• Training Course for Healthcare Practitioners
• Why are we here?

We are here because you will have to make a choice
Either you believe in your patients:

I need help! I keep gaining weight and i'm eating normally!

I don't know what to do to make this weight gain stop! Oh, and I also run for 40 minutes every morning on my treadmill at 6.3mph and do some light weights. I'm so afraid that I am going to just keep gaining and I don't know what to do anymore!! I am so frustrated. I am still okay with my body now (i wear probably around an 8/10 now) but I definitely don't want to gain anymore

OR.....

- **Obesity Epidemic: Overeating Alone to Blame**
  
  America's Obesity Problem Is caused by overeating rather than inactivity, new study says
Fat people are just greedy, says BMA chief

The head of the British Medical Association has sparked a row after claiming that fat people are simply greedy.

Dr Meldrum said an obsession with labels may be stopping overweight people from tackling their problems. He said: "We are saying 'This patient has a hyper-appetite problem' rather than 'They are just greedy'."
Just greedy, or gluttons?
### Table 1

Features of Diet Studies With Long-Term Follow-Ups (and No Control Groups)

<table>
<thead>
<tr>
<th>Study</th>
<th>Years of follow-up</th>
<th>N</th>
<th>% of N in follow-up</th>
<th>% self-reporting weight</th>
<th>% on additional diets (or mean number of diets)</th>
<th>% reporting regular exercise</th>
<th>Mean change from baseline to end of diet (kg)</th>
<th>Mean change from baseline to follow-up (kg)</th>
<th>% regain all lost weight (or more)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anderson et al. (1999)</td>
<td>5-7</td>
<td>52</td>
<td>12</td>
<td>30</td>
<td>20&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-</td>
<td>-29.7</td>
<td>-5.2</td>
<td>-</td>
</tr>
<tr>
<td>Foster et al. (1996)</td>
<td>5</td>
<td>55</td>
<td>47</td>
<td>0</td>
<td>65 [M = 1 diet]</td>
<td>-</td>
<td>-21.1</td>
<td>+3.6</td>
<td>50% were &gt; 5 kg above baseline</td>
</tr>
<tr>
<td>Graham et al. (1983)</td>
<td>4.5</td>
<td>60</td>
<td>43</td>
<td>0</td>
<td>(M = 3 types of treatments) &lt;br&gt; &gt;50&lt;sup&gt;b&lt;/sup&gt;</td>
<td>35</td>
<td>-4.5</td>
<td>-3.3</td>
<td>-</td>
</tr>
<tr>
<td>Hensrud et al. (1994)</td>
<td>4</td>
<td>21</td>
<td>88</td>
<td>0</td>
<td>&gt;50&lt;sup&gt;b&lt;/sup&gt;</td>
<td>22</td>
<td>-12.5</td>
<td>-1.6</td>
<td>37</td>
</tr>
<tr>
<td>Jordan et al. (1985)</td>
<td>5</td>
<td>111</td>
<td>25</td>
<td>100</td>
<td>-</td>
<td>-</td>
<td>-8.4</td>
<td>-5.2</td>
<td>-</td>
</tr>
<tr>
<td>Kramer et al. (1989)</td>
<td>4</td>
<td>152</td>
<td>77</td>
<td>7</td>
<td>(M = 1.3 diets/year)</td>
<td>-</td>
<td>-11.9</td>
<td>-3.1</td>
<td>38</td>
</tr>
<tr>
<td>Lantz et al. (2003)</td>
<td>4</td>
<td>54</td>
<td>48</td>
<td>0</td>
<td>-</td>
<td>-</td>
<td>-7.0</td>
<td>-3.3</td>
<td>-</td>
</tr>
<tr>
<td>Murphy et al. (1985)</td>
<td>4</td>
<td>25</td>
<td>33</td>
<td>0</td>
<td>38 (M = 1.6 diets)</td>
<td>46</td>
<td>-7.7</td>
<td>-0.5</td>
<td>-</td>
</tr>
<tr>
<td>Pekkarinen and Mustajoki</td>
<td>5.5</td>
<td>24</td>
<td>88</td>
<td>13</td>
<td>12% lost &gt; 10 kg on other diets&lt;sup&gt;c&lt;/sup&gt;</td>
<td>-</td>
<td>-22.9&lt;sup&gt;d&lt;/sup&gt;</td>
<td>-5.8</td>
<td>29</td>
</tr>
<tr>
<td>Stalenos et al. (1984)</td>
<td>5</td>
<td>36</td>
<td>81</td>
<td>22</td>
<td>(M = 2 diets)</td>
<td>-</td>
<td>-4.7</td>
<td>+0.7</td>
<td>46</td>
</tr>
<tr>
<td>Stunkard and Penick</td>
<td>5</td>
<td>26</td>
<td>81</td>
<td>63</td>
<td>-</td>
<td>-</td>
<td>-8.8</td>
<td>-5.4</td>
<td>31</td>
</tr>
<tr>
<td>Wadden and Frey (1997)</td>
<td>5</td>
<td>281</td>
<td>22</td>
<td>100</td>
<td>43</td>
<td>-</td>
<td>-25.6</td>
<td>-6.6</td>
<td>32</td>
</tr>
<tr>
<td>Wadden et al. (1989)</td>
<td>5</td>
<td>55</td>
<td>72</td>
<td>47</td>
<td>55</td>
<td>-</td>
<td>-14.6</td>
<td>-0.6</td>
<td>64</td>
</tr>
<tr>
<td>Walsh and Flynn (1995)</td>
<td>4.5</td>
<td>143</td>
<td>47</td>
<td>100</td>
<td>36</td>
<td>-</td>
<td>-21.4</td>
<td>-5.1</td>
<td>-</td>
</tr>
</tbody>
</table>
• We sustain:

1. That *overeating is not the main cause for obesity, but its consequence*. Weight gain is the visible sign of an underlying regulatory disorder.

2. That the cause for obesity lies in the hypothalamic area.

3. That no dietary procedure will improve the disease, unless some type of correction of the diencephalic disorder is being simultaneously provided.

4. That, as by now, the only rational approach to treat the disease is the hCG method for weightloss.
Diseases associated to obesity

- Diabetes
- **Cancer:** Obesity increases the risk of cancer development by 25-33%. Obese person is likely to develop esophageal cancer, prostate cancer, breast cancer, kidney cancer, endometrial cancer or colon cancer.
- Congestive Heart Failure
- Heart Enlargement.
- Stroke
- Polycystic Ovarian Syndrome
- Pulmonary Embolism
- Gastro-esophageal Reflux or Heartburn
- Osteoarthritis
- Fatty Liver Disease
- Erectile Dysfunction
- Chronic Renal Failure
- Lymph Edema
- Urinary Incontinence
- Dislipidemias
• Depression
• Cellulitis
• Gallbladder Disease
• Gout
• **Pickwickian Syndrome**: Excess weight adds pressure on the pulmonary system, hence, leading to Pickwickian syndrome characterized by sleep apnea.
• **Hernia**: This condition is causes weak and enlarged diaphragm
• Metabolic syndrome
• OBESITY?
Obesity according to current definitions

- Obesity definition is very straightforward: obesity is simply defined as a condition of being overweight.
Overweight according to current definitions

- In one sense it is a way of saying imprecisely that someone is heavy(?).

The other sense of "overweight" is more precise and designates a state between normal weight and obesity (?).
  - [www.encyclo.co.uk/define/Overweight](http://www.encyclo.co.uk/define/Overweight)
And, finally, **obesity** is…….

The state of being well above one's normal weight (overweight).

• Therefore, obesity and overweight are directly related terms. Right?
No.
• Obesity and overweight are not always related terms
Let’s us redefine obesity....

- Obesity is a clinical disorder characterized by the capacity of the hypothalamus to accumulate fat well over daily requirements.

  These adipose tissue masses are located in conspicuous body areas

- Overweight, may or may be not, related to obesity.
<table>
<thead>
<tr>
<th></th>
<th>Diencephalic disorder</th>
<th>Adipose tissue mass</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal weight</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>Overweight not obese</td>
<td>No (eventually later)</td>
<td></td>
</tr>
<tr>
<td>Obese no overweight</td>
<td>Yes</td>
<td>or:</td>
</tr>
<tr>
<td>Obese and overweight</td>
<td>Yes</td>
<td></td>
</tr>
</tbody>
</table>
First Myth

- The hCG method is a medical procedure that can cure obesity, “resetting” the hypothalamus and allowing all types of foods after treatment.
What we do with the hCG method for weight loss:

- We provide a safe, time-tested, reliable and effective method for obesity treatment.
- Weight loss will be accomplished at the expense of adipose tissue and not lean mass.
- Our patients feel in a good mood throughout the entire treatment period.
What we don’t do with the hCG method for weight loss:

- We do not cure obesity.
- We do not “reset” the hypothalamus.
- We do not allow all type of foods after the treatment.
Second Myth

- The hCG protocol is an unsafe and unreliable procedure for obesity treatment.
Obesity - Overcoming Myths

- Over 6,500 first-hand treated patients in our practice, and 14,000 patients reviewed at the Bellevue Klinik, in Switzerland.

- Twenty-nine years of personal experience on the method.

- No complications described.
Third Myth

- The scale is an useful tool to assess the efficacy of an obesity treatment.
Fat percentage

7% 10% 14% 20% 25% 30%

Fat percentage

The use of the scale to assess obesity is an unreliable tool to estimate the disease.

**Obesity Trends* Among U.S. Adults**
**BRFSS, 1990, 1999, 2008**
(*BMI ≥30, or about 30 lbs. overweight for 5’4” person*)

Source: CDC Behavioral Risk Factor Surveillance System.
Fourth Myth

- Exercising and “shutting your mouth” are the most effective tools for obesity treatment.
Exercise alone is probably insufficient to bring about significant fat loss except in individuals who are extremely motivated.

Although prolonged, intense physical exercise may promote weight loss, more moderate exercise, as practiced by non-athletes may not induce significant weight loss.

In some cases, weight gain has actually been reported.

http://www.exrx.net/FatLoss/ExTherapy.html
Exercise or no-exercise for weight loss

- **Diet**
- **Exercise**
- **Diet + Exercise**

Weight Loss (kg)

0 Months to 6 Months
• It seems the old mantra:

“Shut your mouth and exercise” were not effective for obesity treatment

Obesity Trends* Among U.S. Adults
(*BMI ≥30, or about 30 lbs. overweight for 5’4” person)

Source: Behavioral Risk Factor Surveillance System, CDC.
Fifth Myth

- Overeating always results in weight gain.
Figure 2. The dietary intake and weight gain in subject P. W., Group IV Protocol as in Figure 1. Note that this subject took a much greater excess of calories above the basal requirement, but failed to gain more than 14 per cent above his initial weight. 2700 kcal/M² in this instance was inadequate to maintain weight gained.
Figure 3. Weight gain from ingestion of an excess of dietary fat alone in subject S. T., Group V. Note that weight is apparently gained more efficiently than in the case of the subjects of Figures 1 and 2, taking a mixed diet. Also note that the excess weight is maintained by a caloric intake equal to the basal level.
Sixth Myth

- Obesity is a psychological disorder, characterized by anxiety, gluttony and/or greed.

- Therefore, psychotherapy or behavior therapy can provide relief to the disease.
Are cats and rats obese because a psychological disorder?


The act of eating, may, or MAY NOT be related to a desire for food. Rather, it suggests that a regulatory disorder is present in the hypothalamic area.
• The disorder responsible for obesity genesis is located BELOW the brain cortex.
• Therefore, no amount of psychological support may help obese patients.
• We are dealing with a hypothalamic regulatory disorder.
• Neuronal pathways of obesity closely resemble those related to an addiction.
The diencephalic theory of Obesity
The hypothalamus functions (I):

- Pituitary gland regulation.
- Blood pressure regulation.
- Hunger and salt cravings feeding reflexes.
- Fat deposition and release.
- Thirst.
- Body temperature regulation.
- Hydration and water preservation.
- Heart rate.
- Bladder function.
The hypothalamus functions (II):

- Hormonal/neurotransmitter regulation.
- Ovarian and testicular function.
- Mood & behavioral functions.
- Wakefulness metabolism.
- Sleep cycles.
- Energy levels.
- Adaptation to stress.
The hypothalamus - Obesity

- The hypothalamus functioning may become disrupted by a series of psychological (divorce, death of a relative, etc.) and physiological (concomitant diseases, etc.) agents.

- Once this disorder triggers one of the most sensitive areas within the hypothalamus are those related to adipose tissue metabolism.

http://www.psycheducation.org/emotion/L1HYPOTHL.jpg
The hypothalamus - Obesity Experimental field

- Surgical destruction of Ventromedial and Lateral Hypothalamic Nuclei causes hyperphagia, hyperinsulinemia and obesity in experimental animals.
In humans
Figure 1. Neuronal response to visual foods cues in thin individuals in the eucaloric state. The neuronal response in thin individuals to visual stimuli of foods of high hedonic value as compared to non-food objects in the eucaloric state is shown (EUH > 0). Robust activation is observed in the insula, sensory cortex, posterior cingulate, ventral striatum, posterior hippocampus, parietal cortex, and inferior temporal visual cortex. Statistical maps thresholded at an FDR corrected threshold of q < 0.05 and overlaid onto the group average anatomical image. Data are shown in the radiological convention (right hemisphere on the left).

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