Toxicity of the Adipose Tissue and Human Thinking

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When an organ becomes toxic for our body? When an organ becomes too large (hypertrophy, hyperplasia) and produces large quantities of hormones or other products, then we may have toxic results. Like the toxic goiter, for example, in thyroid diseases. The role of adipose tissue as an endocrine gland has been studied in detail by many medical specialties. When the adipose tissue becomes toxic due to its large size one main alteration is produced in thinking; thinking may be dependent in hormonal changes and may alter dramatically in obesity. Obesity has many times been connected to an addiction for eating. Addictions have been confirmed as serious and difficult or impossible to treat neurological conditions, with massive influence in the intelligence and emotional status of the patients. Obesity has not been treated as a genuine neurological problem, but classically as a metabolic disease. Of course, the psychological aspects of obesity have been studied in depth, and the cognitive methods for obesity treatment have been presented in detail in research studies, but the intelligence problem has not been considered a neurological consequence so far. The concept of toxicity in the human thinking caused by the overdeveloped adipose tissue should be considered. The vicious circle that makes obese people to eat without control or adds more stress to the stress eaters are toxic phenomena, which resemble to toxicity (impulse for eating, inability to resist and think more wisely when the impulse comes).

Toxic results of the Adipose Tissue in Obese

a) Toxic Result 1 - Food motivation in obese

Martin et al. showed that brain function associated with food motivation differs in obese and healthy weight adults and may have implications for understanding brain mechanisms contributing to overeating and obesity, and variability in response to diet interventions. Food motivation, which has been found to be higher in obese compared to healthy weight individuals. Martin et al. used functional magnetic resonance imaging to examine changes in the hemodynamic response in obese and healthy weight adults while they viewed food and nonfood images in premeal and postmeal states. During the premeal condition, obese participants showed increased activation, compared to healthy weight participants, in anterior cingulate cortex and medial prefrontal cortex. Moreover, in the obese group, self-report measures of disinhibition were negatively correlated with premeal anterior cingulated cortex activations and self-report measures of hunger were positively correlated with premeal medial prefrontal cortex activations. During the post-meal condition, obese participants also showed greater activation than healthy weight participants in the medial prefrontal cortex.

b) Toxic Result 2 – Depression, anxiety and schizophrenia in obese

According to Rivenes AC et al., abdominal fat distribution (as measured by waist to hip ratio) appears to be the key mediator in the relationship between obesity and depression; the authors support the hypothesis that obesity and depression link via metabolic disturbances involving the hypothalamic-pituitary-adrenocortical axis.

Dr. Rivenes et al. examined the relationship between depression, anxiety, and two different measures of obesity in a large community-based sample. The HUNT-2 study was used to conduct a cross-sectional study of 65,648 adults between 20 and 89 years of age. During a clinical examination, trained nurses took various anthropometric measurements allowing participants’ body mass index (BMI) and waist-hip ratio (WHR) to be calculated. Anxiety and depressive symptoms were ascertained using the Hospital Anxiety and Depression Scale. Information regarding physical activity, level of social support, and medical comorbidity was also obtained. According to the results, elevated WHR was associated with increased prevalence of both anxiety and depression. After adjustment for BMI, physical activity, social isolation, and somatic diseases, WHR remained independently associated with depression in both males and females and with anxiety among males. Obesity, as defined by BMI, was associated with depression; however, this association was strongly attenuated by WHR. There was a negative association between BMI and anxiety in both genders. Levels of obesity are higher in those with schizophrenia and depression.

c) Toxic Result 3- Compulsive overeating behavior as an addiction disorder; is overeating a neurological symptom?

Davis C and Carter JC correlated compulsive overeating and addiction disorders. They supported that compulsive overeating has compelling similarities to conventional drug addiction. Their concept is based on the comparable clinical features, the biological mechanisms they have in common, and on evidence that the two disorders have a shared diathesis. In making the argument for overeating as an addictive behaviour, it is clearly not appropriate to include all cases of excessive food consumption in this taxon. The authors did not claim that obesity and addiction are one and the same. However, it was proposed that Binge Eating Disorder is a phenotype particularly well-suited to such a conceptualization, and that sound clinical and scientific evidence exists to support this viewpoint. Also the authors have provided some recommendations for treatment modifications that recognize the similarities between treating drug dependence and compulsive overeating.

d) Toxic Result 4- Stress-eating and emotional pleasure by eating

Dallman MF supported the hypothesis that the stress and emotional brain networks foster eating behaviors that can lead to obesity. The neural networks underlying the complex interactions among stressors, body, brain and food intake are...