Night Eating Syndrome: An Overview
By Roberta Pearle Lamb, MPH, RD

Of the multiple methods of obtaining diet history information, the 24-hour diet recall is perhaps the one most often used by dietitians in a clinical setting. During my dietetics training 28 years ago, I was not trained to interview patients specifically about waking and eating in the middle of the night, yet over the past several years I have come to appreciate how many individuals struggle with what has become identified as Night Eating Syndrome (NES). Though not formally defined as an eating disorder in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), it is widely considered by eating disorders specialists as an emerging eating disorder that requires specific treatments, many of which vary considerably from treatments for other eating disorders (1, 2).

First identified by Albert Stunkard, MD in the 1950’s (3), NES is finally getting the attention it deserves. That’s a good thing, considering it an estimated 1.5% of the general population and 9 to 14 % of people seen in clinics for the treatment of obesity, according to Gluck, Geleibter and Satov (4). Estimates from studies of bariatric patients seeking surgery for obesity indicate that between 9 and 42% have NES (5).

What is NES? NES is characterized by a lack of appetite in the morning, overeating at night and waking to eat in the middle of the night. Many people snack at night, but those with NES eat a majority of their food during the evening. They experience little or no hunger in the morning, but tend to overeat at dinner and after dinner. For people with NES, falling and staying asleep may be challenging, so a bedtime snack may be used to help. People with NES usually wake up and eat at least once in the night.

Some individuals with NES may have obesity. But a dietitian may discover night eating in an individual who presents with a body weight that is within the ideal range; such an individual may have only been engaged in night eating for a shorter period of time, perhaps secondary to the onset of a psychiatric condition.

Many individuals with NES are susceptible to depression, substance abuse or mood disorders. Some describe a worsening of mood as the day progresses, not unlike the so-called “sundowning effect” sometimes noted in individuals with dementia. Supporting the conclusion that NES is associated with an elevated risk of Axis I disorders, such as mood disorders, anxiety and substance abuse, one study found that 12% of individuals visiting outpatient psychiatric facilities had NES (6).

When night eaters become sleep deprived, they become less able to function well during the day, perhaps relying on the nighttime eating patterns as a form of self medication to help with stress or sleep. They describe feeling out

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FROM THE CHAIR
Jessica Setnick, MS, RD, CSSD

How thrilling to start my term as Chair of Behavioral Health Nutrition (BHN) dietetic practice group (DPG)! Obtaining respect and reimbursement for our roles in Addictions, Eating Disorders, Intellectual & Developmental Disorders, and Mental Health has been a dream of mine since I joined the Executive Committee (EC) 5 years ago, and I feel poised and ready to seize the day. I am thrilled that some of the ideas that were just conversation at the American Dietetic Association (ADA) Food & Nutrition Conference & Expo (FNCE) in San Antonio, TX have become reality, and that BHN is on the verge of a new presence within and outside of ADA.

In the coming year, I hope to build on the achievements of the past and bring even more of those ideas to fruition, ideas that just a few years ago seemed out of reach. With the help of an enthusiastic EC, a plan for the coming year is in place that will help you, our members, receive even more value from your BHN membership than ever before. Because BHN would not exist without members, we have put you first, so that you will always feel your investment is worthwhile. We know your DPG dollar must stretch, and so we have made a commitment to earn it.

One example is our second pre-FNCE workshop, Saturday, October 25th in Chicago, IL. The workshop is a continuation of the standing-room-only session in Philadelphia on Psychotropic Drug-Nutrient Interactions given by Dean Elbe and BHN member extraordinaire Zaneta Pronsky. We will pay you back your DPG membership fee when you attend this event, in the form of a $25 discount off the regular fee. And if you bring a colleague who is not a BHN member, we will make them a BHN member just for coming to the workshop! Look for the link to register for this workshop on the BHN website www.bhndpg.org.

Another commitment we have made is to increase the recognition, respect, and demand for BHN members in the workplace. One exciting project in this vein is starting the process for a specialty certification exam in each of our four practice areas. A committee is forming with other DPGs to create Standards of Practice for Eating Disorders, the first step in the process. If you have input for the committee (and I know you do!) be sure to attend the FNCE open discussion meeting at McCormick Place on Monday October 27. Being able to say you are a Certified (fill in your practice area here) Dietitian will certainly be worth the price of your membership fee!

The work BHN members do is amazing and detailed. The way to showing others how much we contribute is through research. BHN is collaborating with the Research DPG to create a guide for members to make it easy for us to document the amazing results we are already continued on page 5
Night Eating Syndrome
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of control, and usually blame themselves for a lack of willpower. Some researchers are studying the night eating patterns associated with stress. Research suggests that teaching individuals who identify specific stressors how to use techniques for reducing stress, they will successfully lessen their reliance on night eating behaviors (7).

Defining NES
Since Albert Stunkard, M.D. first identified NES in 1955 (3), the definition for NES has been modified repeatedly, perhaps a sign of the psychiatric community’s uncertainty as to at what point night eating advances from being a bad habit to a clinical illness.

Dr. Stunkard first described NES as the presence of anorexia in the morning, hyperphagia in the evening and insomnia at night. He described NES patients as eating 25% of their daily food intake after 6 p.m. and determined that the disorder is linked to stress.

More recently, NES has been described as a delay in a circadian pattern of eating, manifested by: 1) evening hyperphagia (i.e., more than 25% of calories after the evening meal), 2) awakenings accompanied by nocturnal ingestions at least three times a week, while in full consciousness or 3) both. (8).

Nocturnal Sleep-Related Disorder (NS-RED)
NES differs from sleep-related disorders, such as nocturnal sleep-related eating disorder (NS-RED). Eating at night is a hallmark of both disorders; the distinguishing characteristic between the two disorders is how conscious individuals are when they eat at night. People with NS-RED are generally not fully conscious and may even be sleepwalking during their nighttime eating episodes. Further, those with NS-RED typically have no recollection of having eaten the night before. NS-RED is associated with restless leg syndrome, obstructive sleep apnea and other sleep disorders, such as somnambulism. For those with NS-RED, health professionals should recommend an evaluation by a sleep specialist, especially since sleep duration is increasingly recognized as an important factor in determining obesity risk (9).

Eating Patterns
A skilled dietitian in a clinical setting can identify an individual struggling with night-eating patterns relatively quickly. Night eaters usually report that, not only do they skip breakfast and possibly lunch, they describe a lack of appetite during much of the day. Their diet histories indicate that they eat often eat a large dinner, one that might begin with a meal size snack. Most striking is that they continue eating after dinner, and they might even delay going to sleep to accommodate another meal or snack before bedtime.

Many people snack at night, but those with NES eat a majority of their food during the evening.

Take the case of a 40 year old female who works full time and takes care of her 12 year old son. Her husband struggles with binge eating, so after dinner snacking has become part of the family behavior patterns. She often finds herself rushing through her work days, skipping meals, and eating most of her calories in the evening. She reported a history of hiding food dating back to her childhood. She had resorted to hiding food after the evening. She reported a history of binge eating disorder (10).

Night eating-related depression is often distinguishable from other forms of depression because the depressed mood begins later in the day, as the evening approaches. Many people with NES report that their mood is stable when they wake up, but that they experience a type of sundowning effect towards the end of the day (8). Night eaters may unknowingly be attempting to treat underlying depression with food. Often they grew up in homes where night eating was common, so they might not recognize their eating style as unusual. One person recently seen at our treatment center reported that her own mother had eaten in the middle of the night throughout her childhood. She had never before identified her eating pattern as unusual. A recent study suggests a potential link between childhood emotional abuse or neglect and NES and binge eating disorder (10).

NES and Anxiety
Often night eaters awaken during the night with anxiety and they find that the only way they can get back to sleep is by eating. Typically they report that the night eating occurs without experiencing hunger, but is more often a response to anxiety.

Biological Influence
Individual stories of past traumas and other triggers to the development of night eating patterns might lead the treatment team to focus on environmental causes of NES. Yet a strong case for biologically induced night eating is slowly emerging (11). Ghrelin, a gut hormone that stimulates appetite, has been explored in NES, and a recent study suggests that people with NES may have lower levels of ghrelin (12).

Treatment of NES
The dietitian plays a critical role in the successful assessment, implementation and monitoring of the patient with NES. Simply encouraging night eaters to rearrange their caloric intake to daytime hours are not adequate. Successful behavior change requires several steps and can only be accomplished when the individual is ready for treatment. Much of the research on treatment modalities comes from the University of Pennsylvania’s Weight and Eating Disorders Program. Kelly Allison, PhD, Albert Stunkard, MD and Sara Their wrote an excellent resource for professionals and patients who are interested in learning about NES (2).

To help NES patients become aware of their eating patterns, dietitians may suggest keeping a food diary. Some patients may enter treatment very aware of what they are eating, but not aware of what their triggers, feelings or emotional struggles might be. In these cases, keeping track of food eaten may take a back seat to keeping track of daily activities, mood, difficulty of falling asleep, waking
patterns, or feelings. Once patients become aware of their patterns, dietitians can assist patients in improving nutritional intake during the day, decreasing intake during the night, while discouraging compensatory measures such as daytime restriction or excessive exercise.

In the program we developed to treat NES at our facility, we have combined several aspects of CBT including behavioral chain analyses, stress management, exposure through the introduction of group meals, and aerobic exercise guided by an exercise physiologist, personalized for each participant's individual needs. The program provides individual and group programming for behavioral and nutritional skill development. Pharmacotherapies have shown success with NES including sertaline and other antidepressants of the selective serotonin reuptake inhibitors (SSRI) class (14). Another medication frequently used to treat NES is topiramate (topamax) (15).

Gastric bypass surgery can be a viable treatment option for a small percentage of night eaters. For many individuals struggling with night eating, behavioral treatment, with the introduction of structured eating, may be indicated.

Emerging Awareness of NES

A well-informed practitioner can accurately assess biological and behavioral influences, which may help inform and motivate people struggling with NES. Dietitians working in behavioral care can play a central role in treatment, particularly when diet is combined with motivational interviewing skills and an in-depth understanding of the role of nutrition in treating individuals with obesity who present with multiple previous dieting attempts. Individualized treatment that incorporates nutrition strategies, CBT and other behavioral approaches, combined with pharmacotherapy, can be effective in treating this emerging eating disorder that affects as many as 6 million Americans.

References:
10. Allison KC, Grillo CM, Masheb RM, Stunkard AJ. High self-reported rates of neglect and emotional abuse, by persons with binge eating disorder and night eating syndrome. Behav Res Ther 2007; 45(12):2874-83

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LINKING NUTRITION TO MENTAL HEALTH:
A Scientific Exploration by
RUTH LEYESE-WALLACE PHD, RD
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To truly live well Americans must start addressing mental health with the same urgency as physical health.

Ruth Leyse-Wallace, PhD, RD reviews scientific research from around the world and many disciplines to demonstrate how diet, vitamins and minerals, genetics, medications, and health conditions influenced by nutrition can affect an individual’s mental well-being as much as physical well-being.

Nutrients linked by science to conditions such as schizophrenia, depression, eating disorders, alcohol abuse, dieting, and bariatric surgery are discussed.

Use this highly referenced, indexed publication, rich in professional resources, to augment your nutrition practice. Dr. Leyse-Wallace has practiced clinical dietetics for 30 years. She earned her PhD at the University of Arizona in 1998.

Health U.: Weight Loss and Down Syndrome

By Renee Scampini, M.S., R.D.

Overweight and obesity are common among children with Down syndrome (DS). Recent studies suggest that the prevalence of overweight and obesity in this group is higher than the general population (Bell, 1992; Rubin, 1998) and than that of other individuals with intellectual disabilities (Bhaumik, 2008; Melville, 2005). Despite the pressing need for preventive and treatment programs, little research has been done to determine how to help persons with DS lose weight or to learn about healthy lifestyle choices.

Dr. Richard Fleming and an interdisciplinary research team at the University of Massachusetts Medical School/Eunice Kennedy Shriver Center have developed an educational program called Health U. The Health U. curriculum consists of 16 sessions that focus on nutrition and physical activity, with materials and activities modified to meet the cognitive needs of the participants. Health U. is currently being conducted as a randomized controlled trial to determine the best approach for promoting weight loss in adolescents and young adults (ages 13-26) with DS.

Participants are assigned to one of two conditions: a nutrition and physical activity education alone (NAE) group; or a behaviorally-based parent-supported weight reduction (PSWR) group. In both groups, participants and their parents attend the 16 Health U. nutrition and physical activity classes led by a dietitian and an adaptive physical education specialist. In addition, parents in the PSWR group also receive a detailed behavior modification curriculum, taught weekly by a behavioral psychologist. The PSWR group will be compared to the NAE group to determine if the addition of parent-directed behavior modification increases weight loss and/or healthy lifestyle choices.

Through Health U., participants and their parents learn how to initiate and maintain a healthy lifestyle by making healthful nutrition choices and increasing daily physical activity. The lessons are designed to be interactive, social, fun, and engaging with an emphasis on hands-on, interactive activities. Nutrition topics include the importance of fruits, vegetables and low-fat dairy products. Portion sizes, healthy choices, eating at restaurants and mindful snacking are included. Simple, healthy recipes with few steps and ingredients are also provided. All lessons include “taste-tests” for making and sampling healthy foods and drinks such as: novel vegetables and fruits; healthy ‘cracker stackers;’ and smoothies. The goal of the “taste tests” is to encourage participants to try new foods and to potentially expand their food repertoires. Thus far participants have been willing to try new foods, even though some have had trepidation in doing so. As a result, participants have found new foods to enjoy. Participants also attempt and practice modified physical activities, focusing on stretching, strengthening and cardiovascular exercises. Formal exercise plans, yoga mats, gym bags, and exercise balls and bands are some of the incentives that participants take home to increase their opportunities to exercise.

At the onset of the program, an individualized healthy eating plan is developed for each participant based on body size, age and activity level. The eating plans are designed to promote gradual weight loss (approximately 1/2 lb per week). The plans are based on a dairy, fruit, vegetable, grain, and protein needs assessment from the mypyramid.gov recommendations combined with portion sizes similar to the carbohydrate exchange list. In this way, a calorie-based healthy eating plan is developed for each individual. Participants are given a specific number of servings of fruits, vegetables, grain, protein, and dairy represented in picture form on their healthy eating plans.

During multiple nutrition lessons, participants are taught about portion sizes and healthy food choices within each food group. While calorie-based, the nutrition lessons are...
Thin Bones Seen In Boys with Autism and Autism Spectrum Disorder


Results of an early study suggest that dairy-free diets and unconventional food preferences could put boys with autism and autism spectrum disorder (ASD) at higher than normal risk for thinner, less dense bones when compared to a group of boys the same age who do not have autism. The study, by researchers from the National Institutes of Health and Cincinnati Children’s Hospital Medical Center, was published online in the Journal of Autism and Developmental Disorders. The researchers believe that boys with autism and ASD are at risk for poor bone development for a number of reasons. These factors are lack of exercise, a reluctance to eat a varied diet, lack of vitamin D, digestive problems, and diets that exclude casein, a protein found in milk and milk products. Dairy products provide a significant source of calcium and vitamin D. Casein-free diets are a controversial treatment thought by some to lessen the symptoms of autism.

Funding for the study was provided by the NIH’s National Institute of Child Health and Human Development and National Center for Research Resources. The research team that conducted the study was led by Mary L. Hediger, Ph.D., a biological anthropologist in NICHD’s Division of Epidemiology, Statistics and Prevention Research.

"Our results suggest that children with autism and autism spectrum disorder may be at risk for calcium and vitamin D deficiencies," Dr. Hediger said. "Parents of these children may wish to include a dietitian in their children’s health care team, to ensure that they receive a balanced diet." Dr. Hediger stressed that the current study results need to be confirmed by larger studies. Until definitive information is available, however, it would be prudent for parents of children with autism and ASD to include a dietitian in their care, particularly if the children’s diets do not include dairy products or they are not otherwise eating a balanced diet, she said. Because girls are much less likely to have autism or ASD than are boys, the researchers were unable to enroll a sufficient number of girls within the short time frame of the study to allow them to draw firm conclusions. Dr. Hediger added that if a girl with autism or ASD is not eating dairy products or eating a balanced diet, it would be prudent for a dietitian to be included in her health care team.

Autism is a complex brain disorder involving communication and social difficulties as well as repetitive behavior or narrow interests. Autism is often grouped with similar disorders, which are often referred to collectively as autism spectrum disorders. The underlying causes of autism and ASD are unclear. There is no cure for the disorders and treatments are limited.

When the boys were enrolled in the study, the researchers asked the boys’ parents if the boys were taking over-the-counter or prescription medications, were taking any vitamin or mineral supplements, or were on a restricted diet. During the study, researchers X-rayed the hands of 75 boys between the ages of 4 and 8 years old who had been diagnosed with autism or ASD. The researchers then measured the thickness of the bone located between the knuckle of the index finger and the wrist and compared its development to a standardized reference based on a group of boys without autism.

Dr. Hediger said that the research team measured cortical bone thickness. She added that this procedure was done as a substitute for a conventional bone scan, which measures bone density. Bone density is an indication of bones’ mineral content. Less dense bones may indicate a risk of bone fracture. The researchers used the measure of bone thickness because many of the boys were unable to remain still long enough for the conventional scan, which requires individuals to lie immobile for an extended period of time. To successfully complete the bone scan, many of the boys would have required sedation — a step the researchers were reluctant to take for an early study. The hand X-ray, Dr. Hediger explained, offers an approximate indication of bone density. She added, however, that because the researchers were unable to use a conventional bone scan, the results of the current study should be confirmed by additional studies using conventional bone scans.

The investigators found that the bones of the boys with autism were growing longer but were not thickening at a normal rate. During normal bone development, material from inside the bone is transferred to the outside of the bone, increasing thickness, while at the same time, the bones are also growing longer. At 5 or 6 years of age, the bones of the autistic boys were significantly thinner than the bones of boys without autism and the difference in bone thickness became even greater at ages 7 and 8. The bone thinning was particularly notable because the boys with autism and ASD were heavier than average and would therefore be expected to have thicker bones.

The researchers do not know for certain why the boys had thinner than normal bones. A possible explanation is lack of calcium and vitamin D in their diets. Dr. Hediger explained that a deficiency of these important nutrients in the boys’ diets could result from a variety of causes. Many children with autism, she said, have aversions to certain foods. Some will insist on eating the same foods nearly every day, to the exclusion of other foods. So while they may consume enough calories to meet their needs — or even more calories than they need — they may lack certain nutrients, like calcium and vitamin D. Other children with autism may have digestive problems which interfere with the absorption of nutrients. Moreover, many children with autism remain indoors because they require supervision during outdoor activity. Lack of exercise hinders proper bone development, she said. Similarly, if children remain indoors and are not exposed to sunlight, they may not make enough vitamin D, which is needed to process calcium into bones.

The boys in the study who were on a casein-free diet had the thinnest bones. In fact, the 9 boys who were on a casein-free diet had bones that were 20 percent thinner than normal for children their age. Boys who were not on a casein-free diet showed a 10

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percent decrease in bone thickness when compared to boys with normal bone development. The study authors wrote that bone development of children on casein-free diets should be monitored very carefully. They noted that studies of casein-free diets had not proven the diets to be effective in treating the symptoms of autism or ASD. Only 9 boys on casein-free diets were available to participate in the study. Dr. Hediger said. When conducting a scientific study, it’s easier to obtain statistically valid results by studying a larger number of individuals than with a smaller number of individuals. However, the dramatic difference in the boys’ bone thickness when they were either on a casein-free diet or an unrestricted diet and when compared to normally developing bones strongly suggest that the bone thinning the researchers observed was statistically valid.

The researchers recommended that larger studies be conducted to confirm their results. Until those studies can be conducted, Dr. Hediger offered the following advice: "Our study shows that it couldn’t hurt — and would probably help — if parents of children with autism or autism spectrum disorder consulted with a dietitian during their children’s routine medical care to make sure that their diets are balanced."

General information about autism and ASD is available from the NICHD’s Web site, at http://www.nichd.nih.gov/publications/pubs/autism/overview/index.cfm. The NICHD sponsors research on development, before and after birth; maternal, child, and family health; reproductive biology and population issues; and medical rehabilitation. For more information, visit the Institute’s Web site at http://www.nichd.nih.gov/.

The National Institutes of Health (NIH) — The Nation’s Medical Research Agency — includes 27 Institutes and Centers and is a component of the U.S. Department of Health and Human Services. It is the primary federal agency for conducting and supporting basic, clinical and translational medical research, and it investigates the causes, treatments, and cures for both common and rare diseases. For more information about NIH and its programs, visit http://www.nih.gov/.

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Alcohol-Related Birth Defects, The Past, Present and Future

By Kenneth R. Warren, Ph.D., and Laurie L. Foudin, Ph.D.

The term fetal alcohol syndrome was introduced in 1973 by Jones and Smith (1973), whose original diagnostic criteria have changed very little even after being reconsidered by other groups, such as the Fetal Alcohol Study Group of the Research Society on Alcoholism (Rosett 1980; Sokol and Clarren 1989). However, after the FAS diagnostic criteria were introduced, it became clear that there were people who likely had been adversely affected by prenatal alcohol exposure but who did not completely fulfill the criteria for a diagnosis of FAS. One term that had been introduced to include such cases was fetal alcohol effects (FAE) (Clarren and Smith 1978). But, unlike the term FAS, not all clinicians and researchers used the term FAE uniformly. Consequently, the IOM addressed this confusion by introducing more refined definitions, which have helped to provide consistency in the terminology used to describe the problems caused by prenatal alcohol exposure. For this reason, it is worthwhile to review the diagnostic criteria in the IOM report in some detail.

The IOM developed five diagnostic categories (see textbox). The first two pertain to FAS itself. The other categories address various aspects of the spectrum of alcohol-related disorders. Researchers previously had disagreed whether an FAS diagnosis could be made without evidence of maternal alcohol use. Some investigators had argued that the phenotype (i.e., the visible characteristics) of FAS appeared to be sufficiently unique to permit the diagnosis to be made even in the absence of information on maternal drinking; other investigators, however, felt uncomfortable about making an FAS diagnosis without a confirmation of maternal drinking. The issue of drinking history as one of the diagnostic criteria is important, because maternal drinking history is frequently unknown. Because no validated objective biological marker currently exists to confirm maternal drinking during pregnancy or alcohol exposure of the fetus (although researchers are trying to find one), researchers must rely on maternal self-reports as well as reports from the mother’s collateral acquaintances. However, many affected children are in foster care or adopted, and accurate drinking information for the birth mother, even from her collaterals, is not readily available for these children. The IOM addressed this problem by creating two FAS categories that differed from each other only on whether maternal alcohol exposure could be confirmed. At-risk maternal drinking during pregnancy was defined as a pattern of excessive intake characterized by substantial, regular intake or heavy episodic drinking (Stratton et al. 1996 a, p. 77). Several indicators of this type of risk drinking were listed, including evidence of withdrawal episodes and of social or legal problems attributable to drinking. These are also indicators of dependence. If all other diagnostic requirements are present for FAS except confirmation of maternal alcohol exposure, cases could be assigned to category 2. However, if confirmation existed that risk drinking did not occur, a diagnosis of FAS would not be made even if the affected person appeared to have all the signs of FAS.

The other elements of the FAS diagnosis in the IOM definitions do not with those observed in FAS. The latter deviate significantly from the original indicator and includes a complex pattern of descriptions provided by Jones and Smith (1973) and Clarren and Smith (1978). These elements include evidence of growth functions involved in guiding behavior retardation (e.g., low birth weight, lack of weight gain over time, or a low weight-to-height ratio); evidence of neurodevelopmental (e.g., a small sized brain [i.e., microcephaly] or other structural brain abnormalities); and a characteristic pattern of mild facial

IOM-Recommended Diagnostic Criteria for FAS and Alcohol-Related Effects

Category 1. FAS With Confirmed Maternal Alcohol Exposure
1. Confirmed maternal alcohol exposure *
2. Characteristic pattern of facial anomalies, including short palpebral fissures, and abnormalities of the premaxillary zone (e.g., flat upper lip, flattened philtrum, flat midface)
3. Growth retardation, such as low birth weight, lack of weight gain over time, disproportional low weight to height
4. Neurodevelopmental abnormalities of the CNS, such as small head size at birth; structural brain abnormalities with age-appropriate neurological hard or soft signs (e.g., impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor eye-hand coordination)

Category 2. FAS Without Confirmed Maternal Alcohol Exposure

Characteristics 2-4 as in Category 1

Category 3. Partial FAS With Confirmed Maternal Alcohol Exposure
1. Confirmed maternal alcohol exposure *
2. Some components of the FAS facial pattern either 3, 4, or 5 below:
   3. Growth retardation as in Category 1
   4. CNS neurodevelopmental abnormalities as in Category 1
   5. Complex pattern of behavioral or cognitive abnormalities inconsistent with developmental level and unexplained by genetic background or environmental conditions (e.g., learning difficulties; deficits in school performance; poor impulse control; problems in social perception; language deficits; poor capacity for abstraction; specific deficits in mathematical skills; and problems in memory, attention, or judgment)

Category 4. Alcohol-Related Birth Defects (ARBBD)
1. Confirmed maternal alcohol exposure *
2. One or more congenital defects, including malformations and dysplasias of the heart, bone, kidney, vision, or hearing systems

Category 5. Alcohol-Related Neurodevelopmental Disorder (ARND)
1. Confirmed maternal alcohol exposure *
2. CNS neurodevelopmental abnormalities as in Category 1, and/or
3. Complex pattern of behavioral or cognitive deficits as in Category 3

* Maternal alcohol exposure is defined as a pattern of excessive alcohol intake characterized by substantial, regular intake or by heavy episodic (i.e., binge) drinking. Evidence of this pattern may include signs of alcohol dependence.

CNS = central nervous system; FAS = fetal alcohol syndrome; IOM = Institute of Medicine.

anomalies, including small eye openings (i.e., short palpebral fissures), a thin upper lip, or flattened ridges between are normal, the affected individual is the base of the nose and the upper lip assigned to category 5, alcohol-related (i.e., a flattened philtrum). (See figure)

The other three IOM diagnostic categories describe conditions that do not meet the FAS criteria. All require a confirmation of substantial maternal alcohol use because the phenotypes for these diagnoses are not unique enough to be ascribed to prenatal alcohol exposure without evidence of maternal drinking.

Category 3 includes partial FAS with confirmed maternal alcohol exposure – in other words, some, but not all, of the facial characteristics required for an FAS diagnosis must be present as well as confirmed evidence of maternal alcohol exposure. In addition, at least one of the three following indicators also must be present: growth deficits normally characteristic of FAS, neurodevelopmental abnormalities, or behavioral and cognitive problems consistent with those observed in FAS. The latter indicator includes a complex pattern of deficits in learning, school performance, impulse control, and the cognitive functions involved in guiding behavior. Category 4 encompasses ARBD and was proposed for people with heart, bone, kidney, vision, or hearing defects who had been prenatally exposed to alcohol (i.e. PEA) in their research (e.g., Riley et al.1995) to describe children who have been exposed to alcohol prenatally without the specific requirement for the presence of any particular deficit. Streissguth and O’ Malley (2000) proposed the term fetal alcohol spectrum disorders (FASD) for inclusion in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). FASD describes the full range, from mild to severe, of disturbances of physical, behavioral, emotional, and/ or social functioning attributable to in-utero alcohol damage. These terms may prove useful in some circumstances, provided that conflicting definitions for FAS, ARBD, and ARND are not introduced

Determining the Prevalence and Risk Factors

One of the biggest challenges in determining the true prevalence of FAS and the associated disorders is how to recognize the syndrome, which depends in part on the age and physical features of the person being diagnosed (Larkby and Day 1997). Several distinct screening tools have been proposed to assist in making an FAS diagnosis (Astley and Clareen 1995, 1996, 2000; Burd and Martsolf 1989).

Investigators have used three different approaches in attempting to measure the prevalence of FAS: passive surveillance systems, clinic-based approaches, and active case ascertainment in a segment of the general population. Based on recent findings, May and Gossage estimate that the prevalence of FAS in the United States during the 1980s and 1990s was 0.5 to 2.0 cases per 1,000 births.

Not every woman who drinks during pregnancy will give birth to a child with FAS or even ARND. Abel (1995) estimated that 4.3 percent of heavy drinkers give birth to an FAS child. Coles (1991) reported that half of the children of heavy drinking women were not abnormal. Therefore, defining the factors that place certain women at risk of giving birth to an alcohol-affected child is a key research issue. Risk factors include maternal age (Sokol et al. 1986; Jacobson et al. 1996), socioeconomic status (Abel 1995), ethnicity (Abel and Hannigan 1995), genetic factors (Goodlett et al.1989; Streissguth and Dehaene 1993; Rasheed et al. 1997; Su et al. 2001; Warren et al. 2001), and maternal alcohol metabolism (Chernoff 1980; Warren et al. 2001), among others. Maier and West discuss the risks associated with different drinking patterns. These studies reveal that it is not so much the total amount of alcohol that is consumed, but rather, the high number of drinks consumed at one occasion, producing a high peak blood alcohol concentration, that appears to be the greater risk factor for prenatal injury from alcohol. In fact, Jacobson and colleagues (1998) have shown that drinking expressed as average drinks per occasion is more informative than average drinks per week. They found deficits in infant performance at the level of five drinks per occasion at least once per week. However, further research is needed to evaluate the relative contributions of the various risk factors for FAS. Identification of risk factors strongly associated with alcohol-related birth outcomes could help identify high-risk pregnancies for intervention.

Discovering the Mechanisms Involved

Clearly, no single mechanism is responsible for the array of alcohol-derived fetal injuries. However, some putative mechanisms are particularly significant in early pregnancy, such as excessive cell death in a special population of embryonic cells that give rise to facial structures and certain peripheral nerves (i.e., cranial neural crest) (Cartwright and Smith 1995; Kotch and Sulik 1992), whereas other mechanisms appear to be more significant later in pregnancy (e.g., loss of specific brain cell numbers in the cerebellum [West 1993] ).

Preventing Prenatal Alcohol Use

The conundrum confronting efforts to prevent FAS, ARND, and ARBD is obvious

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to everyone who has attempted to address this issue. As the IOM noted in its report, the problem may appear simple on the surface: Women who drink excessively while pregnant are at high risk for giving birth to children with birth defects. Therefore, to prevent these defects, women should stop drinking alcohol during all phases of pregnancy (Stratton et al. 1996 a, p.1). However, many women who drink continue to do so while they are pregnant. In fact, reports indicate a disturbing trend in recent years toward increased drinking during pregnancy, especially binge drinking (Ebrahim et al. 1998, 1999). Some women may be unaware of the risks involved, whereas alcohol-dependent women may be unable to abstain. Yet, even women who are aware of FAS and ARND, and who intend to abstain from alcohol during pregnancy, may nonetheless consume alcohol in early gestation before they realize that they are pregnant.

Given the various degrees of effort needed to address the problem of drinking in pregnancy among different populations and at different levels of risk, the IOM proposed a comprehensive intervention program encompassing a spectrum of approaches. Adapting a model originally described by Gordon (1983), the IOM report (Stratton et al. 1996 a) describes three levels of prevention. Universal prevention targets an entire population group and can include such components as health advisories, public service announcements, and health articles and brochures distributed through a variety of outlets. For example, NIAAA issued a health advisory in 1977 (Department of Health, Education and Welfare 1977), which was updated with the Surgeon General’s Advisory on Alcohol and Pregnancy in 1981 (Department of Health and Human Services 1981) that recommended abstinence. Another salient example of universal prevention is the Federal law (Public Law 100-690) that requires the following label warning about the dangers of drinking while pregnant on all alcoholic beverages sold in the United States: Government warning: 1) According to the Surgeon General, women should not drink alcoholic beverages during pregnancy because of the risk of birth defects. In the previous ARBD issue of Alcohol Health & Research World, Hankin (1994) described a study to measure the effect of the alcohol warning label and recently reported on the results of that research (Hankin et al. 1996 a, b).

Selective prevention efforts target specific groups whose risks are higher than the population in general. For example, these people may reside in a community with heavy per capita alcohol use.

Indicated prevention targets individuals, rather than groups, known to be at high risk because of specific risk factors; for example, a person with a known drinking problem or who has previously given birth to a child with FAS. Indicated prevention efforts may encompass a range of support activities from counseling to case management. Indicated prevention cannot be implemented until a pregnant woman who is drinking at levels that place her fetus at risk for FAS or ARND is identified. Ongoing research is helping to develop the clinical tools necessary to meet this goal.

Assisting People Born With FAS and ARND

Children born with FAS and ARND are in critical need of interventions that can reduce the effect of their cognitive and behavioral deficits. FAS and ARND have life-long consequences, with out-comes that are often more complex than those experienced by FAS patients who are mentally retarded. In a review of the literature on adolescents and adults, Streissguth and O’Malley (2000) found evidence of mental health problems, school problems, legal difficulties, and problems with alcohol and other drugs. However, they also found that people who receive appropriate supportive services fare better with respect to secondary disabilities and life functioning than those who do not receive such services. Multiple approaches are needed, including social support, special education, behavioral and cognitive therapy, and medications.

Efforts are under way to obtain a full understanding of the specific neuropsychological functions that are impaired or spared among people with FAS and ARND. The new knowledge on FAS should help clinicians and researchers in developing approaches for assisting people in overcoming behavioral and cognitive deficits and thereby improve the quality of their lives. New approaches to amelioration, such as dietary supplementation (Thomas et al. 2000) and specialized therapeutic training (Klintsova et al. 2000) are being explored in animal models.

Looking to the Future

Although great strides have been made in identifying and characterizing the physical and neurobehavioral problems of FAS and ARND, further research is needed to accomplish the following important objectives:

• Improve the clinical recognition of women’s at-risk drinking behavior before and during pregnancy
• Intervene more effectively to modify drinking behavior during pregnancy
• Develop in-utero approaches derived from basic research to prevent or minimize alcohol-induced prenatal injury
• Determine more effective ways to identify FAS and ARND across the lifespan, especially in infants and children
• Develop strategies to address the neurodevelopmental and learning problems of children with FAS and ARND, including the use of appropriate behavioral and cognitive therapies, medications, and special education programs.

Multidisciplinary approaches encompassing basic laboratory animal research, human clinical research, and epidemiology will pave the way for translating scientific knowledge into practical approaches for preventing and treating FAS, ARND, and ARBD.

References

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not calorie driven. Calories are rarely mentioned to ensure that the program remains focused on healthy, balanced and fun eating, not to make calorie-counting a focus of the Health U. experience. Believing that it is difficult and tedious for this population to "count" calories, the program's approach is based on food group and portion size to prescribe the participants' diets.

Discretionary calories are allotted daily for each participant and converted into treats and extras; both incorporated into the participant's healthy eating plans.

The study measurement protocol is comprehensive. At the start of the program, baseline weight, height, 7 day physical activity (using accelerometers), and a 3 day food diary are recorded. These measurements are repeated at sessions 10 (11 weeks) and 16 (6 months), as well as 6 months after the final session (1 year). As data emerges on weight loss and physical activity levels, feedback received from parents has been incredibly encouraging.

Attendance has been very high, even during inclement weather, and the participants report truly enjoying the lessons. Parents state that their children have learned to make many healthier choices in their daily lives. Because weight loss and maintenance is a life-long struggle, Health U. is geared toward helping participating parents and parents make independent, realistic and lasting healthful food and physical activity choices. Analysis and interpretation of the program data is expected by Summer 2009.

For more information about Health U. and other on-going projects at UMass Med School/EK Shriver Center go to:

http://www.umassmed.edu/shriver/recruit/health.aspx
http://www.umassmed.edu/shriver/service/community/familyServices/Health.aspx

Citations:

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Coming Soon!
New BHN Publication
The American Dietetic Association is the world's largest organization of food and nutrition professionals. ADA is committed to improving the nation's health and advancing the profession of dietetics through research, education, and advocacy.

BHN 2008 FNCE Schedule of Events

**Saturday, October 25**
12:00-3:00 p.m.
Hyatt Regency Hotel on Wacker Drive Columbus Room AB
Pre-FNCE Workshop

“What Dietitians Need to Know About Psychiatric Medications – Including Weight Gain, Nutrient Interactions, and Pediatric Issues”

Presenting: Zaneta Pronsky, MS, RD, LDN, FADA and Dean Elbe, BSc (Pharm), BCPP, authors of the indispensable “Food-Medication Interactions”.

After speaking to a packed house at last year’s FNCE, we have asked Zaneta and Dean to create an even more info-packed workshop to update you on the latest information about psych meds and nutrition. If you work in clinical or community nutrition, pediatrics, chemical dependency, behavioral health, and/or eating disorders treatment, you will find this workshop to be extremely beneficial. An afternoon snack is included!

**Sunday, October 26**
5:00 – 6:00 p.m.
Hyatt Regency Hotel on Wacker Drive Wright Room
Member Reception and Awards Presentation

Join us for this valuable opportunity to network and build alliances with your peers and to applaud our recognized leaders in our areas of practice.

**Monday, October 27**
8:00 – 9:30 a.m.
McCormick Place West Room 470
Priority Session

“From Addiction to Recovery: The Role of the Dietitian”

Presenting: Kevin McCauley, MD and Theresa Stahl, MS, RD

Discuss the misconceptions about addictions and the role of the dietitian. Identify the special nutrition needs of recovery. Explore techniques for group nutrition education for patients with addiction.

**Monday, October 27**
10:30 – 1:00 p.m.
McCormick Place West
Member Showcase

Stop by our display and see what your BHN Executive Committee has been up to! Bring along your friends and encourage them to join our dynamic practice group. Be the first to purchase the new IDD Resource CD and other BHN Publications.

**Monday, October 27**
1:00 – 2:00 p.m.
McCormick Place West

SOP Open Spaces Forum
Your View of Behavioral Health Care Practice Standards: Open Discussion

Does your work focus on meeting the nutritional needs of persons with eating disorders (ED), intellectual and developmental disabilities (IDD), addictions or mental illness? If so, then we need your opinions about practice standards for dietitians in Behavioral Health Care. Join us for an open discussion.