cell transplantation or liver transplantation. As these methods are not currently feasible, interventions should focus on decreasing accumulation of toxic metabolites.

Currently pharmacologic agents are not used in the treatment of galactosemia, although the effects of pharmacological dosages of folic acid in galactosemia have been studied. Segal and Rogers, through studies most recently conducted in rats, concluded that normal transferase enzyme can be manipulated through pharmacological dosages of folic acid; this manipulation could result in increased galactose utilization and better long-term outcomes. However, whether or not this can actually enhance residual transferase activity in galactosemic humans is still undetermined. Therefore, pharmacologic doses of folic acid are not currently included in treatment regimens.

**Long-term Complications**

Despite following appropriate dietary therapy, most patients develop one or more long-term complications. It is suggested that there are as many social as physical complications. Chronic or long-term conditions in childhood may delay milestones, causing increased dependence on parents and reduced peer interaction. Approximately 60% of patients with classic galactosemia show defects in speech and language development. Bosch reports that 15–50% of patients demonstrate verbal dyspraxia; specifically, lower IQ levels are correlated with the most severe speech disorders. 

Apart from the high mortality rate in untreated newborns with galactosemia, life expectancy has never been studied; most patients appear to reach adulthood when adhering to dietary recommendations. As suggested earlier, long-term complications may be the result of endogenous galactose synthesis. Controversy arises over whether damage occurs in utero and/or later in life. Abnormally high levels of galactose-1-phosphate, galactitol, and galactose are found in cord blood and amniotic fluid in the third trimester of pregnancies with a galactosemic fetus, suggesting galactose toxicity may occur in utero. The enzymatic pathways of galactose metabolism develop around the tenth week of gestation; and by 20 weeks of gestation, elevated concentrations of metabolites are noted in fetuses.

One complication more specific to adulthood is ovarian failure. Hypergonadotrophic hypogonadism is found in nearly all females with galactosemia. Eighty percent of girls 12 years and younger were found to have an elevated FSH level, indicating ovarian dysfunction. It is suspected that the effect of GALT deficiency on the ovary may be local, not related to galactose exposure. In one study, eight of 34 patients over age 17 presented with primary amenorrhea, and many others developed secondary amenorrhea after several years of menarche. Yet in this same study, 30% of women over the age of 22 had normal menstruation. Fourteen pregnancies have been reported in 9 out of 37 women over the age of 17 with classic galactosemia. Patients have been found with normal ovarian function during childhood but with abnormalities noted in late adolescence and after childbirth. Early identification and treatment of galactosemia does not necessarily prevent this complication. This being said, pregnancy is a rare occurrence in galactosemic patients. Few cases have been studied involving pregnancy and outcomes in women with classic galactosemia. Schadewaldt et al. monitored five patients who had galactosemia and reported their pregnancy and breastfeeding outcomes. Lactose production in mammary glands seems to exert minimal impact on the metabolic control in an affected mother; breastfeeding on demand had no effect on the concentration of galactose metabolites. Breastfeeding of non-galactosemic offspring can be recommended, and additional metabolic monitoring while breastfeeding would not be necessary.

Bone density can be affected in children with galactosemia, however females are more susceptible to decreased bone mineral density related to ovarian failure. Despite dietary calcium intake and normal plasma calcium concentrations, decreased bone mineral density has been detected in children. Calcium and vitamin D supplementation is recommended to meet requirements while consuming a dairy-free diet.

There is no conclusive evidence that galactosemia impacts male reproduction. Male testicular function has been determined to be normal. Gubbels et al. recently suggested that galactosemic males may face some fertility alterations possibly due to abnormal psychosocial development, but infertility is still deemed rare in males.

Cataracts are one of the few understood pathogenic complications of galactosemia. Galactitol accumulates in the lens and develops within two months in untreated cases. Cataracts are mostly likely seen in the neonatal period; they may subside and then reappear in older patients who fail to comply with dietary interventions.

Some studies have revealed a decrease in IQ with increasing age among galactosemic patients, yet studies with individual longitudinal testing have not been completed. This problem intensifies as they enter school or adulthood and attempt to become independent. Waisbren et al. studied 33 adult patients with classic galactosemia to determine if classic galactosemia is a progressive disease with neurodegeneration. The study documented four primary findings: symptoms in adults were not related, older subjects did not exhibit poorer physical health, symptoms further suggest central nervous system involvement, and genotypes (p.Q188R specifically) were unable to predict outcomes in patients with zero residual GALT activity. Waisbren et al. concluded that data did not support the theory that galactosemia is a progressive neurological disease. Little is known about the neuropathology in classic galactosemia. In 1982, Nelson et al. reported abnormal white-matter signals, focal white-matter lesions, large ventricular size, and mild cerebral atrophy in brain images of early-treated patients. However, in recent years, little research has been conducted specific to these findings.

Although some organ damage occurs in utero, a significant contribution to long-term complications among patients with galactosemia stems from continuous galactose toxicity as life progresses. It is interesting that neither age at time of diagnosis nor severity of
clinical illness at time of diagnosis correlate with the presence and severity of later complications. However, presence and severity of verbal dyspraxia as well as premature ovarian failure are correlated with the genotype p.Q188R homozygotes, a higher erythrocyte galactose-1-phosphate after the age of one year, and a lower total body galactose oxidation. Cognitive problems have also been correlated with genotype—worse outcomes were associated with p.Q188R homozygotes—but not with galactose-1-phosphate concentrations.

As with many chronic conditions, health-related quality of life is a concern. In Dutch patients with classic galactosemia, the disease had significant impact on cognition in all age groups and specifically on social function for those aged 16 years and older. Classic galactosemia patients are significantly less frequently married and significantly less frequently employed compared to the general population. It has been deemed essential that parents and clinicians encourage children with classic galactosemia to participate in peer-related activities to stimulate social performance, thus producing a more normal course of life. These quality of life associations are thought to be due to the result of complications with classic galactosemia rather than the burden of a chronic disease or the lifelong dietary changes.

A recent study found that despite long-term dietary adherence, more than 60% of adults with galactosemia have motor dysfunction, a third of them with symptoms. Detection of motor dysfunction is important to provide treatment. Sixty-two percent of participants with motor complications presented with speech difficulties, but speech difficulty was found in only 12% of those without motor complications. Premature ovarian failure was likewise higher in patients with motor complications (93%) versus in those without motor complications (66%).

Dietary therapy has proven beneficial specifically in relation to liver disease. With adherence to a galactose-free diet, prognosis and survival without liver disease are more likely when compared to those not including diet therapy in treatment. However, there are rare reports of patients with severe galactosemia surviving off of diet therapy since childhood; these reports have raised concerns about whether lactose restrictions must be maintained after infancy. While dietary changes reduce the burden of accumulated galactose and metabolites, they do not reverse the enzyme deficiency.

Conclusion

Over time, the understanding of classic galactosemia has been greatly improved. Initially, galactosemia, along with all IEM, was considered a pediatric ailment. There are still many problematic complications associated with this IEM, even with dietary compliance. Adults with classic galactosemia have been reported to have a lower health-related quality of life, especially in relation to cognitive and social function. To improve quality of life for this population, efforts must be focused on decreasing or eliminating complications. Lack of data in adults with galactosemia hinders the ability to predict disease progression. Despite the fact that galactosemia remains a disease incompletely treated—complications are inevitable despite dietary compliance—comprehensive, multisite evaluations of adult cohorts do not exist. This in part prevents clinicians from being able to predict adult morbidity.

As previously stated, complete restriction of dietary galactose is life-saving to infants. Further studies are necessary to determine the influence of modifier genes and accessory salvage pathways of galactose metabolism and individual galactose tolerance.

Nutritional recommendations for galactosemia by metabolic specialists support a life-long diet therapy. As shown above, however, this does not eliminate the disease’s negative implications.

While nutrition certainly impacts outcomes of galactosemia, it is not a cure! It is generally recognized that there is a need for new strategies in treatment to accompany dietary galactose elimination since current dietary therapy has failed to prevent complications. New treatment strategies may include increasing GALT enzyme activity via exogenous GALT enzyme, liver cell transplant, or liver transplantation, or decreasing accumulation of toxic metabolites through endogenous production. Methods to increase enzyme activity are not currently available in practice or are too high-risk; therefore, the latter should be further explored. As a part of medical nutrition therapy, diet adherence is critical from birth into adulthood to minimize complications and maximize patient quality of life.

References

A Flourishing Approach to Mental Health in Patients With Diabetes

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Abstract

Therapeutic approaches addressing mental health often focus on mental illness (i.e., depression, stress, anxiety, and burnout) rather than mental health. This has led to treatment approaches primarily focused on what clinicians characterize as “coping.” We suggest additional treatment approaches rooted in mental health in an attempt to expand the treatment repertoire of health care providers, explore salutogenesis (i.e., the study of what causes health), and introduce the concept of “flourishing with diabetes.”

Introduction

The mental health aspect of living with diabetes is often referred to in negative terms, such as coping with depression, denial, stress, shame, and guilt. Working from this negative perspective embodies a “coping mindset,” which focuses on what is not working, followed by a problem solving process that attempts to move the patient to what is considered “normal” functioning. Further, patients are generally guided to work at avoiding negative outcomes, such as complications, overweight, high blood glucose values, depression, and anxiety.

Remarkably, little attention is given to people who are living well with diabetes and, in fact, may be experiencing a benefit living with diabetes. Such people may be described as “flourishing” in that they have moved beyond coping and are living healthy, happy, and meaningful lives, not despite their diabetes but because of it. Exploring the causes of flourishing yields a different treatment approach that we term working from a “flourishing mindset.” The “flourishing mindset” may be described as one of collaboration, wherein the practitioner and patient together determine mutually selected goals, successful approaches that build on the patient’s strengths, and effective solutions.

A review of the literature explains the concept of flourishing and provides case studies and practical tips that health care providers can use to aid patients in designing forward-looking approaches to care.

Table 1. Comparison of Coping and Flourishing Treatment Strategies

<table>
<thead>
<tr>
<th>Treatment Characteristics</th>
<th>Coping Mindset</th>
<th>Flourishing Mindset</th>
</tr>
</thead>
<tbody>
<tr>
<td>Approach</td>
<td>Cope and repair</td>
<td>Design and build on what is already working</td>
</tr>
<tr>
<td>Goal</td>
<td>Come up to “normal”</td>
<td>Go beyond “normal” and flourish physically and psychologically</td>
</tr>
<tr>
<td>Direction</td>
<td>Avoid what you don’t want</td>
<td>Move toward what you do want</td>
</tr>
<tr>
<td>Focus</td>
<td>The disease, what is going wrong, and corrective actions</td>
<td>The patient in personal life context, what is going well, and building on successes</td>
</tr>
<tr>
<td>Health care provider-patient relationship</td>
<td>Health care provider is the expert and decides, tells, and explains what the patient should do</td>
<td>Health care provider and patient are both experts who leverage each other’s strengths to co-design a way forward</td>
</tr>
<tr>
<td>View of diabetes and impact on one’s life</td>
<td>A burden that one must fight/battle/overcome and that makes life smaller/limiting</td>
<td>Bestows benefits, integrates into one’s life, and makes life bigger/possibilities</td>
</tr>
</tbody>
</table>

Literature

Emerging research investigating the flourishing mindset includes knowledge on salutogenesis, solution-focused brief therapy (SFBT), and positive psychology.

Most literature on diabetes and mental health focuses on mental illness. Titles of mainstream books are illustrative: Diabetes Burnout: What to Do When You Can’t Take It Anymore,2 Psyching Out Diabetes: A Positive Approach to Your Negative Emotions,3 Depression and Diabetes,4 and The Mind-Body Diabetes Revolution: The Proven Way to Control Your Blood Sugar by Managing Stress, Depression, Anger and Other Emotions.5

Hislop and colleagues6 investigated the prevalence of psychological distress in young adults with type 1 diabetes. They found that 64.8% had experienced no distress at all, which the authors considered “normal,” and 35.2% had experienced moderate and severe distress, which they considered to be “abnormal.” Instead of investigating why 64.8% had experienced no distress, the authors focused solely on those whom they considered abnormal.

In contrast, medical sociologist Aaron Antonovsky has conducted inquiries into the origins of good health.7,8 To his surprise, his research showed that nearly one third of a research population of holocaust survivors were maintaining good health and leading satisfying lives. He asked the salutogenic question, “how can a person be moved toward greater health?”

His research identified biological, material, and psychosocial factors that he defined as generalized resistance resources, including optimism, self-efficacy, learned resourcefulness, hardiness, money, social support, intelligence, and tradition. The commonalities in these factors are captured in his “Sense of Coherence” (SOC) theory, which identifies three key elements necessary to move people to greater health:

continued on page 13
A Flourishing Approach... continued from page 12

1. Understanding the challenge (comprehensibility)
2. Believing that resources are within or available (manageability)
3. Being motivated (meaningfulness)

Antonovsky concludes that the stronger a person’s SOC, the more successful would be his or her attempts at creating health.9 Thus, a treatment plan that addresses these three points will likely enable patients to be more successful in reaching good health.

SFBT is a psychotherapy-based coaching approach whose orientation is “solution-focused” rather than “problem-focused”.9-11 SFBT aims to assist patients in setting goals and designing solutions and strategies to promote movement forward. Among the SFBT practices are helping patients to discover their strengths; search for “exceptions”; identify times when the problem or situation does not exist and contributors to that; and recall successes, exploring the factors and choices that led to their creation.

The guiding principle of SFBT is a coach and thinking partner approach. Many of the techniques can be helpful for health care professionals working with people with diabetes.12 Three representative techniques are: 1) scaling questions, in which patients determine where to place themselves on a scale from 0 (extremely poor/nonexistent) to 10 (excellent) in terms of self-management tasks and psychological well-being; 2) identifying strengths and exceptions; and 3) asking the “Miracle Question,” in which patients describe their desired future state and visualize solutions.9 Patients are then asked to design a next step to bring them closer to the envisioned ideal state.

The positive psychology movement also advocates seeking health through a more positive approach.13 Martin Seligman, PhD, considered by many to be the father of this movement, writes: “Positive mental health is a presence: the presence of positive emotion, the presence of engagement, the presence of meaning, the presence of good relationships, and the presence of accomplishment. Being in a state of mental health is not merely being disorder-free; rather it is the presence of flourishing”.14

Barbara Fredrickson, PhD, has developed a “broaden and build” theory, stating that positive emotions such as joy, curiosity, contentment, love, play, gratitude, and appreciation broaden an individual’s mindset to be more open, see new possibilities, create social ties, and be more creative and flexible.15 These capacities build an individual’s well of inner strengths and resources from physical and intellectual to social and psychological, enlivening mental health. Frederickson also proposes that negative emotions such as fear, guilt, and shame narrow a person’s mindset, leaving individuals with fewer capabilities and resources. The positive psychology approach would encourage asking the unthinkable question: “What good has diabetes given the patient?”

Focusing on what causes health, rather than what causes illness yields additional treatment options that may lead to improved outcomes for both patients and providers (Table 1).13,16

<table>
<thead>
<tr>
<th>Situation</th>
<th>Flourishing Approach</th>
<th>Observed Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>2010. 65-year-old married woman who had been diagnosed with type 1 diabetes 9 months previously. With the difficulty she is having in managing blood glucose, she is stressed and frustrated. Referred by her Certified Diabetes Educator.</td>
<td>Three sessions. Patient was asked to tell her life story. Provider and patient together identified the patient’s strengths and co-designed mechanisms for healthier habits.</td>
<td>Improved understanding of carbohydrate counting and the impact of food on blood glucose, increased confidence, and improvement in blood glucose control.</td>
</tr>
<tr>
<td>2011. Children with Diabetes Workshop. Parents of children with type 1 diabetes and adult patients with type 1 diabetes.</td>
<td>After introducing the coping and flourishing mindsets, the provider asked, “What is one positive thing that diabetes has given you?”</td>
<td>All 35 participants wrote one positive thing on pieces of paper; all stood, and smiling, shared what they had written, using a microphone.</td>
</tr>
<tr>
<td>2010. Diabetes Sisters Workshop. 100 female participants with either type 1 or type 2 diabetes.</td>
<td>Participants worked in pairs, sharing a difficulty in their lives that they had overcome. Partner listened for strengths. Three stories were shared with the group, noting how the identified strengths could facilitate better diabetes management.</td>
<td>Participants felt pride in recalling their successes and recognizing personal strengths. New insights were gained regarding how to resolve issues and to use strengths to improve diabetes management.</td>
</tr>
<tr>
<td>2013. Two-day workshop for the Diabetes Prevention &amp; Treatment Program for the Pascua Yaqui Tribe. First day for health care providers only. Second day for returning health care providers and patients they invited. Day one had 28 participants and day two had 52 participants.</td>
<td>Health care providers learned empathic listening techniques, identifying strengths, and coaching practices as well as how to apply these with their patients on day 2. Patients were asked to identify one good thing diabetes has given them.</td>
<td>Health care providers were invigorated by the approaches they learned/practiced. Patients identified personal successes and internal resources and expressed pride, increased confidence, and a renewed desire to improve their diabetes care. Each patient offered one positive thing diabetes had given them. Energy was high.</td>
</tr>
</tbody>
</table>

Clinical Applications
Table 2 illustrates samples of the first author’s experience in applying the flourishing approach in her workshops and coaching practice.

Conclusions
Based on emerging scientific evidence and anecdotal reports, the addition of a flourishing approach to existing treatment and coping strategies can generate new and effective methods of support and promotion of well-being in people with diabetes. Health care professionals can incorporate the strategies related to the flourishing mindset to leverage the expertise of patients and collaboratively find ways forward that are context-sensitive.

What remains is an urgent need for scientific and clinical research to analyze and evaluate the impact of this approach on self-efficacy, clinical
A Flourishing Approach...  

continued from page 14

outcome indicators such as the glycated hemoglobin, and long-term sustainability. In addition, implementation of the flourishing approach to treatment may challenge the skill sets of some health care professionals, which requires further research.

References

Tips for Working From a Flourishing Mindset

1. Begin each session asking, “What’s improved since we last met?” This encourages the patient to reflect on successes, thereby guiding the visit in a positive direction.
2. Ask the patient to share a challenge or difficult life event and describe the steps he or she took to overcome it. Listen for strengths that were used, provide congratulations, and ask, “How can you use these strengths to help improve your diabetes management?”
3. When looking at a patient’s logbook or discussing proposed nutrition interventions, focus on what he or she is doing well, such as blood glucose numbers that are in range or the two vegetables a week he or she does eat. Ask “How did you do this?” and “What can you do to make this happen more often?”
4. Provide patients with suggested areas where improvement is needed and ask them to identify areas of focus and goal setting. Patients are more likely to be successful when they feel ownership for the goal. Discuss ideas for improvement and encourage the patient to implement one or two of them. Even if the selected approach(es) is/are not successful initially, the patient is more likely to engage in alternative approaches and future recommendations by the health care provider, if given the opportunity to choose.
5. Be present, attentive, and mindful in your visit with a patient. Show genuine curiosity and interest. As is often quoted in medicine, “Patients don’t care how much you know until they know how much you care.”


Academy’s Find a Registered Dietitian Nutritionist—Behavioral Health Expertise

The Academy of Nutrition and Dietetics’ Find a Registered Dietitian Nutritionist online referral service allows consumers to search a national database of Academy members for the exclusive purpose of finding a qualified registered dietitian nutritionist who is right for them. Behavioral Health has been added as an additional “expertise” area for consumers to select during their online search. Market yourself and your expertise to thousands of potential clients/patients.

Academy/BHN members in the Active category can sign up for Find an RDN at www.eatright.org/myAcademy.
Student Corner:
Nutrition Therapy for Alcoholic Liver Disease (ALD):
Current Practice and Future Research
Ariana L. Fiorita

Introduction & Background:
The liver is a critical organ in the human body, and without a healthy liver, we would not be able to survive for a long duration of time. The liver is the largest glandular organ in the body and provides a variety of different functions to keep our bodies up and running. The liver weighs an average of about three pounds and is responsible for producing bile to digest fats, detoxify the blood, and provide a storage site for certain vitamins and minerals, among many other vital functions.1,2

Alcohol is one of the most abused drugs throughout our history and today; however, it was not until the 1960’s that alcohol was proven to be toxic to the liver.3 From the 1970’s, mortality from alcoholic liver disease (ALD) was decreasing, presumptively due to stricter regulations on the sale of alcohol and also more public education about the risks and detrimental effects that alcohol has on the body.3 Unfortunately, in the past decade, the numbers of individuals dying from ALD is back on the rise.4 In the United States, about 60% of individuals drink and about 8-10% of those who drink admitted to consuming two or more drinks per day, putting them in the high-risk-drinking category. Deaths related to alcohol are the third most prevalent cause of death only behind smoking and hypertension.3 As of 2006, research shows that 7-9 out of 100,000 individuals die due to alcoholic liver disease in the United States.4 The World Health Organization (WHO) has set a goal to reduce mortality rates from alcoholic cirrhosis to less that 3.2 people out of 100,000 by 2020.5

Alcohol has a number of adverse effects on the liver when consumed in excess. The majority of individuals who are considered heavy drinkers (2-3 drinks/day) will develop fatty liver, 10-35% will develop alcoholic hepatitis, and about 8-20% will progress to developing liver cirrhosis.3,4 Liver cirrhosis occurs when the tissue of the liver becomes scarred as a result of disease.2 Alcohol is the second biggest cause of liver cirrhosis after hepatitis C, contributing to 20-25% of all cases and 50% of hospital admissions for liver cirrhosis.3

Abstinence is the only way to completely prevent alcoholic cirrhosis,3 however, what about the individuals who already have liver cirrhosis? Of course, sobriety from alcohol is the top priority, however, due to the addictive effects, this is evidently easier said than done. Consequently, the prognosis for this disease is not always hopeful. Liver disease without the presence of co-morbidities is a great indication for transplantation; however it is often not an option, due to strict guidelines for acceptance to the transplant list. Patients with liver disease have exhibited a high survival rate post-transplantation; however, transplantation in this population remains a controversial and ethical dilemma.5,6

Patients with alcoholic liver disease often receive pushback and delays in being added to the transplant list due to the belief that ALD is a self-inflicted diagnosis. There is also a question of the presence of alcohol-related damage in other parts of the body, such as infection from existing hepatitis C, autoimmune disease, malignancies and other organ damages.6 Most importantly, there is questionable compliance to post-operative recommendations following a liver transplant in some patients. It has been documented that many patients started drinking again after transplantation. According to a study published in the Journal of Hepatology, out of 424 patients who received a liver transplant, 24.3 % continued to consume alcohol excessively, 12.4% consumed alcohol on occasion, and 7% had an accidental slip, while 53.3% remained abstinent.7 The average period of abstinence before consumption of alcohol post-transplant was 37.1 ± 32.5 months.7 This is a very controversial issue due to the shortage of livers available to patients who need a transplant. Without a transplant, the five-year survival rate is as low as 23%.5 It is not a secret that this remains a problem in our healthcare industry, requiring a need for alternative methods of treatment for patients with alcoholic liver disease who are ineligible for transplantation. Alternative therapies and treatments can increase the potential survival rates among this population.

Current Nutrition Therapy for Alcohol Substance Abuse
Malnutrition is a large concern in this population, stemming from a myriad of nutrition-related issues including inadequate dietary intake, metabolic changes, gastrointestinal issues, and reduced absorption and increased excretion of nutrients.8 Specifically, alcoholics experience increased liver enzymes, increased immunodeficiency, vomiting, diarrhea, preoccupation with body appearance, bleeding gums, sore tongue, shortness of breath, and unusual muscle weakness.8 It is not uncommon for alcoholics to consume the majority of calories per day from alcohol. For this reason, over and above recommendations for abstaining from further alcohol consumption, current nutrition therapy includes implementation of a meal plan that incorporates a variety of nutrients, encourages balanced-meals at regular intervals, and oral supplementation based on nutrition assessment and laboratory results.9 Oral supplement recommendations include thiamin, folic acid, riboflavin, vitamin B-6, vitamin B-12, vitamin C, vitamin D, vitamin A, iron, magnesium, selenium, and zinc.9 Several alternative supplements have also been noted as possible therapeutic agents such as milk thistle and S-adenosylmethionine.9

Furthermore, it is fairly common for recovering alcoholics, who are
abstaining from alcohol, to fulfill their addiction in another way. Specific to foods, the addiction is often fulfilled in the form of sugar or fat-laden foods and caffeine.8 Consequently, it is important for dietitians to encourage infrequent use of added sugars due to the increased risk in this population for hypoglycemia, low to moderate fat intake, and weight gain during recovery from alcoholism.9 Due to the addictive nature of alcoholism and to encourage a healthy body weight, The Academy’s Nutrition Care Manual recommends increased complex carbohydrates, protein, and fiber with a moderate to low fat intake due to the tendency for this population to overeat or experience compulsive overeating while in recovery.8 Due to the increase in mortality rates, and the World Health Organization’s (WHO) initiatives to reduce deaths from alcoholic cirrhosis, research has provided some innovative therapies that may hold promise to this high-risk population.

Saturated Fats and Liver Disease

The last decade of research has provided some interesting insights for patients with liver disease. Studies in mice have shown that saturated fatty acids have a protective effect against the development of alcoholic liver disease. Although the mechanism is not entirely clear, researchers have found several pathways that saturated fatty acids may target with potential methods of protection against the development and progression of ALD.

A 1998 study revealed that countries where a higher fat diet is consumed have shown to have a lower prevalence of alcoholic liver disease.10 A study that was published in 2004 in mice revealed that dietary saturated fat reduced serum triglycerides, polyunsaturated fatty acids, and total free fatty acids.11 A more recent study, published in 2005, exhibited that alcoholic liver disease can be reversed in animals with an increase in dietary saturated fat even with continued alcohol consumption.10 An increasing amount of saturated fat consumed in the diet is thought to increase the liver’s resistance to oxidative stress. As the saturated fat intake was increased to about 30% of the diet, steatosis of the liver decreased.11 Therefore, the studies suggest an inverse relationship between liver pathology and saturated fat intake.

Another proposed mechanism involves a hormone known as adiponectin, which is solely expressed and excreted in adipose tissue.12 A promising therapeutic strategy may involve the nutritional modulation of this protein hormone because circulating levels of adiponectin were significantly elevated by chronic alcohol consumption in mice consuming a high saturated fat diet. Delivery of adiponectin to mice who also consumed a high saturated fat diet, exhibited reduced injury and steatosis in the liver.12 In a separate study using middle-aged human subjects, moderate alcohol consumption was shown to elevate plasma adiponectin.12 Therefore, it was proposed that circulating adiponectin might be gauged on the amount of alcohol that is consumed. Manipulation of adiponectin may be an attractive therapy for these individuals by alleviating alcoholic fatty liver through the up-regulation of adiponectin in adipose tissue.12

On the contrary, polyunsaturated fatty acids have shown to promote the development of alcoholic liver disease,11 which is the opposite of what most people without liver disease practice for a general healthy diet. Although polyunsaturated fats have shown to promote the development of alcoholic liver disease, ingestion of a high concentration of dietary saturated fat can reduce steatosis even when unsaturated fats remain part of the diet.11

What does this mean for dietitians?

What is the role of the dietitian in addressing nutrition issues in this patient population? Should we tell our ALD patients to eat as much saturated fat as they can because it is “protective” and will reduce their disease if they change nothing else? The answer is of course not. Since alcoholic patients are faced with malnutrition related to nutritional deficiencies, including vitamins and minerals, and fat malabsorption, among an array of other issues, it is important for the dietitian to address these concerns in an overall, evidence-based manner. Currently, answers may include nutrition supplementation, especially of fat-soluble vitamins,7 and encouraging intake of calories from nutrient-dense sources since the majority of alcoholics consume most of their daily calories in the form of alcohol.8 More research is warranted regarding ingestion of dietary saturated fatty acids, however, it cannot be discounted. Following current guidelines and keeping up with the recommendations is critical in the ever-evolving field of dietetics.

About The Author

Ariana L. Fiorita is a dietetic intern at the Cleveland Clinic. She graduated from Ohio University with a bachelor's degree in Food and Nutrition Sciences. Ariana can be reached via email at ariana.fiorita@gmail.com.

References

1. that the participants will be able to:

2. Expand members’ awareness, utilization, and development of business and management resources.

3. Develop strategies to utilize, expand and sustain business and management skills.

4. Apply business and management skills in all areas of practice.

5. Recognize, seize and create business and management opportunities.

Business and management skills span all areas and levels of practice and help elevate the profession of nutrition and dietetics. Many of our members miss opportunities for advancement in their jobs or other opportunities, due to a lack of business and management skills. Dietitians are entering private practice in increasing numbers where business and management skills are essential. Opportunities for using management skills in grant writing and creating new service delivery programs abound in almost every area of nutrition and dietetic practice.

Three highly successful dietitians are described in the HOD background paper that you will find most remarkable. They are Judy Bonner, PhD, Ellyn Elson, RD, and Jane Boudreaux, PhD, whose stories inspire all of us with their expertise in business and management. I worked with Judy Bonner, PhD, when she was a dietitian in the pulmonary program at Children’s Hospital of Alabama, little knowing that she would become the first woman president of the University of Alabama. Judy stresses the importance of networking, strategically learning the business side of your department and thinking creatively, being a team player and leader, and seeking continuing education in the area of business and management. Ellyn Elson is the founder of Computrition© and well-known as an entrepreneur. Ellyn stresses the importance of communication, marketing, and taking business courses to understand the financial aspects of a business. Jane Boudreaux has served as department head and dean of the school of Health and Human Sciences, University of Southern Mississippi and now is head of three nutrition consulting companies.

The second mega issue to be addressed in the HOD is the practice issue involving lack of internships in dietetics training programs. There has been a great deal of discussion thus far around this issue as well as proposed solutions.

I look forward to feedback from you on these two issues in advance of the meeting of the HOD, October 17 and 18. Please respond to the Mega Issues survey sent out in September and to the information placed on the BHN website www.bhndpg.org and Facebook. If you want to respond directly, my email is harriet.h.cloud@ gmail.com.
BHN Member Spotlight!

Eating Disorders Resource Professional

Marci Anderson MS CEDRD LDN CPT
eatingdisorderresourceprofessional@bhndpg.org

Hi BHN members! Thank you for taking the time to learn a little about me and my role as BHN’s Eating Disorder Resource Specialist. My name is Marci Anderson and I am passionate about working with people who are working towards recovery from an eating disorder. I own a group nutrition practice in Cambridge, MA where we help clients heal their relationship with food, exercise, and their bodies. I am 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 my team, I am an adjunct professor for Plymouth State University’s Eating Disorder Institute as well as for Northeastern University in Boston. I have spoken nationally on the topics of eating disorder recovery, Intuitive Eating and the role of the RDN on the eating disorder treatment team. I love social media so tweet me @marciRD. I have also developed a comprehensive training program for RDNs and dietetic students interested in treating eating disorders. I strongly believe in educating RDNs in the art of integrating therapeutic concepts into nutrition counseling.

As your Eating Disorder Resource Professional I hope to support each of you in your work. Whether it’s sharing research articles directing you to resources or answering questions, I’m here for you! I’m also eager to learn from your expertise and knowledge as well. Feel free to contact me directly: marci@marciRD.com, and I highly recommend posting questions directly on the EML. If you have a question or concern, many others probably do too!

Currently, BHN is working on developing Fact Sheets for RDNs. We hope these fact sheets will support and advance the work you do. If you have ideas of what ED related fact sheets you would like to see us provide, please email me privately. I would love your thoughts and feedback as we work on developing resources for our professionals.

In the BHN Pipeline!

Please Join BHN in Atlanta!

BHN Member Appreciation Breakfast
When: Sunday, October 19, 2014; 7:00AM - 8:30AM
Where: Spruce room at the Omni at CNN Center
Program:
- Lee Shelly Wallace, MS, RDN, LDN, FADA, FAND presenting How Adults Learn (1 Hour CPE)
- BHN Awards and Recognition

FNCE Spotlight Session
Topic: Dysphagia, Mealtime, and Intellectual and Developmental Disabilities
Speakers: Jennifer Meyer, MA CCC-SPL; Joan Guthrie Medlen, MEd RDN LD;
Moderator: Sharon Lemons, MS RDN, CSP, LD, FAND
When: Monday, October 20, 2014 from 8:00AM - 9:30AM
Where: Georgia World Congress Center / Room: Thomas Murphy Ballroom 1-2
Objectives:
- Compare the relationship between the gross motor skills and oral motor feeding skills related to dysphagia
- Apply SOP/SOPP to the RDN responsibility in identification and treatment plan for dysphagia in persons with IDD across environments and lifespan through case studies
- Discuss the importance of addressing sensory and motor components of dysphagia before implementing a primarily behavioral program for persons with IDD across the lifespan

BHN Showcase, Booth #2
When: Monday, October 20, 2014; 9:30AM - 12:30PM
Where: Level 3 Foyer, near the educational sessions and the escalators of the Georgia World Congress Center
Unzip Your Minds with BHN!
- Stop by to see what’s new with BHN
- Receive a complimentary coin purse
- Visit with fellow members and officers
- Browse our resources and new member Fact Sheets

Congratulations to this year’s BHN Award Winners!

Distinguished Member
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Excellence in Practice
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Eating Disorders • Rebecca Bitzer, MS, RD, LD

Please join us at the Member Breakfast at FNCE® on Sunday, October 19th at 7am at the Omni Spruce Room for the presentation of awards
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A complete list of BHN Executive Committee members and volunteers is available at www.bhndpg.org.

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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