Welcome to the COPE Webinar Series for Health Professionals!

May 28th 2014 webinar:
Night Eating Syndrome: Diagnosis and Treatment Options

Time: 12 – 1 PM EST
Moderator: Rebecca Shenkman, MPH, RDN, LDN
Program Manager
MacDonald Center for Obesity Prevention & Education

Handouts of the slides are posted at: www.villanova.edu/COPE

MacDonald Center for Obesity Prevention and Education (COPE) Goals

- Enhance Education
- Participate in Research
- Partner with agencies and organizations
- Provide Continuing Education

Handouts

NIGHT EATING SYNDROME: DIAGNOSIS AND TREATMENT OPTIONS

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Objectives: Learner will be able to:
1. Recognize the diagnostic criteria for NES
2. Describe the relationship of NES to other disorders
3. Identify treatment options for NES

Credits: This webinar is approved for 1 contact award by ANCC and 1 CPEU awarded by CDR. Suggested CDR Learning Need Code: 5370, 5200, 6000, 6020; Level 2.

Disclosures

- I, Kelly Allison, Ph.D., have served as a consultant to Pfizer Pharmaceuticals. I also receive royalties from the New Harbinger Publications and Guilford Publications.

Non-normative Eating vs. Eating Disorder

- What are the roles of:
  - Environmental cues?
  - Social & cultural cues?
  - Internal cues?

- Distress present?
- Impairment in functioning?
- Adverse health effects/disability?
  - Mental
  - Physical

Tanofsky-Kraff & Yanovski, Obes Res, 2004
OBESITY
NIGHT EATING SYNDROME

Background

- NES – described 50 years ago (Stunkard et al., 1955), but definitions have varied
  - Morning anorexia
  - Evening hyperphagia
  - Insomnia
- With inclusion under “Otherwise Specified Feeding and Eating Disorders” in DSM 5, it calls for more standardized definition
- How clinically meaningful is NES?

Proposed Diagnostic Criteria for NES

A. The daily pattern of eating demonstrates a greatly increased intake in the evening and/or night time, as manifested by one or both of the following:
   1. At least 25% of daily food intake is consumed after the evening meal
   2. At least 2 eating episodes per week occur upon awakening during the night

B. Awareness and recall of evening and nocturnal eating episodes are present.

C. The clinical picture is characterized by at least three of the following features:
   1. Lack of desire to eat in the morning and/or breakfast is omitted on > 4 mornings per week
   2. Presence of a strong urge to eat between dinner and bedtime and/or during the night
   3. Sleep onset and/or sleep maintenance insomnia are present > 4 nights per week
   4. Presence of a belief that one must eat in order to get to sleep
   5. Mood is frequently depressed and/or mood worsens in the evening

D. The disorder is associated with significant distress and/or impairment in functioning.

E. The disordered pattern of eating has been maintained for a minimum of 3 months.

F. The disorder is not secondary to substance abuse or dependence, a general medical disorder, medication, or another psychiatric disorder.

Prevalence of NES

- 1.5% in general population
  (Bear et al, Int J Eat Disord, 1997)
- 9-14% in obesity clinics
- 3.8% in type 2 diabetic population
  (Allison, Crow, Stunkard et al, Obesity, 2007)
- 12% in psychiatric clinic patients
  (Lundgren, Allison, Crow et al, Amer J Psychiat, 2006)
  - Obese patients 5.2 times more likely to have NES than normal weight patients
- 8 - 42% in prospective bariatric surgery candidates
  (Allison et al, 2000; Hsu et al, 1996)
Diabetes and Night Eating

- N = 216 Type 2 diabetic patients – Patients with NE showed higher HbA1c values, higher scores on the disinhibition and the perceived hunger scale, lower scores on the quality of life scale and higher depression scores vs. patients without NE. (de Zwaan et al., Psychother Psychosom Med Psychol, 2012)
- N = 714 type 1 and type 2 diabetic patients - evening hyperphagia significantly predicted an HbA1c>7, obesity, and having two or more diabetes complications (Morse et al., Diabetes Care, 2006)
- N = 845 Type 2 diabetic participants – no relation with HbA1c or other physical measures, except BMI (Allison et al., Obesity, 2007)

Participant Reflections

- “My husband sleeps on a cot in our kitchen to help remind me to not eat when I wake up... sad situation, but it helps my mood during waking hours which makes for a better marriage overall.”
- “Now I lock the refrigerator and pantry most nights before I go to bed. This keeps me from eating and is the only thing that has worked for me.”
- “Easier to give up and just eat... feel more hopeless about control.”
- “My husband locks a gate at the top of the stairs so I can’t get to the kitchen”
- “I think I have learned to live with it. I have no choice.”

Major themes

- Cravings (often named a specific food)
- Anxious/Agitated
- Need to eat to fall (back) asleep
- Physically hungry/Compelled to eat

Cravings

3 am: “I bolted out of bed with a radar (that’s what it feels like — I remembered where there was chocolate without having to travel far). This is gross. I knew I threw out a box of chocolate truffles because I don’t like them. I rifled through the trash... found the box and ate two.”

2 chocolate truffle candies

“I feel ridiculous – I just want to stop doing this.”

Cravings, Anxious/Agitated, & Hunger

1:56 am: “Can’t sleep – feeling pretty hungry. Maybe if I eat something I can go to sleep and feel a little more relaxed.”

2 Fried oysters, 6 oz. cup of Italian ice

2:15 am: “Still feel restless and hungry.”

3:09 am: “Still feeling hungry and restless. I want a chocolate bar.”

1 Mr. Goodbar 1.75 oz.

3:30 am: “Feeling a bit more relaxed. I will try to sleep now.”
**Eat to Sleep & Cravings**

1:00 am: “It’s now 1 am. I have no snacks with me upstairs. I have the munchies. What should I do? If only I could go back to sleep without eating… but I can’t. I’ll go downstairs and get some ice cream.

1 cup strawberry ice cream

“I feel content after eating the ice cream. Now I can go back to sleep, I hope.”

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**Nighttime Eating Assessment**

1. **Physical hunger**—feeling physical signs of hunger
2. **Craving food**—desiring specific foods
3. **Compelled to eat**—having a drive to eat, to put something in your stomach, not necessarily for a specific food
4. **Anxious**—having anxiety-provoking thoughts, ruminations, racing thoughts, etc.
5. **Agitated**—having the physical feeling of not being able to sit still or remain in bed, often linked to anxiety
6. **Sad**—feeling depressed or wanting to eat to help improve depressed mood not at all extremely
7. **Bored**—looking for an activity to pass the time
8. **Tired**—feeling fatigued and just wanting to get to sleep

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**Timing of Sleep Onset and Offset (Night 2)**

The timing of sleep onset and sleep offset were not different between NES and Control subjects in the laboratory (PSG night 2; Rogers et al., 2006) or at home (diary and actigraphy), and the laboratory PSG data were consistent with the home data (O’Reardon et al., 2004).

<table>
<thead>
<tr>
<th></th>
<th>NES subjects</th>
<th>Control subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep onset time (PSG)</td>
<td>23:38 ± 1:59</td>
<td>22:52 ± 1:04</td>
</tr>
<tr>
<td>Sleep onset time (home)</td>
<td>23:57 ± 1:33</td>
<td>23:32 ± 1:06</td>
</tr>
<tr>
<td>Sleep offset time (PSG)</td>
<td>7:04 ± 0:48</td>
<td>7:06 ± 0:41</td>
</tr>
<tr>
<td>Sleep offset time (home)</td>
<td>7:35 ± 1:11</td>
<td>6:59 ± 1:12</td>
</tr>
</tbody>
</table>

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**Results from Polysomnography (Night 2)**

<table>
<thead>
<tr>
<th></th>
<th>NES subjects</th>
<th>Control subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep onset latency</td>
<td>26 ± 16</td>
<td></td>
</tr>
<tr>
<td>TST (hour:min)</td>
<td>5:54</td>
<td>7:01 * P = 0.049</td>
</tr>
<tr>
<td>Sleep efficiency</td>
<td>72%</td>
<td>85% * P = 0.03</td>
</tr>
<tr>
<td>Latency to 1st awaken (min)</td>
<td>71 ± 50</td>
<td></td>
</tr>
<tr>
<td>Awakenings (number)</td>
<td>4.5</td>
<td>3.2 * (P = 0.069)</td>
</tr>
<tr>
<td>WASO (min)</td>
<td>59 ± 39</td>
<td></td>
</tr>
<tr>
<td>REM onset latency</td>
<td>72 ± 85</td>
<td></td>
</tr>
<tr>
<td>Stage 1 (min)</td>
<td>30 ± 37</td>
<td></td>
</tr>
<tr>
<td>Stage 2 (min)</td>
<td>165 ± 235</td>
<td>* P = 0.012</td>
</tr>
<tr>
<td>Stage 3 (min)</td>
<td>29 ± 38</td>
<td>* P = 0.023</td>
</tr>
<tr>
<td>Stage 4 (min)</td>
<td>24 ± 26</td>
<td></td>
</tr>
<tr>
<td>SWA (min)</td>
<td>54 ± 64</td>
<td></td>
</tr>
<tr>
<td>REM sleep (min)</td>
<td>95 ± 86</td>
<td></td>
</tr>
</tbody>
</table>

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**Mathematical Model of Food Intake**

- Top panel will be the sum of three Gaussian curves to describe the average cumulative caloric intake of Control and NES participants. The bottom panel will depict the individual Gaussian curves that describe the average rate of eating during each of three separate meals.

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**Intake Models of NES**

- Control participants (n = 68)
- NES participants (n = 148)
### NES Characteristics by Group

<table>
<thead>
<tr>
<th>Variable</th>
<th>NES</th>
<th>Controls</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Daily Calories</td>
<td>2546</td>
<td>2230</td>
<td>.03</td>
</tr>
<tr>
<td>% calories after dinner</td>
<td>37.5%</td>
<td>10.9%</td>
<td>&lt; .001</td>
</tr>
<tr>
<td># awakenings/week</td>
<td>9.8</td>
<td>2.9</td>
<td>&lt; .001</td>
</tr>
<tr>
<td># nocturnal ingestions/wk</td>
<td>7.6</td>
<td>0.1</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

### NES Characteristics by Group

<table>
<thead>
<tr>
<th>Variable</th>
<th>NES</th>
<th>Controls</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beck Depression Inv.</td>
<td>16.7</td>
<td>3.9</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>EDE Global</td>
<td>1.95</td>
<td>0.71</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Pittsburgh Sleep Global</td>
<td>9.5</td>
<td>3.5</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Axis I diagnoses (Lifetime – any)</td>
<td>72%</td>
<td>33%</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

### Carbohydrate Consumption

- **p < 0.07**
  - NES: 59%
  - Con: 66%

- **p < 0.03**
  - NES: 68%
  - Con: 76%

- **n.s.**
  - NES: 19%
  - Con: 19%

### Fat Consumption

- **p < 0.001**
  - NES: 22%
  - Con: 13%

- **n.s.**
  - NES: 19%
  - Con: 19%

### Protein Consumption

- **p < 0.05**
  - NES: 10%
  - Con: 11%

- **n.s.**
  - NES: 22%
  - Con: 21%

### Circadian analysis of neuroendocrine data

**GLUCOSE** showed an inverted phase (delayed in NES by 11.6 hours) relative to Control subjects (*p* < 0.0001)

**INSULIN** was phase delayed in NES by 2.8 hours (*p* < 0.004), and was half the amplitude (*p* < 0.001) relative to Control subjects.

Goel et al., J Biol Rhythms, 2009
Circadian analysis of neuroendocrine data
(nonlinear mixed harmonic regression)

**LEPTIN** was phase delayed in NES by 1.0 hour relative to Control subjects ($p=0.042$).

**GHRELIN** was phase advanced in NES by 5.2 hours ($p<0.001$), and was half the amplitude ($p=0.021$) relative to Control subjects.

Leptin is an adipocyte-derived hormone that suppresses appetite;

Ghrelin is predominantly a stomach-derived peptide that stimulates appetite.

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**Treatment Background**

First attempt at therapy by Stunkard psychodynamic

- Later case reports (behavioral treatment; 1986 & 1989) with mixed results
- Case reports of phototherapy (2002 & 2006) successful, but 2002 case relapsed when therapy ended

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**Pharmacotherapy trials**

- Case reports
  - D-fenfluramine helpful (Spaggiari, 1994; O’Reardon et al., 2004)
  - SSRIs – paroxetine - Successful in four cases
    - fluvoxamine - Successful in one case (Miyaoka et al., 2003)
- Open label trial of sertraline
  - O’Reardon, Stunkard, & Allison, 2004

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**Outcome Measures**

- Night Eating Symptom Scale
- % Intake after Evening Meal
- # of Nocturnal Ingestions in Past Week
- # Awakenings
- Weight change
- Quality of Life (Quality of Life, Enjoyment, and Satisfaction Questionnaire)
- Mood (Beck Depression Inventory – II)

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**Randomized controlled trial of sertraline**

- Patients randomized to sertraline or placebo for 8-week active phase
- 17 on sertraline, 17 on placebo
- 1 participant from each group was unblinded early at their request due to no response
- 11 females, 3 normal weight, 12 Caucasian

O’Reardon et al., Am J Psychiatry 2006;163:893-8
Randomized controlled trial of sertraline

- 71% in sertraline group vs. 18% in placebo group – "responders"
- 41% of sertraline group – "remitters"

O’Reardon et al., Am J Psychiat 2006;163:893-8

Open label treatment with escitalopram

- 342 screened → 75 attended screening → 31 participants (18 completers)
  - 68% female
  - 45% Caucasian, 39% African American
  - Mean age: 45.4 ± 11.6 yrs.
  - Mean BMI: 31.8 ± 6.4 kg/m²
- Baseline screening:
  - NEQ, NESHI, EDE, SCID, BDI, Food/Sleep logs

Allison et al., Eating Behaviors, 2013
Participant characteristics

<table>
<thead>
<tr>
<th>Baseline characteristics</th>
<th>Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>% calories after dinner until morning awakening</td>
<td>34.9 (12.3)</td>
</tr>
<tr>
<td>% calories evening meal to bedtime</td>
<td>21.8 (14.8)</td>
</tr>
<tr>
<td>% calories after bedtime to morning awakening</td>
<td>13.1 (10.7)</td>
</tr>
<tr>
<td>No. awakenings/week</td>
<td>8.7 (6.9)</td>
</tr>
<tr>
<td>No. nocturnal ingestions/week</td>
<td>3.7 (3.8)</td>
</tr>
<tr>
<td>Baseline total caloric intake</td>
<td>2743.3 (1051.6)</td>
</tr>
<tr>
<td>Night Eating Questionnaire</td>
<td>31.7 (6.9)</td>
</tr>
<tr>
<td>Beck Depression Inventory</td>
<td>14.8 (11.6)</td>
</tr>
</tbody>
</table>

Allison et al., Eating Behaviors, 2013

Participants - SCID diagnoses

<table>
<thead>
<tr>
<th>Co-morbid psychopathology</th>
<th>N (Percentage)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any lifetime Axis I SCID diagnosis</td>
<td>20 (65%)</td>
</tr>
<tr>
<td>Binge eating disorder (all current)</td>
<td>7 (23%)</td>
</tr>
<tr>
<td>Any lifetime mood disorder</td>
<td>7 (23%)</td>
</tr>
<tr>
<td>Any lifetime anxiety disorder</td>
<td>7 (23%)</td>
</tr>
<tr>
<td>Any lifetime substance/alcohol abuse or dependence</td>
<td>3 (16%)</td>
</tr>
</tbody>
</table>

Allison et al., Eating Behaviors, 2013

Change in NESS

NESS – Night Eating Symptom Scale, decreased by 49%, \( p < 0.001 \)

Allison et al., Eating Behaviors, 2013

Change in Evening Hyperphagia

Decreased by 62%, \( p < 0.001 \)

Allison et al., Eating Behaviors, 2013

Change in Awakenings & Nocturnal Ingestions

80% decrease in NIs; 67% decrease in Awakenings, \( p \% < 0.001 \)

Allison et al., Eating Behaviors, 2013

Cognitive Behavioral Therapy (CBT)

- Cognitive behavioral therapy has been validated for treatment of AN, BN, BED, insomnia, and depression.
- Started by collecting data on thoughts related to night eating episodes
- Main goal:
  - Shift the delayed eating schedule back to the day by promoting a regular daytime eating schedule and eliminating nocturnal ingestions.
CBT for NES
Patient characteristics

- Pilot study (Allison et al., 2010, Am J Psychotherapy)
- 25 enrolled (14 completers)*
- 6 males (3 completers)
- 17 Caucasians, 6 African Amer., 1 Hispanic, 1 Other
- 8 normal weight
- 10 obese
- Mean age: 46.8 ± 13.4 yrs.
- Mean BMI: 29.5 ± 7.5 kg/m²

* drop-outs had more awakenings & nocturnal ingestions at baseline

Change in % kCals after Dinner

Change in awakenings and ingestions

Change in NESS & depressed mood

Conclusions for total sample

- CBT was effective in reducing nocturnal ingestions and percentage of caloric intake after dinner, mainly due to decrease in calories after initial sleep onset.
- There were overall improvements, as measured on NESS and BDI.
- Weight decreased significantly overall by 3.1 kg (6.8 lbs).
Normal Wt. vs. Ovwt/Obese

<table>
<thead>
<tr>
<th>Treatment Baseline</th>
<th>Normal Wt.</th>
<th>Ovwt/Obese</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI (kg/m²)</td>
<td>22.6</td>
<td>32.9</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Quality of Life</td>
<td>56.4</td>
<td>43.2</td>
<td>.01</td>
</tr>
<tr>
<td>% Calories after dinner</td>
<td>46.1</td>
<td>31.3</td>
<td>.015</td>
</tr>
<tr>
<td># Nocturnal ingestions/wk</td>
<td>13.1</td>
<td>6.7</td>
<td>.21</td>
</tr>
<tr>
<td>Baseline total caloric intake</td>
<td>2010.9</td>
<td>2531.0</td>
<td>.32</td>
</tr>
</tbody>
</table>

NESS, BDI, age did not differ. All normal weight patients were white, females.

Treatment Outcomes by Weight

% Calories after dinner # Nocturnal ingestions
All changes significant at p < .05 or smaller. NW (n = 8), Ovwt/ObE (n = 17)

Implications for Treatment

- With **overweight and obese** patients, focus is on regulating circadian pattern of eating (decreasing evening overeating and nocturnal ingestions) and reducing overall caloric intake
- With **normal weight** patients, focus is on regulating circadian pattern of eating and reducing compensation (restriction and excessive exercise).

Worth mentioning...

- Bright light therapy has been effective in 3 published cases of NES (Friedman et al., 2006; Friedman et al., 2002).
- Given the circadian shift, it seems worth pursuing!
- Topiramate – three case series, 1 RCT underway

Summary

- NES will likely be gaining more attention with its mention in DSM 5
- NES is related to circadian shifts (delays) in neuroendocrine patterns and sleep disruption
- Psychological and nutrition component
- Treatment so far – SSRIs and CBT

Conclusion...

- Those with NES are distressed, experiencing increasing BMI and may be at risk for increased metabolic disorder = CLINICALLY MEANINGFUL!
- Therefore, it is important to assess for night eating – it’s just a question – you may be surprised by what you hear…
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Thank you!

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• Please complete the evaluation soon after you receive the email. The evaluation does expire after 3 weeks. Once expired, you cannot obtain a certificate.
• Once the evaluation is completed, the CE certificate will be emailed separately within 2 business days.

Questions and Answers!

Moderator: Rebecca Shenkman, MPH, RDN, LDN
Email: cope@villanova.edu
Web site: villanova.edu/COPE

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