Case Studies: Lessons From Practice

Editor’s Note: The term “Case Study” has been defined as: “A process or record of research in which detailed consideration is given to the development of a particular person, group, or situation over a period of time”. The following case study is 1 of 6 recently written and published by BHN members who have given detailed consideration of a particular client and their approaches to nutrition therapy. Case studies are an excellent opportunity for practitioners to learn best practices from each other and acquire knowledge and techniques that have been successful for other professionals. Our thanks to these dedicated RDNs for sharing their studies. More like these may be found on the BHN website.

A hypothesis-based intervention for this adult patient, diagnosed as an infant with a rare heritable enzyme deficiency, was influenced by timing and situational circumstances.

**Objectives:** Upon completion of reviewing this case study, the participant will be able to

1. Describe the hypothesis underlying the trial nutrition intervention,
2. Describe use of selected nutrient supplementation
3. Describe barriers to completion of the study.

1. **Anonymous ID Number:** 2016-1
2. **Primary Behavioral Health Category:** ☐ AD; ☐ ED; ☒ IDD; ☐ MH
3. **Age:** 54
4. **Gender:** Female
5. **Diagnoses:** Pyruvate Kinase Deficiency (a rare diagnosis) See also: Supplementary Material – Literature Review
6. **Medical Conditions:** Hemolytic anemia requiring blood transfusion. Due to the number of blood transfusions the individual has received, it is now difficult to find an acceptable blood match due to antibody formation and can take 2-3 days before an acceptable match can be located. Decreasing the frequency of transfusion might decrease this problem but would be ideally started early in life. While documentation is limited, there has been about three years between the most recent transfusions.
7. **Medications:** Rx: Folate; 1 mg. Supplemental iron is contraindicated – no other supplementation has been required to date.
8. **Relevant Family History:** Parents are carriers; has cousin born in 1990s who also has PKD without developmental disability. See also: Supplementary Material - Additional Family History
9. **Relevant Laboratory Results:** Hb 9.3, HCT 28.1 See also: Table 1. Higher than normal young erythrocytes are typical for patients with hemolytic anemia.
10. **Nutrition Physical Exam:** Height: 62”; Weight: 193.6 lbs; BMI: 35.4;

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From the Chair
Diane Spear, MS, RDN, LD, FAND

While some people may appear to be “born leaders” most leaders are “made” by learning, listening and doing what they were meant to do, especially when passionate about it.

Leadership is not just big acts by single individuals with big titles. Everyone has some leadership capacity and can lead and contribute in many ways. Each of us has within us the power and the responsibility of leadership. How we use this power and fulfill our responsibility is up to each of us.

Leadership opportunities pop up around us at every turn, sometimes when least expected. Former Academy President, Sylvia A Escott-Stump, MA, RD, LDN once noted that most effective leadership traits are:

• Being committed to being the best;
• Encouraging input from others;
• Being open-minded;
• Not micromanaging others;
• Being a “calculated risk” taker;
• Having futuristic thinking, and
• Keeping the whole picture in mind, not just your own sphere of influence.

As practitioners in behavioral health nutrition, these leadership traits are especially critical in our everyday practice approach to counseling and delivery of nutrition services. Successful leadership is ultimately about relationships; to recognize great ideas, support those ideas, get everyone aligned toward the same goal, and break down barriers along the way.

The Academy of Nutrition and Dietetics defines leadership as “the ability to inspire and guide others toward building and achieving a shared vision.” In BHN, we share a common vision for optimizing the physical and cognitive health of those we serve through nutrition, nutrition education and behavioral health counseling.

BHN is invested in cultivating leaders within the organization. By virtue of our various roles in behavioral health, we recognize and value the abilities of our peers and respect the knowledge and skills of others regardless of ability and barriers to personal success. In BHN there is something for everyone who desires to foster their leadership potential.

Are you interested in shaping the future of practice in behavioral health nutrition? Ask for a leadership role in BHN, be it large or small, there are many ways to volunteer. Get connected by visiting our webpage www.bhndpg.org that highlights volunteer opportunities, contact BHN at nominatingcommitteechair@bhndpg.org or contact any of the officers or members of BHN’s executive committee.

Your Chair,
Diane Spear
Case Studies: Lessons From Practice  
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11. Reported Diet & Supplements:  
Attempted trial diet intervention: low simple carbohydrate diet; Supplement: folate

12. Information/Hx from Consults/Referrals: Pt. was born at healthy, normal weight one year after Pyruvate Kinase deficiency was identified and published in 1961. On the day after birth, bilirubin levels were high, and she was transported to Children’s Hospital.

She was officially diagnosed at age 18 months, with pyruvate kinase enzyme levels reported at “10% of the normal”. Brief notation shows bilirubin ordered on day of birth and the following day. After initial diagnosis, mom reports she took the patient to Children’s Hospital about every 2 months for further research. A splenectomy was not determined to be a required treatment. Her parents were told she would not live past the age of 5 years. She received 3 to 4 blood transfusions annually until approximately age 40 years when menses ceased. For about the last 10 years, transfusions were yearly to every 2 years.

Prior to Patient’s birth, neither family was aware of being carriers of the recessive trait. Since Patient’s birth and diagnosis, her siblings have been tested, with one being a carrier and one not. Pt. also has a second cousin diagnosed with PK deficiency; he is a teenager, has never had a transfusion and does not have a developmental disability. Pt. and her cousin are the only individuals with PK deficiency we are aware of in the area. The family believes Pt’s developmental disability is due to lack of oxygen during her initial treatment.

Developmentally, Pt. did not learn to walk or potty train until her next younger sibling began developing the skills and Pt. imitated her sibling. This delay was related to the developmental disability rather than PK deficiency. Due to mom’s advocacy, Pt. attended public school in a “trainable” class, for children determined to only be capable of being trained in everyday activities, such as washing dishes, brushing teeth, etc. By age 17, Pt. was only able to speak 1-2 words, and had been tested by the school system as functioning at a 4.3 years-old-level. Pt. is now able to communicate verbally with limited language. She is also able to write her name, although her skills have diminished with age. Due to her disabilities, Pt. has received state-sponsored services since she was 18 years old. Medicare and medicaid provide medical services. Private insurance has never covered services, and mom reports the father was once told that if he added Pt. to his insurance, he would lose his job. The reason for private insurance refusal was listed as “pre-existing” condition.

When Pt. was about 8 years old, she began having grand mal seizures at a rate of 9 to 10 per month. Last month, Pt. had 3 seizures in one week, but now typically has multiple seizures only one week out of a month with the remaining weeks being seizure-free. Seizures have decreased in intensity, with her often having a small seizure without losing control of her functions nor resulting in falling. Seizures are usually accompanied by angry outbursts, which is not a normal trait for Pt.

Per mom’s report, a neurologist thought seizures might be due to her PK deficiency. A physician letter from 1984, stated several medications were tried for seizure control but resulted in “intermittently good and then poor control”, and this physician felt she would always have difficulty controlling seizures.

Pt.’s last transfusion was in June 2016, after about 2 years without a transfusion, per mother’s report. Pt. has taken folate supplements since birth. At one time, a physician tried steroid treatment to keep blood levels higher without success. Pt. began having problems with weight gain as an adult, possibly due to changes in eating habits with the introduction of staff into her care. Physical ability, seizures, and lack of motivation limit her exercise.

13. Relevant Observations: while PKD was noted as a medical diagnosis in the patient’s chart, the direct care team did not realize it was involved in carbohydrate metabolism prior to this intervention.

14. Nutrition Diagnoses: Decreased ability to metabolize glucose related to Pyruvate Kinase Deficiency as evidenced by breakdown of red blood cells due to decreased ATP production and causing anemia requiring blood transfusion. PKD possibly fits under Nutrition Diagnosis NC 2.1 – Impaired Nutrient Utilization; the nutrient being glucose. Addressing the fact that it is genetically related might be useful in future nutrition diagnoses options, such as “changes in metabolism related to genetic influences”.

15. Guidelines or Criteria Utilized: Guidelines for dietary intervention are not available in the literature. This intervention was based on a hypothesis, with the criteria for success being a decrease in the rate of blood transfusion to longer than 2 years between transfusion.

16. Nutrition Care Plan  
A. Because pyruvate kinase is involved in converting glucose to pyruvate and then used to produce ATP (energy), the theoretical basis for the diet intervention was to limit total carbohydrate in the diet to 45 percent of kilocalories (lower limit of AMDR for carbohydrate) and to decrease intake from sugar, refined flour and other starchy foods.

Our goal was to test whether controlling carbohydrate intake would decrease the frequency of required blood transfusions. At this time, decreasing the frequency of transfusion is the goal for this individual. To date there is not a cure for this condition but limiting symptoms could increase quality of life for those who have the condition. This care plan was in place for less than one year at the time of the case study report, due to the RDN being on the case for 15 months, but the intervention is planned to continue.

B. A second nutrition intervention was to encourage as much exercise as tolerated for two purposes:  
continued on page 4
weight loss for overall health, and mobility. For this individual, excess weight was a concern to her parents and a concern for limiting her health risk due to overweight in the future.

17. Patient Response: Documented meal intake revealed carbohydrate intake decreased from about 60% of calories to an average of 50% of caloric intake. In the last 2 months prior to reporting results, dining out decreased to 1-2 times per week, making it easier to limit carbohydrates, and sugary foods decreased. Weight began at 194 pounds, decreased to 189 pounds prior to excessive staff turnover, and then increased to a peak of 200 pounds; now returning to 193.6 pounds.

While Pt. lives in her parents’ home and mom purchases her groceries, most of her meals are prepared by staff who receive 30 minutes of training on the diagnosis and diet, with no other specialized training in providing meals for an individual with her diagnosis. Menus were provided but strict compliance could not be enforced.

Some compliance issues were: 1) due to her developmental disability Pt. does not understand her diagnoses and requested and received foods that were high in simple carbohydrates, 2) staff turnover was high during the test period so staff were often not trained nor motivated to take time to cook a meal at home and Pt. was often dining out unrestricted approximately 3-4 times per week. One staff believed she was being nice by bringing Pt. donuts for breakfast.

18. RDN Response/Expectation: During the 6-month intervention, we were unable to get adequate compliance to determine whether intervention was effective in decreasing need for transfusion. The RDN is continuing this intervention as it is a healthy diet and may help assist with weight loss, along with the possibility of decreasing the rate of blood transfusion.

19. Follow-up/Progress: Table 1. Bloodwork through Adulthood ranges are typical for young erythrocytes (reticulocytes) in patients with hemolytic anemia.

20. Lessons Learned from this Case: To pursue this further, finding children who had been recently diagnosed would be a better population in which to try an intervention, as parents would be motivated to comply. Beginning intervention at a young age would have been more helpful, as this client had habits (eating high carb), and had so many transfusions, that it was difficult to determine whether we could improve quality of life. The RDN has been on this case for approximately 15 months, which is an inadequate time to attempt intervention for 2 years or more. This was an IRB-reviewed time-limited class project. Because the individual’s blood transfusion rate has decreased to about once every two years, an intervention would need to run for over 2 years to assess whether decreasing carbohydrates in the diet would decrease the frequency of transfusions. Finding documentation of labs, interventions, and exact dates of transfusion over the fifty years of treatment was difficult due to detailed records not being kept by the family, and the physician offices purging files after a period of time.

It is suggested that an additional category of “Nutrition Diagnostic Codes for conditions related to demonstrated genetic variation, which can be modified by nutrition interventions”, be considered for use by RDNs.

Table 1.

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Supplementary Material – Literature Review

Pyruvate Kinase (PK) is an enzyme in glycolysis, thought to be the major regulatory enzyme. Clinical features vary. A review focusing on genotype/pheno-type categorizes symptoms into “severe, moderate, and mild”; the “severe” definition best fits the patient in this case study. “Severe” phenotype typically has diagnosis in childhood, with most patients presenting with severe neonatal jaundice and most requiring exchange transfusion and possible splenectomy (Zanella, 2007). Infants may have hyper-bilirubinemia. Infants and children may require regular blood transfusions. Splenectomy may decrease transfusion needs. Anemia tends to stabilize in adulthood, but may be exacerbated by acute infection, stress, and pregnancy (Oski 1973). While fatigue would be expected with decreased ATP production, it has not been reported with these patients, and they are more exercise tolerant than would typically be expected (Oski 1973). Hemolysis can be triggered by some medications and fava beans (NORD 2016).

Red blood cell destruction appears to affect young cells, with more mature erythrocytes surviving well (Mentzer, 1971). Decreased ATP levels cause irreversible membrane damage, resulting in premature RBC destruction in the spleen and liver (Aizawa, 2003); it also causes decreased capacity for RBC to protect itself against free radicals and oxidative stress (Olivier, 2015). Oxidative phosphorylation compensates PK-deficiency by maintaining ATP levels (Mentzer, 1971). The dominant isoenzyme in fetal form is PK-M2 (Zanella, 2007), and may account for the variability in the severity of anemia (Zanella 2005).

At the current time, treatment is supportive rather than curative and focuses on treating the hemolysis. This typically involves transfusions of varying frequency to maintain an adequate hemoglobin. In these patients, hemoglobin stabilizes at about 6-8 g/dl (Zanella 2000). Some “severe” cases require splenectomy. With rapid red cell turnover, supplemental folate or multivitamin containing folate is indicated (Zanella 2000). Supplementation with iron is contra-indicated, and over 50 percent of patients experience iron overload resulting from breakdown of red blood cells (Zanella 1993). Attempts to supplement with riboflavin and sulphydryl compounds has been unsuccessful (Zanella 1976 & Staal 1975). Left untreated, death typically occurs before age 4 years (Bowman, 1965). Iron overload appears to involve hemolysis, ineffective erythropoiesis, splenectomy and hemochromatosis (Zanella 2001), along with transfusion dependency (Mojzikova 2014). Hepcidin (negative regulator of iron absorption and recycling) levels are reduced in patients with PK deficiency (Mojzikova, 2014). There is one reported case of bone marrow transplant in a child with PK deficiency who now has normal hemoglobin and red blood cell PK activity (Tanphaichitr, 2000).

References


Supplementary Material – Additional Family History

Pt’s cousin had a child in the late 1990s, also diagnosed with PK deficiency. He weighed 6# at birth and was jaundice; he went home on a normal schedule. During the first 2 weeks after birth, mom reports he was lethargic. When she took him to the physician he was given iron supplement, but this resulted in projectile vomiting. By 1 month old, he had lost weight to 4# 10 oz.; he remained undiagnosed. At this point the family began to suspect he might have pyruvate kinase deficiency. They were sent to a blood specialist at Children’s Hospital, blood was drawn and sent for analysis; diagnosis was confirmed. The cousin reports her son was susceptible to illness through childhood, did not suffer from developmental disability, has his spleen and has never received a blood transfusion. He has been able to live a normal life, including playing multiple sports. By age 9, the physician reported they no longer needed to be followed for PK deficiency, and no related problems have been noted since this time.

Check one of the following:

☐ HIPAA identifiers, including unique patient characteristics were removed prior to submission and publication.

☐ Consent form has been signed by individual or responsible party and retained by the author for future reference.
CPE Questions for Case Studies: Lessons From Practice

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Objectives: Upon completion of reviewing this case study, the participant will be able to
1. Describe the hypothesis underlying the trial nutrition intervention,
2. Describe use of selected nutrient supplementation,
3. Describe barriers to completion of the study.
4. The theoretical basis for the diet intervention in PKD is to
a. Increase insoluble fiber to treat constipation.
b. Increase iron rich foods and ascorbic acid.
c. Limit the sodium and potassium to reduce stress on the kidneys.
d. Limit the total carbohydrate in the diet to 45% of kcalories.

1. The hypothesis for the trial intervention was based on the fact that a Pyruvate Kinase deficiency results in
   a. Decreased ability to digest protein.
   b. Decreased ability to metabolize glucose.
   c. Decreased absorption of iron.
   d. Inflammation of the immune system.

2. Supportive treatment, rather than curative treatment, for this case
   a. Consisted of Iron supplementation due to low hemoglobin levels.
   b. Consisted of supplementation with folate due to rapid cell turnover.
   c. Was intended to reduce the frequency of transfusions.
   d. b and c

3. Providing appropriate menus for the trial nutrition intervention
   a. Requires ability of the patient to understand the purpose of the intervention.
   b. Requires adequate support staff training.
   c. Requires sufficient duration of trial period to yield the potential effect.
   d. b and c

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While most of us do not work with alcoholics per se, many of the patients we see do imbibe varying amounts of alcohol. There is ample literature on alcoholism and nutrition. But what do we know about more moderate “social” drinking? How much is too much? What are the health implications, if any? Is there an association between alcohol use and dietary intake? What is our role, as RDNs, in counseling moderate drinkers?

How much should we and do we drink?

The U.S. Dietary Guidelines consider a “standard drink” as 5 fl oz of wine, 12 fl oz of beer, or 1.5 fl oz of distilled spirits. Each contains ~15 g of alcohol. Moderate drinking is defined as up to 2 drinks/day for men and up to 1 drink/day for women. Low-risk drinking is no more than 4 drinks on any day for healthy men under 65, and no more than 3 drinks on any day for men over 65 and healthy women. Both low-risk and moderate definitions include an upper limit of 14 drinks a week for men and 7 a week for women. This takes into account that most people drink more on weekends than weekdays and holidays.

In recent years, consumers have been bombarded by media trying to convince them to drink alcohol for a variety of alleged health benefits. Not exactly peer-reviewed journals, such articles often tug at emotional issues, cite a glimmer of truth (such as antioxidants or esoteric animal studies), and proceed to imply that champagne can help with dementia, red wine is good for your heart and cures impotency, beer promotes longevity, and so on.

Indeed, NHANES data (2009-2010)\(^1\) found that on a given day 17.6% of men and 10.6% of women exceeded the Dietary Guidelines for alcoholic beverage consumption. Data from 2 NIAAA (National Institute for Alcohol Abuse and Alcoholism) surveys comparing volume, frequency, and binging vis a vis various alcoholic beverages in 2012 to 2001 found that the drinking climate is becoming considerable “wetter”\(^2\) with fewer lifetime abstainers. Although younger drinkers and men continued to have greater rates of drinking, women and older folks had greater increases (69.5 to 75.3% and 6.1 to 11.8%, respectively). Overall, wine and liquor intake was up, while beer was down.

Health Effects of Alcohol

We know that alcohol affects every organ system of the body. It can cause liver, pancreatic, and GI disease, compromise immune function, potentiate cancers, contribute to muscle wasting, and predispose to metabolic syndrome. The preferential treatment of alcohol calories by the liver can lead to both primary and secondary malnutrition.

Alcohol has been touted as protective against cardiac disease, diabetes, cancer, osteoporosis, and even dementia. In general, it can be considered a “tonic or toxin” with a dose-dependent U-shaped curve – health benefits until a certain point (1 – 3 drinks) and then increasingly toxic results at larger doses.\(^3\) Each person is a unique specimen - age, gender, body composition, genetics, health status, diet, and the type of alcohol consumed, all play a role in determining whether what is safe or even beneficial intake for one person, augurs health problems for another. For example, studies show that women are more vulnerable to the effects of alcohol than men, requiring less alcohol over a shorter period of time to be at risk for alcoholic liver disease and brain damage.

Encouraged by the myth that red wine is universally healthy, many people, trying to maximize their benefits, may drink several glasses a day. Doing the math for 2 glasses an evening for a year at 125 kcal per 5 fl oz serving, this adds up to an extra 26 lbs. on the body. Fourteen cans of Busch Ice per week would add another 35 lbs/year. This added weight alone is a health concern. At moderate levels, alcohol stimulates the appetite, is more likely additive rather than replacing nutritive kcals. It is efficiently metabolized, behaving like fat in promoting obesity, hence central obesity, or “beer belly.”

Using data from 4 cycles of NHANES (1999-2006), dietary intakes of over 16,000 men and women (classified as never, former, or current drinkers) were compared against the HEI (the USDA’s Healthy Eating Index) and adjusted for age, smoking, and BMI.\(^4\) Overall, none of the groups scored well but the diets of drinkers were even poorer with an inverse relationship between level of drinking and diet quality. For example, men who drank more had lower intakes of fruit and greater intake of saturated fat; the more women drank, the lower their intake of whole grains and milk products. The article concludes that some of the adverse health consequences of heavy alcohol use may be due to poorer diet quality.

What’s an RDN to do?

(Our mission)

Traditionally,\(^5\) the role of the dietitian in alcohol treatment programs includes all the usual clinical tasks: assessment and nutrition care plan, provision of meals and supplements, encouragement of normal eating patterns, dietary counseling for specific comorbidities, and education on the role of diet in recovery. We know that diet can either support or sabotage one’s program of recovery so it is vital to incorporate nutrition into the whole picture of recovery, seeing it as a means of self-nourishing, self-care and skills for successful independent living.

But what do we offer more moderate drinkers? Our first course of action is to incorporate screening for alcohol intake into our routine. Research has shown that self-reports are more accurate when alcohol is included as part of a general food frequency questionnaire or 24-hour recall rather than as an alcohol-only survey.\(^6\) You may need to prompt about frequency, and amounts for each type of drink, using actual glasses to determine amount
Addressing Moderate Alcohol Use – Health, Calories, Assessment, and Counseling

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– much as you would do with food models. Inquire, as well, about “bar food” – those nuts and pretzels that often accompany alcoholic beverages. Attitude is key – inquire in a nonjudgmental, gentle manner, the same as you would for other ingestibles.

Become conversant with alcohol content, kcals, and terminology. Proof refers to alcohol content and is twice the percent alcohol in the product. For example, whiskey, gin, rum, and vodka are usually 40% alcohol or 80 proof. Wine is generally 8–14% alcohol; beer 4–9% with a trend towards higher alcohol content than several years ago.

At 7.1 kcal/gm, alcohol provides over 75% more calories than carbohydrates and protein. We already know that the standard 5 fl oz serving of wine is 125 kcals. Distilled spirits weigh in at 100 kcal for 1.5 fl oz. For mixed drinks, the sky is the limit for calories due to the multitude of added ingredients. Beer calories vary widely by alcohol content – the standard 12 fl oz serving is supposedly 150 kcals but IPAs and darker beers can pack as much as 300 kcals. And, of course, the devil is in the portion size. Some beers are now packaged in 16–20 fl oz bottles. And few people have a 5 fl oz line on their wine glass.

Unlike other ingestales, alcohol is not regulated by the FDA – no mandatory kcal or ingredient labeling is required – which can be a real problem for people with allergies, sulfite sensitivity, vegetarians, or other special needs. Contamination due to ants and bees that fall into vats can cause serious reactions in those allergic to insect venom. Products may contain animal and insect based ingredients such as gelatin and colorings.

As usual, results of the physical assessment can be helpful in formulating a nutrition care plan and as motivational strategies with your clients. Be sure to measure waist/hip circumference, BMI or other measures of body composition (indications of “beer belly” and muscle wasting), as well as simple visual inspection for muscle wasting, skin issues (rosacea, psoriasis, facial redness and bloating), as well as other outward manifestations of nutrient deficiencies common to heavy alcohol use. Blood chemistries, including HDL, liver enzymes, blood glucose, MCV, and vitamins A and D, may indicate heavy alcohol use, damage to the liver and pancreas.

Our first priority is to identify those who drink to excess and ensure that they are referred on for further assessment and treatment. We can help set moderate and low-risk drinkers on the right course by bringing alcohol use to awareness, educating about its health effects, dispelling myths, and guiding them to resources for cutting down on or lessening the health impacts of their drinking.

Personalize the results of the assessment. How does their intake compare to the official recommendations? How many and what percentage of their calorie intake is from alcohol? How does this translate into pounds? What is their safe level of intake? – what is safe for one person may not be for someone else. Are they taking any medications that adversely interact with alcohol? Do they have any medical conditions that are exacerbated by alcohol intake? Some people are motivated best by visual presentations of assessment results: graphs and pie charts of body composition, comparisons of calorie intake by macronutrient type, longitudinal lab values, etc.

For those who want to continue drinking, we can offer tips to lessen the health impact of their drinking by using what we know about alcohol metabolism.

1) Eat food first and snack while drinking to delay absorption. Up to 20% less alcohol is diffused through the wall of the full stomach which gives alcohol dehydrogenase time to break it down. Fasting, on the other hand, limits enzyme production so alcohol’s effects are felt more rapidly.

2) Pace yourself: Sip slowly – no more than one drink per hour. The liver can only process ½ oz of alcohol per hour. More than this and the excess alcohol and its intermediate breakdown product, acetaldehyde (aka vinegar), continue circulating throughout the body, destroying and pickling organs.

3) Take extreme care with medications. The same MEOS (microsomal ethanol-oxidizing system) metabolizes alcohol as well as other drugs. Unmetabolized drugs can build up and their effects amplified. When heavy drinking ceases, then the enlarged MEOS may metabolize medications too quickly.

4) Quench thirst with water. Alcohol depresses ADH (anti-diuretic hormone) so more fluid is lost by the body, leading to thirst. Plus, bartenders put out free salty snacks to sell more drinks. Advise a glass of water before each glass of alcohol.

5) Just say ‘know’ to binge drinking. It kills in many ways: an increase in risky behaviors leading to unprotected sex, accidents (motor vehicle and other), assaults, plus it can quickly anesthetize the brain’s respiratory center and breathing stops.

An excellent (free) on-line resource is “Rethinking Drinking” (rethinkingdrinking.niaaa.nih.gov). In addition to help in calculating how much you drink, calories, comparing intake to guidelines, etc., it offers helpful decision-making tools, tips on getting support, plus a Q&A section answering queries such as “why is being able to ‘hold your liquor’ a concern?” and “Why are women’s limits different than men?”

Another easily reproducible strategy is outlined by de Visser’s research.8 Two groups of students were asked to pour alcoholic drinks into glasses and guess how much they had poured. One group received feedback as to whether their guess was accurate or not; the other just poured. At two months, the feedback group had better knowledge of recommended intakes and actual lower intake.

Conclusions

Being proactive regarding alcohol intake is easy to incorporate into your practice. Include alcoholic beverages as part of your routine diet recall. Educate your patients about the health risks of alcohol use. Know when and where to refer patients on to resources and support for cutting down or quitting their alcohol use. Do not recommend nondrinkers start drinking for the health benefits!
Addressing Moderate Alcohol Use – Health, Calories, Assessment, and Counseling

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About the Author
Renée Hoffinger, MHSE, RD retired from the North Florida/South Georgia Veterans Health System in Gainesville, FL after 20 years. She helped pioneer hands-on nutrition education with the VA’s Substance Abuse Treatment program and disseminate such programs to VA centers nationwide. Since her retirement in 2013 she has been writing about and leading hands-on-nutrition education workshops: Hands-on Nutrition Education – Teaching Healthy Eating Skills through Experiential Learning, published by The Academy, debuted at FNCE 2017. Renée serves as the Addictions Resource Professional for the Behavioral Health Nutrition-DPG and has presented at many conferences and webinars on the topics of diet and substance abuse, HIV/AIDS, incorporating research into clinical practice, as well as hands-on nutrition education. She is also the author of The Recovery Diet (Adams-Media, 2012) and numerous articles for DPG newsletters and other publications.

References

Nutritional Aspects of Bipolar Disorder
by Ruth Leyse-Wallace PhD

Introduction
Bipolar Disorder (BD), also known as Manic-Depressive Disorder, is a condition known for wide swings of mood lasting weeks to months. The manic phase is described as having racing thoughts, increased speed of talking, grandiosity, inappropriate euphoria or elation, irritability, markedly increased energy, poor judgment (such as financial or sexual), and decreased need for sleep. The difference between mania and hypomania is “the degree of severity” as well as an absence of psychosis (delusions or hallucinations) in hypomania and no negative impact from “high” symptoms in work and social functioning.1-2 The depressive phase is described as having fatigue, difficulty making decisions, feelings of hopelessness, guilt, worthlessness, insomnia or oversleeping, digestive disorders, headaches, or chronic pain, and weight change due to either overeating or loss of appetite.

Genetics
Bipolar disorder is 80% heritable.3 Polymorphisms of genes related to B-vitamin absorption, metabolism and function have been linked to the increased incidence of psychiatric and cognitive disorders. Mitchell et al comments that effects in studies may be small and inconsistent and that future studies need to take into account diet and nutritional status of subjects.4 Folate-sensitive fragile genetic sites were found to be more frequent in patients with BD than in controls.5 Patients and relatives had specific alterations in MTHFR (methylene tetrahydrofolate reductase) and were shown to have elevated homocysteine, low folate and low vitamin B12.5

Co-morbidities
Metabolic syndrome is more common in those with Bipolar Disorder than controls or those with other psychiatric disorders. Silarova et al found that 28% of those with BD had Metabolic Syndrome, compared to 20.2% of those with Major Depressive Disorder and 16.5% of control subjects. Risk was also associated with higher depression scores and abdominal obesity.7 Bly also found the rate of metabolic syndrome was higher in individuals with bipolar disorder (33%) and schizophrenia (47%) compared to matched NHANES controls.8

Brain-derived neurotropic factor (BDNF) has been proposed as a biomarker for Bipolar Disorder, individuals with BD having lower levels of BDNF than controls. Peripheral BDNF was positively correlated with lifetime depression episodes, psychiatric hospitalizations, and suicide attempts. The BDNF gene provides instructions for making a protein, found in the brain and spinal cord, which promotes the survival of nerve cells (neurons) by playing a role in their growth, maturation (differentiation), and plasticity. The BDNF protein is found in regions of the brain that control eating, drinking, and body weight; it likely contributes to the management of these functions. The negative correlation between BDNF and number of mood episodes was moderated by impaired glucose metabolism.9

Nierenberg et al reviewed the evidence for mitochondrial dysregulation, which could implicate mitochondrial dysfunction as an important component of the pathophysiology of Bipolar Disorder. They identify several tolerable and readily available mitochondrial modulators (MM) as potential treatments, including N-acetyl-cysteine (NAC), acetyl-L-carnitine (ALCAR), S-adenosylmethionine (SAME), coenzyme Q(10) (CoQ10), alpha-lipoic acid (ALA), creatine monohydrate (CM), and melatonin. They conclude that clinical trials of individual MMs as well as combinations are warranted.10

Biological processes that may be dysregulated in BD are monoamine activity, immune and inflammatory processes, and oxidative stress.

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Diet and Eating Habits
Kilbourne reports that individuals with BD reported eating fewer than two meals a day, had difficulty obtaining or cooking food, and were not likely to discuss their eating habits with health care providers. A study of adolescents found a higher prevalence of binge eating and emotional eating among participants with BD than in control participants. Nutritional behaviors were related to stress eating and impulsivity, which authors said demonstrated a need to screen for nutrition factors and intervene in this group.

Bly et al found that both subjects with bipolar disorder and those with schizophrenia consumed fewer total calories, carbohydrates and fats, as well as more fiber (p < 0.03), when compared to NHANES controls.

A study in Canada compared the food records of 97 adults diagnosed with mood disorders with the “Eating Well with Canada’s Food Guide” and the “North American Dietary Reference Intakes,” and found participants consumed less than recommended servings of grain foods and vegetables and fruits and less than the Adequate Macronutrient Distribution Range (AMDR) for α-linolenic acid. The group consumed greater than recommended amounts of high-fat whole-grain foods, processed meats and higher sugar, fat, or salty foods. Thirty-nine percent of meals and snacks were from sources outside the home. Forty-four percent had cholesterol levels >6.2 mm/L.

Comparing seven-day food records and fasting plasma levels, Evans et al found that individuals with BD had significantly lower intake of Se and PUFAs: EPA, DHA, AA, DPA (docosapentaenoic-a mix of n-3/n-6) and significantly increased intake of saturated fats. The authors suggest these differences in PUFA metabolism between individuals with BD and controls may impact health outcomes for individuals with BD, and that correcting these disparities may be helpful.

In a later publication Evans et al found significant associations between plasma LA, and worse outcomes on scores on Patient Health Questionnaire (PHQ) and the Life Functioning Questionnaire (LFO) and the Short Form Health Survey (SF12).

In contrast, Pomponi, et al., using fasting plasma levels, gas chromatography/mass spectrometry, found that DHA was significantly decreased compared to controls but that patients with BD had significantly higher levels of AA, ALA and EPA. They suggested that DHA may be a possible intervention. They noted that plasma levels were more reliable and reproducible than assays of erythrocyte FA content. However, Hallahan and Hibbeln found in an extensive meta-analysis that EPA has a greater anti-inflammatory effect in the brain, which may contribute to the anti-depressant effect.

It is possible for caffeine to induce denovo psychotic and manic symptoms as well as aggravate previous disorders, as discussed by Wang, Woo and Bahk.

Supplements
Use of Supplements
In a U.S. study, 348 patients with Bipolar Disorder self-reported that, in addition to prescribed psychiatric drugs, 29% (N=101) used a dietary supplement for at least 7 days and 20% used a supplement long term (at least 50% of days). Of those who used supplements, 71.3% took one supplement daily. These 101 patients tried over 40 different supplements and the long-term users took 19 different supplements. The most commonly taken supplements for both groups were fish oil, B vitamins, melatonin, and multivitamins.

The use of a dietary supplement was defined as taking one supplement for at least 7 consecutive or non-consecutive days during the study. Long-term use of a dietary supplement was defined as taking one supplement for at least 50% of the study days. Effects of Supplemental Nutraceuticals
Sarris, J, D. Mischoulon, and I. Schweitzer reviewed findings of positive evidence with large effect sizes (see box below) with use of vitamin and mineral supplements for individuals with mania. For bipolar mania, several nutraceuticals reduced mania with strong clinical effects: a chelated mineral formula (d=0.83), L-tryptophan (d=1.47), magnesium (d=1.44), folic acid (d=0.40), and branched-chain amino acids (d=1.60). Mixed, but mainly positive, evidence was found for supplementing omega-3 fatty acids for bipolar depression, while no evidence was found for use in mania. No significant effect on BD outcome scales was found for inositol (possibly due to small samples).

A meta-analysis for the effects of nutraceuticals on depression showed primarily positive results for replicated studies testing S-adenosylmethionine (SAMe), methylfolate, omega-3 (primarily EPA or ethyl-EPA), and vitamin D. Positive isolated studies were found for the effect of creatine, folinic acid, and an amino acid combination. Mixed results were found for zinc, folic acid, vitamin C, and tryptophan, with nonsignificant results for inositol. Minor digestive disturbance was the only adverse effect noted. A meta-analysis of adjunctive omega-3 versus placebo revealed a significant and moderate to strong positive effect of omega-3. Conversely, a meta-analysis of folic acid revealed a nonsignificant difference from placebo. BD treatment outcomes may potentially be improved by adding use of certain nutraceuticals along with conventional pharmacotherapies. However, the authors commented that caution should be extended in interpreting the large effects of several isolated studies, as they have not yet been replicated in larger trials.

Drug – Nutrient Interaction
Lithium
Lithium salts have been in use for the treatment of bipolar disorder for more than 50 years, but their pharmacological mode of action is not clearly established. Since Li(+) and Mg(2+) share many properties, many reported cellular targets for Li action involve Mg-activated enzymes, which
Lithium salts / Lithium carbonate: Eskalith (capsule), Lithobid (tablet)

Lithium and goiter
Lithium inhibits the release of thyroid hormone (TH) and lithium treatment has been associated with the development of goiter. Goiter in patients receiving lithium therapy ranges from 20% of patients in iodine-replete areas, to 87% of patients residing in iodine-deficient areas, or who are on long-term lithium therapy. Goiter has been observed within several weeks of initiation of lithium therapy, although in most cases it takes months to several years before goiter develops.

Medical Nutrition Therapy for Patients with Bipolar Disorder
• Nutrition Assessment of patients diagnosed with BD should pay special attention to: O-3 fatty acids, folate intake, Mg, Na/fluid balance, weight changes, changes in glucose and lipid labs, alcohol and caffeine intake.
• Low to moderate intake of caffeine (≤ 200 mg at one time) is preferred. Caffeine may lower lithium level and/or bolster manic behavior.
• Consistent sodium and fluid intake (2-3 L/day) is important for preventing Li toxicity.
• Review supplement use with a recommendation for moderate use of supplements that complement diet insufficiencies. Discontinue iodine supplements.
• Include observation and palpation for goiter when conducting a nutrition-focused physical examination.
• Taking Lithium with meals decreases possible GI distress.
• Both manic or depressive phases often influence regularity of meals and snacks as well as content. A general eating schedule that includes foods that are readily available, storable and easily prepared supports a nutritionally adequate intake.
• Monitor laboratory reports that reflect any developing metabolic syndrome (FBS, HgA1c, lipids)
• Schedule follow-up appointments to review changes in weight, food and supplement intake and changes in eating habits.

Research on Bipolar Disorder is increasing rapidly. Well-designed studies of metabolism, genetics, and effects of nutrients and nutritional status will likely continue to impact nutrition care. Clinical Dietitians in Behavioral Health will need to keep abreast of ongoing developments.

About the Author
Dr. Leyse-Wallace retired from clinical practice and has published three books: Linking Nutrition To Mental Health and Nutrition and Mental Health as well as a reader-friendly version of her doctoral dissertation The Metaparadigm of Clinical Dietetics: Derivation and Applications. She lives in Alpine, California in San Diego County and has three adult children and five grandchildren.

References
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Students, are you looking for a place to share your writing?

BHN students and interns are invited to submit posts or topics for consideration. All posts should stick to BHN's all-foods-fit philosophy, but are encouraged to highlight your own unique voice and focus! Please include short “about me” when submitting, including your education and interests. We welcome experienced bloggers and new bloggers alike! Click here to visit the student blog, and contact Amanda Adkison to get involved.

In need of a good resource?
Try the Quality Resource Collection.

With over a 100 different resources listed, the Quality Resource Collection serves to develop quality management knowledge and skills as a critical component of nutrition and dietetics practice. This collection published by the Academy’s Quality Management Committee, includes resources used in practice by the Quality Leader Alliance and reflects their areas of expertise.

Access the Quality Resource Collection here: [www.eatrightpro.org/qualitystrategies](http://www.eatrightpro.org/qualitystrategies)
In the BHN Pipeline!

**Member forum online**
We encourage members to check out the online forum as another means besides our EML to connect with BHN members, ask questions and get responses. Read the instructions to post and get started!

**Mentoring program in the works**
We are continuing to work on developing our own program. We are looking for a member to coordinate this service with student member assistance. Please contact the Membership Chair at membership@bhndpg.org if you have an interest in being a part of this program in any way.

**Member Marketplace online**
Members can now post books and guides that they developed for sale or other services such as training and web-based services.

**Calendar of Events**
We now have a web – based calendar of activities related to our areas of practice that can include local, state, national and international events sponsored by service and professional organizations, colleges and institutions, government and our members. Members submit an event for posting online to Jamie Dannenberg at jamiedannenberg@gmail.com, our student volunteer working with our Web Coordinator to provide this service.

**Case Studies**
Case Studies have been finalized and quizzes available to get CEUs online.

**BHN Speaker’s Bureau**
Go to the BHN website “Store” and the Speakers Bureau drop down to complete a survey if you are interested in participating.

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**Nominations Committee is pleased to announce BHN Election Results!**

The following officers will assume duty on June 1, 2017:
- Chair Elect: Megan Kniskern MS, RD, LD/N, CEDRD
- Treasurer: Jenni Costello, RD, LD, LCSW
- Nominating Committee: Christina Drobisch, RDN

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**Are you interested in getting more involved with Behavioral Health Nutrition?**

This is a great opportunity to expand your professional network and contribute to the growth and impact of BHN. We are looking for BHN members for the positions of:
- Public Relations Chair
- Event Co-chair

If you would like position descriptions, would like to express your interest, or if you have any questions, please contact BHN nominating chair, Rachael McLellan at rachaelpress@gmail.com

We look forward to hearing from you!
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chair@bhndpg.org

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Kathryn Russell, MS, RDN  
treasurer@bhndpg.org

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hodrepresentative@bhndpg.org

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Lester Rosenzweig, MS, RDN, CDN  
membershipchair@bhndpg.org

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addictionsresourceprofessional@bhndpg.org

Marci Anderson Evans, CEDRD, CPT, LDN  
eatingdisordersresourceprofessional@bhndpg.org

Sharon Lemons, MS, RDN, CSP, FAND  
telehealthresourceprofessional@bhndpg.org

Mental Health Resource Professional (2014-2016)  
Ruth Leyse-Wallace, PhD, RDN  
mentalhealthresourceprofessional@bhndpg.org

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Student Liaison Committee Chair  
Emily Conner  
studentliaisoncommitteechair@bhndpg.org

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*Nominating Committee Chair  
Rachael Press, RD, LD  
nominatingcommitteechair@bhndpg.org

Nominating Committee Member (2016-2018)  
Theresa Wright, MS, RDN, LDN  
nominatingcommitteemember1@bhndpg.org

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Megan Kniskern, MS, RDN, CEDRD  
publicrelationsdirector@bhndpg.org

Sponsorship Chair  
Dana Magee, RD, LD, CLT  
sponsorshipchair@bhndpg.org

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Eugenia Goh, MS, RDN, LD  
webinarcoordinator@bhndpg.org

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Kacy Grossman, MS, RDN, CPT  
socialmediacoordinator@bhndpg.org

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Jacqueline Larson, MS, RDN  
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Carol Bradley, PhD, RDN, LD, BCBA  
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publicationchair@bhndpg.org

Newsletter Editor (2016-2017)  
Hanna Kelley, RD, CD  
newsletteeditor2@bhndpg.org

Assistant Newsletter Editor  
Becky Hudak, RDN  
assistantnewslettereditor1@bhndpg.org

Student Newsletter Editors  
Valerie Della Longa  
studentassistantnewslettereditor1@bhndpg.org  
Shana Spence  
studentassistantnewslettereditor2@bhndpg.org

CPE Test Writer  
Kathryn Mount, MS, RDN, LDN  
cpetestwriter@bhndpg.org

Newsletter CPE Manager  
Caitlin Royster, RDN, LDN  
newslettercpecmanager@bhndpg.org

DPG/MIG RELATIONS
Manager, DPG/MIG Relations  
Katie Gustafson  
The Academy of Nutrition and Dietetics  
kgustafson@eatright.org

*Voting Member

Contribute an article or topic for future BHN Newsletter issues!  
Contact newslettereditor2@bhndpg.org or one of the BHN leaders listed in this newsletter.

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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