GLUCOSTATIC THEORY OF APPETITE CONTROL pdf

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The glucostatic theory was not abandoned altogether, as it was still thought to be important for short-term appetite control, but newly discovered peptides such as leptin became more likely candidates for long-term control.

Hunger pangs[edit] When hunger contractions start to occur in the stomach , they are informally referred to as hunger pangs. Hunger pangs usually do not begin until 12 to 24 hours after the last ingestion of food. A single hunger contraction lasts about 30 seconds, and pangs continue for around 30 to 45 minutes, then hunger subsides for around 30 to minutes. Individual contractions are separated at first, but are almost continuous after a certain amount of time. Emotional states anger, joy etc. Levels of hunger are increased by lower blood sugar levels, and are higher in diabetics. They reach their greatest intensity in three to four days and may weaken in the succeeding days, although research suggests that hunger never disappears. Hunger contractions are most intense in young, healthy people who have high degrees of gastrointestinal tonus. Periods between contractions increase with old age. Neural signals from the GI tract[edit] One method that the brain uses to evaluate the contents of the gut is through vagal nerve fibers that carry signals between the brain and the gastrointestinal tract GI tract. Stretch receptors work to inhibit appetite upon distention of the GI tract by sending signals along the vagus nerve afferent pathway and inhibiting the hunger center. Nutrient signals that indicate fullness, and therefore inhibit hunger include rising blood glucose levels, elevated blood levels of amino acids, and blood concentrations of fatty acids. CCK is key in suppressing hunger because of its role in inhibiting neuropeptide Y. Glucagon and epinephrine levels rise during fasting and stimulate hunger. Ghrelin , a hormone produced by the stomach, is a hunger stimulant. Liking refers to the palatability or taste of the food, which is reduced by repeated consumption. Wanting is the motivation to consume the food, which is also reduced by repeated consumption of a food [7] and may be due to change in memory-related processes. Thoughts of a food may intrude on consciousness and be elaborated on, for instance, as when one sees a commercial or smells a desirable food. Leptin is a peptide hormone that affects homeostasis and immune responses. Later studies showed that appetite regulation is an immensely complex process involving the gastrointestinal tract , many hormones , and both the central and autonomic nervous systems. Many brain neurotransmitters affect appetite, especially dopamine and serotonin. Opioid receptor -related processes in the nucleus accumbens and ventral pallidum affect the palatability of foods. The few important signalling molecules inside the NAc shell modulate the motivation to eat and the affective reactions for food. They are produced by the digestive tract and by adipose tissue leptin. Leptin, a hormone secreted exclusively by adipose cells in response to an increase in body fat mass, is an important component in the regulation of long term hunger and food intake. When leptin levels rise in the bloodstream they bind to receptors in ARC. The functions of leptin are to: Suppress the release of neuropeptide Y NPY , which in turn prevents the release of appetite enhancing orexins from the lateral hypothalamus. This decreases appetite and food intake, promoting weight loss. Stimulate the expression of cocaine and amphetamine regulated transcript CART. Though rising blood levels of leptin do promote weight loss to some extent, its main role is to protect the body against weight loss in times of nutritional deprivation. Other factors also have been shown to effect long-term hunger and food intake regulation including insulin. Processes from other cerebral loci, such as from the limbic system and the cerebral cortex , project on the hypothalamus and modify appetite. This explains why in clinical depression and stress , energy intake can change quite drastically. Set-point theories of hunger and eating [edit] The set-point theories of hunger and eating are a group of theories developed in the s and s that operate under the assumption that hunger is the result of an energy deficit and that eating is a means by which energy resources are returned to their optimal level, or energy set-point. The set-point assumption is a negative feedback mechanism. The set-point theories of hunger and eating present a number of weaknesses. Humans engage in sexual behavior, not because of an internal deficit, but instead because they have evolved to crave it. Similarly, the evolutionary pressures of unexpected food shortages have shaped humans and all other warm blooded animals to take advantage of food when it is present. It is the presence of good food, or the mere anticipation of it that makes one hungry. However, when a meal is consumed, there is a

homeostasis-disturbing influx of fuels into the bloodstream. When the usual mealtime approaches, the body takes steps to soften the impact of the homeostasis-disturbing influx of fuels by releasing insulin into the blood, and lowering the blood glucose levels. It is this lowering of blood glucose levels that causes premeal hunger, and not necessarily an energy deficit. Similarly, thirst is the craving for water.

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2: How is food intake controlled

The glucostatic theory of appetite control and the risk of obesity and diabetes. Chaput JP(1), Tremblay A. Author information: (1)Division of Kinesiology, Faculty of Medicine, Department of Social and Preventive Medicine, Laval University, Quebec City, Quebec, Canada.

The explanatory power of the Selfish Brain theory[edit] Investigative approach of the Selfish Brain theory[edit] The brain performs many functions for the human organism. Most are of a cognitive nature or concern the regulation of the motor system. A previously lesser investigated aspect of brain activity was the regulation of energy metabolism. The "Selfish Brain" theory shed new light on this function. It states that the brain behaves selfishly by controlling energy fluxes in such a way that it allocates energy to itself before the needs of the other organs are satisfied. The internal energy consumption of the brain is very high. Before now the scientific community assumed that the energy needs of the brain, the muscles and the organs were all met in parallel. The hypothalamus, an area of the upper brainstem, was thought to play a central role in regulating two feedback loops within narrow limits. The "lipostatic theory" established by Gordon C Kennedy in describes the fat deposition feedback system. The "glucostatic theory" developed in the same year by Jean Mayer describes the blood glucose feedback system. In this way a certain glucose concentration is set by adjusting the intake of nutrients. Mayer also included the brain in his calculations. Although he considered that food intake served to safeguard the energy homoeostasis of the central nervous system, he did imply that the energy flux from the body to the brain was a passive process. On the basis of these theories a number of international research groups still position the origin of obesity in a disorder in one of the two above described feedback systems. However, there are scenarios in weight regulation that can not be explained in this way. For example, upon inanition of the body e. The "Selfish Brain" theory links in seamlessly with the traditions of the lipo- and glucostatic theories. Whenever required the cerebral hemispheres direct an energy flux from the body to the brain to maintain its energy status. The "Selfish Brain" theory combines the theories of Kennedy and Mayer, considering blood glucose and fat feedback control systems as a complex. This regulates the energy flux from the environment to the body, i. It is regulated by a hypothalamic nucleus. If these signals are not processed correctly, e. The origin of this is not to be found in the blood glucose or fat feedback control systems, but much rather in the regulating instances within the cerebral hemispheres. Energy procurement by the brain[edit] The brain can cover its energy needs particularly those of the cerebral hemispheres either by allocation or nutrient intake. The corresponding signal to the subordinate regulatory system originates in the cerebral hemispheres. The most phylogenetically recent part of the brain is characterized by a high plasticity and a high capacity to learn with this process. It is always able to adapt its regulatory processes by processing responses from the periphery, memorizing the results of individual feedback loops and behaviors, and anticipating any possible build-ups. Energy procurement by the brain is complicated by three factors. Firstly, the brain always requests energy whenever it is needed. It can only store energy in a very restricted form. Peters therefore refers to this as an "energy on demand" system. Secondly, the brain is almost exclusively dependent on glucose as an ATP-substrate. Lactate and betahydroxybutyric acid can also be considered as substrates, but only under certain conditions, e. The blood glucose has to be brought there via a special, insulin-independent transporter. The healthy and the diseased brain: It diverts blood glucose from the periphery and leads it across the blood-brain-barrier. An important role here is played by the stress system, whose neural pathways lead directly to the organs heart, muscle, adipose tissue, liver, pancreas, etc. This system ensures that the glucose is transported to the brain, and that uptake by the musculature and the adipose tissue is reduced. In order to achieve that, the release of insulin and its effect on organs is halted. The acute supply of energy to the brain from the intake of nutrients presents problems for the organism. In the event of an emergency food intake is only activated if allocation is insufficient, and must be taken as a sign of disease. In this case the required energy can not be requested from the body, and it can only be taken directly from the environment. This pathology is due to defects lying within the control centers of the brain such as the hippocampus, amygdala and hypothalamus. These may be due to mechanical tumors, injuries , genetic defects

lacking brain-derived neurotrophic factor BDNF receptors or leptin receptors , faulty programming post-traumatic stress disorder, conditioning of eating behavior, advertising for sweets or false signals may arise due to the influence of antidepressants, drugs, alcohol, pesticides, saccharin or viruses. Such disorders can have a negative impact on a number of behavioral types: Eating behavior eating, drinking Social behavior e. The following applies irrespective of the nature of energy provision: Peters therefore differentiates the healthy from the diseased brain through its ability to compete for its energy requirements even under adverse conditions where there are excessive demands from the body. He contraposes the "selfish brain with high fitness" that can tap the bodies energy reserves even in times of short food supply at the expense of the body mass, and the "selfish brain with low fitness", that is unable to do this, and which instead takes in additional food and bears the risk of developing obesity. Obesity - a build-up in the supply chain[edit] The "Selfish Brain" theory can be considered as a new way to understand obesity. Whatever the type of disruption that exists, it entails that the energy procurement for the brain is accomplished less by allocation and more by the intake of nutrients even though the muscles have no additional energy requirement. If one imagines the energy supply of the human organism as a supply-chain that passes from the outside world with its numerous options for nutrient intake via the body to the brain as the end user and control organ, then obesity can be considered as being caused by a build-up in this supply-chain. This is characterized by an excessive accumulation of energy in the adipose tissue or blood. An allocation failure is expressed as a weakening of the sympathetic nervous system SNS. The result is that energy intended for the brain mainly enters buffer storage areas, i. Only a small proportion reaches the brain. In order to cover its huge energy needs the brain commands the individual to consume more food. The accumulation process escalates, and the buffer storage areas are continuously filled up. This leads to the development of obesity. In such a situation the adipose tissue and musculature can no longer accept any energy, and the energy then accumulates in the blood so that hyperglycemia results. That was a novel methodological approach for diabetology. The regulation of adenosine tripophoshate content plays a central role a type of energy currency for the organism in the brain. Peters assumes a double feedback structure, where the ATP content in the neurons of the brain is stabilized by measurements from two sensors of differing sensitivity that produce the raw energy request signals. The more sensitive sensor records ATP deficits and induces an allocation signal for glucose that is compensated for by requests from the body. The other less sensitive sensor is only activated with glucose excesses and conveys a signal to halt the brain glucose allocation. The optimal ATP quantity is determined by the balance between these receptor signals. Peters considers that the stress system also operates according to this double feedback structure, which is also closely related to the supply of glucose to the brain. If an individual is confronted with a stress-inducing stimulus, it responds with an increased central-nervous information processing and along with that an increased glucose requirement in the brain. The hormone cortisol, important for regulating stress reactions, and the hormone adrenaline, important for glucose procurement, are released from the adrenal glands. The amount of cortisol that is released is also determined by a balance between a sensitive and a less sensitive sensor, just as is the case with the control of ATP content. This process is terminated if the stress system returns to a resting state. On the one hand it integrates peripheral metabolism research which investigates how energy metabolism functions through intake of nutrients into the organs of the body. On the other it incorporates the results of the brain metabolism expert Luc Pellerin from the University of Lausanne, who found that the neurons in the brain are supplied with energy via their neighboring astrocytes whenever required. This requirement oriented principle for the nerve cells is termed "Energy on demand". With this approach the "Selfish Brain" theory recognizes the description of two ends of a supply chain. The priority of the brain implies that the regulation of energy supply in a human organism is accomplished by the demand rather than the supply principle: Energy is ordered when it is needed. Energy supply chain of the "Selfish Brain". If the ATP concentration drops in the nerve cells of the brain, a cerebral mechanism is pull 1 set in motion which increases the energy flux directed from the body to the brain according to the "Energy on demand" principle. If the energy content in the body falls blood, adipose tissue , the falling glucose and the falling adipose tissue hormone leptin induce another cerebral mechanism pull 2. This entails that more energy is absorbed from the immediate environment into the body ingestion behavior. When the available supplies in

the immediate vicinity disappear, a further cerebral mechanism pull 3 initiates moving and exploration, i. The glucostatic and the lipostatic theories describe the second step in this supply chain area with dark grey background. The "Selfish Brain" theory links to the two traditional theories and expands them by considering the brain as an end consumer in a continuous supply chain light gray The founding of the "Selfish Brain" research group[edit] After the axioms were formulated in Achim Peters sought experts in other specialties to develop his "Selfish Brain" theory further. Already at an early stage he had matched up his ideas with the views of other leading international scientists. Amongst them was the Swiss brain metabolism specialist Luc Pellerin, the renowned obesity expert Denis G. Baskin, the internationally famous stress researcher Mary Dallman and the renowned neurobiologist Larry W. At the University of Luebeck Achim Peters compared his findings with the well-known neuroendocrinologist Prof. A year later in an intensive collaboration was started with the psychiatrist and psychotherapist Prof. Ulrich Schweiger who also worked at the University of Luebeck. In the interdisciplinary research group: Achim Peters was appointed to a professorship that was especially created for the group. He also succeeded in winning over additional reputable scientists for the project, including Prof. Rolf Hilgenfeld, an eminent SARS expert and the developer of one of the first inhibitors of the virus. At this time the research group consists of 18 scientific subproject investigators from a number of specialties including internal medicine, psychiatry, neurobiology, molecular medicine and mathematics. Poor coping strategies in stress situations represent one of these. An association was found between the tendency to evade conflict, and the habit of reducing psychological stress by immediately consuming sweets. The direct supply of glucose circumvents the glucose procurement from the body that would otherwise occur with a normal allocation process following the release of the stress hormone adrenaline. An existing allocation problem with obesity can be made even worse by such bad behavior.

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3: Hunger - Wikipedia

The glucostatic theory of appetite control and the risk of obesity and diabetes. Author(s): Chaput, J. P. ; Tremblay, A. Author Affiliation: Division of Kinesiology (PEPS), Department of Social and Preventive Medicine, Laval University, Quebec City, Quebec, G1K 7P4, Canada.

At its simplest form, overeating is characterised by excessive consumption of food beyond what is needed to achieve satiety. While this is a reasonably common occurrence, compulsive overeating is linked to more serious conditions such as bulimia or binge eating disorder and obesity Stoebe, , p. Although there are physiological cues for hunger and satiety, most people cognitively control what, when and how much food they eat. This chapter aims to determine the physiological hunger cues, patterns of self-regulation and the possible reasons why self-regulation of eating fails. In doing so this chapter also points out practical applications of the theory and research to help the reader avoid lapses in self-regulation that result in overeating. In being a physiological need, the motivation to eat is to some extent biological, controlled by neural and hormonal mechanisms. When stimulated these nerves cause a sense of satiety and eating behaviour is stopped. This theory of short-term appetite control is supported by recent research into the effect of high and low glycaemic index GI foods on hunger and satiety. Low GI foods, which slowly increase BGLs, are linked to increased satiety and a reduction in food intake throughout the day. Stomach distention[edit] Stomach distention is another short-term regulator of appetite. During a meal the stomach expands to accommodate the intake of food. The expansion is detected by gastric stretch receptors, which send a neural signal to various parts of the brain, including the amygdala and insula. This process results in a perception of fullness and causes eating behaviour to stop Wang et al. The stomach empties at a constant rate of approximately kilojoules kJ; aprox calories per hour. The cephalic reflexes are autonomic and endocrine responses, including increased saliva and gastric juice secretion, which prepare the body for digestion. These reflexes are the cause of the drooling over the smell of something cooking, or the tummy grumbles at the thought of a favourite food. While these are primarily physiological responses to sensory inputs we can be trained to associate things, that are originally unrelated to eating, with food. Temperature Based on studies in animals, it may be that people have a greater intake of food in cold environments than warm ones. In cold climates the body must use energy to maintain core body temperature. This phenomenon is possibly caused by increased energy expenditure required to maintain core temperature in colder conditions. The evidence surrounding this physiological control of appetite is limited and this is definitely an area for further research Brobeck, Lipostatic theory[edit] Long-term energy intake is suspected to be regulated by hormones secreted from adipose tissue, namely ghrelin and leptin. As adipose tissue is our store of energy for times of fasting, the body aims to keep fat mass a optimal homeostatic levels. A drop in fat mass stimulates an individual to consume more to replace those fat stores. This is mediated by the hormone ghrelin, which is secreted by adipose tissue when fat stores decrease. Ghrelin causes the stimulation of the[missing something? However, when fat stores exceed the homeostatic level adipose tissue secretes the hormone leptin. Take Home Message There are several biological mechanisms utilised by the body to regulate hunger and satiety. These mechanisms are designed to prevent a person from consuming to much or too little energy. Despite these physiological mechanisms to control food intake people regularly exceed their energy needs through over eating. Clearly the motivation to eat, and therefore overeat, is more than physiological. Self-regulation is influenced by several psychological, social, cultural and environmental factors, many of which contribute to a failure of self-regulation resulting in overeating. According to Baumeister, Heatherton and Tice the factors that cause self-regulation failure are distinguished into two patterns of behaviour: Conflicting standards, a reduction in monitoring, inadequate strength or willpower and psychological inertia. Conflicting standards[edit] In recent decades the Western world has experienced a movement towards the thin ideal, the social desirability of a slender body shape, particularly in women. Dieting is the conscious restriction of food intake to reduce or maintain weight. While restrained eating or dieting aims to reduce food intake, and in some cases this does work, often restrained eating results in increased food intake. The restraint theory suggests that restraint not only precedes overeating

but contributes to it. People consume more when they restrain eating and allow themselves to become exceptionally hungry. Several studies support this theory and demonstrate that restrained eatin, often leads to a loss of control, poor food choices and a tendency to overeat or binge Vitousek ; Polivy and Herman ; Polivy What is more, food restriction causes neurological adaptations relating to the reward of food. Food restriction can prevent negative alliesthesia, the process in which a sweet food becomes unpleasant to taste. Can your goal to eat well overcome the desire to eat something like this? Goal conflict theory[edit] The goal conflict theory of eating was developed by Wolfgang Stroebe and colleagues to provide a realistic model of the cognitive processes by which restrained and unrestrained individuals control their food intake. This theory suggests that restrained eaters experience a conflict between two goals, the goal to loose or maintain weight and the goal to enjoy palatable food. While these are both highly desirably end states for restrained eaters, the desire to loose weight is often the stronger of the two. Despite this, chronic dieters are sensitive to an environment rich in stimuli signaling or symbolising palatable food. Temptation activates automatic attempts to achieve the goal of weight loss through inhibiting thoughts of the pleasure of eating. However continued exposure to palatable foods is likely to override the long-term goal of weight loss in chronic dieters and result in over eating Stroebe, , p. It was found that restrained eaters paid more attention to palatable food when they had been pre-exposed to food cues such as appetising food words, while unrestrained eaters did not increase attention to palatable food. Advertising and food availability[edit] The findings of Papies et al. Priming refers to increasing the sensitivity of an individual to a particular stimuli as a result of a prior experience. Research by Harris, Bargh and Brownell revealed that both children and adults consumed more snack foods after being exposed to an advertisement, compared to other conditions. This increase in food intake was not related to hunger reports and foods that were consumed were not necessarily the foods advertised. This is a particularly important issue in current western society, in which food advertising is so prevalent. Futher, TV advertising exposure is linked to increased fat mass children and adolescence Worsley, Thus, overeating is encouraged by the priming effect of unheathly food advertisement. Conversely, self-regulation can also be enhanced through priming. By being subjected to a diet reinforcing poster, restrained eaters were less likely to snack on foods provided, indicating that diet priming may provide a tool to prevent overeating. Take Home Message By restricting our eating we try to bring hunger and satiety under cognitive control. This self regulation often fails, causing people to overeat. Underreguation through having conflicting standards contributes to this failure. Based on the information presented, strategies to prevent overeating include: Avoid restricting food intake during the day to prevent becoming excessively hungry. You will be less likely to binge, or to cause neurological alterations that are counter productive Avoid exposure to highly palatable foods or unhealthly food advertising before doing the shopping or having a meal. This can reduce the overeating priming affect of these ads. Prime yourself with healthy eating messages before a meal. Watch a youtube clip, look at a picture or think about what healthy eating means, which will prime you to eat onle based on hunger. Be conscious of the priming affect or food advertising when watching TV. By doing so you will be more likely to recognise what is real hunger and what cravings have been bought on by advertising. Reduction in monitoring[edit] The second behavioural pattern of under regulation is a reduction in monitoring of food intake. People fail to self-monitor when they experience reduced self-awareness. Self-awareness can be reduced by a number of distractions including TV, socialisting, variety, and intoxication. Would you eat more popcorn if you are watching a movie rather than sitting at a table? TV[edit] Increased television viewing has been linked to weight gain. Although, this is potentially due to a reduction in physically activity, television viewing is also related to a greater intake of energy dense foods. In a study by Blass et al , individuals consumed considerably more kJ when eating in front of the TV when eating alone. While the time taken to eat a food such as pizza remained the same in both conditions, when eating in front of the TV the time between slices reduced, allowing the subjects to consume more in the same time period. DO you pay as much attention to what your eating when you are with friends? Socialising[edit] People often consume more food when they are with other people than when they are eating alone Salvy et al. Interestingly some research suggests that some people consume less when in the presence of strangers, which may be a result of increase self awareness Salvy et al, Are you more likely to sample one of each food or only pick one? Variety[edit] The link between

variety of foods and overeating has been researched for several decades. Variety, and the desire to sample, distract an individual from monitoring their food intake. It is clear that the greater the variety of food provided the more an individual is likely to consume Remick et al. Do you eat more after a few beverages? Intoxication[edit] Alcohol consumption reduces self-awareness and monitoring by disinhibiting an individual. People consuming alcohol are less likely to keep track of what they are eating and are more likely to overeat Hull, A study by Caton, Ahearn and Hetherington found that the consumption of 4 standard drinks 10g of alcohol increased appetite and food intake in males under laboratory conditions. Take-home message There are several external factors which influence our ability to monitor self-regulation and may cause us to overeat. Strategies to prevent overeating include: Try to be conscious of physiological satiety cues when eating. Intentionally chew every mouthful and recognise when fullness is occurring Avoid eating in front of the TV, computer and other situations that draw attention and awareness away from the process of eating It is impractical to avoid eating with people, but when you are eating with others be mindful of what you are doing and remember that you are more likely to over eat in a social setting. When there is a large variety of food accept that you do not have to try everything - pick a few things that you are really interested in eating and let go of the other foods, they will only cause you to overeat. Drinking will only inhibit your ability to monitor eating and can result in overeating Inadequate strength[edit] The third behavioural pattern of under regulation is inadequate strength or willpower to act against the power of an impulse. An impulse refers to a desire to perform a particular action such as eating a particular food on a particular occasion Baumeister et al, Stopping oneself from having a slice of cake at a birthday party is a difficult task and relies on a great deal of strength or will power. There are some recognised causes of faltering willpower including: Fatigue[edit] People are less effective at self-regulation when they are physically or mentally fatigued.

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4: The Role of Psychobiological and Neuroendocrine Mechanisms in Appetite Regulation and Obesity

The regulation of appetite and glycemia is likely the outcome of a complex interplay of multiple metabolic, hormonal, and neural signals that are not yet completely elucidated 10,12,

This is an open access article licensed under the terms of the Creative Commons Attribution Non-Commercial License http: This article has been cited by other articles in PMC. Abstract Obesity is a multifactorial disease. Among its causes are physical inactivity and overeating. For example, certain hormones including leptin, insulin and ghrelin, may influence appetite and consequently body weight. Obesity frequently co-exists with metabolic disorders including dyslipidemia, hypertension and insulin resistance, thus constituting the metabolic syndrome which is characterized by increased cardiovascular risk. Lack of comprehensive knowledge on obesity-related issues makes both prevention and treatment difficult. This review considers the psychobiological and neuroendocrine mechanisms of appetite and food intake. Whether these factors, in terms of obesity prevention and treatment, will prove to be relevant in clinical practice including reducing the cardiovas-cular risk associated with obesity remains to be established. Obesity, appetite, psychobiology, neuroendocrine mechanisms, leptin, insulin, ghrelin, cardiovascular risk. This process occurs along the gastrointestinal tract, starting with mechanical and chemical processes in the mouth [1 - 3]. Mastication fragments food and involves coordinated rhythmic activity of the neuromuscular system [1 , 3 - 5]. Mastication is important for the digestion of all foods and even more for fruits and raw vegetables. Peptide enzymes act primarily on the surfaces of food particles. How fast digestion is achieved depends on the total amount of food exposed to intestinal secretions [4]. A basic characteristic of individuals that masticate food too quickly is that they do not have a sense of satiety, thus leading to enhanced food intake which predisposes them to increased body weight [1 , 6 , 7]. Of particular interest is the separation and clarification of the concept of food intake and eating behaviour. These mechanisms seem to be influenced by body weight. The complexity of these processes makes it more difficult to understand the psychobiological interactions [10 , 11]. Several researchers believe that the primary disorder involves food intake mechanisms and metabolic disorders. The amount of food intake depends on the number, size and energy content of the meals [11 , 13 , 14]. Each meal can be considered as consisting of 3-phases: Initiation of Food Intake The biological mechanisms involved in the initiation of each meal include stomach contractions, food taste and energy levels. In particular, although for several years stomach contractions were regarded as the main stimulus, it seems that other mechanisms are involved as well such as psychosocial factors and dieting practices [2 , 5 , 11]. Food taste plays a key role on food preference and food choice because of the way that a human can perceive the food but also the sensory affective response to the taste, smell, sight and texture of food [16 , 17]. Food taste for most of the people translates into flavour, smell and oral perception of food texture [17]. Increased concentrations of NEFAs occur in response to lipolysis. So NEFAs are regarded as a biomarker of negative energy balance, if the supply of glucose is insufficient in order to cover energy demands. In obese individuals, NEFAs are constantly increased due to insulin resistance, thus providing a misleading signal of lack of food; as a result obese people eat more than they actually need [18]. Furthermore, there are theories which support the concept that glucose uptake and utilization play a major role in the regulation of hunger, satiety and energy balance. The glucostatic theory maintains that food consumption is activated because of a decrease in blood glucose [19 - 22]. The lipostatic theory specifies that body fat is the answer to regulate the feeding behaviour [22 , 23]. The aminostatic theory proposes that amino acids in the blood have a significant role in defining satiety [22]. The thermostatic theory claims that temperature that develops in specific sensors in the body, such as central thermoreceptors, could act as a food intake sensor for total energy balance [24]. The hepatostatic theory concentrated on the metabolic activity of the liver [25]. The ischymetric hypothesis suggests that the process of feeding can be regulated by our metabolic rate. When the body is in the process of food absorption, the metabolic rate is faster than in fasting [26 , 27]. It may well be that all these factors play a role with their contributions differing in individuals. Meal Duration Meal duration depends on both endogenous and exogenous mechanisms. Endogenous factors, including a variety of stimuli deriving from the

mouth, pharynx, esophagus and the stomach, may cause temporal extension of the meal [28 - 30]. After the food enters the intestine, absorption, an important function of the small intestine, begins. Satiation occurs when the inhibitory mechanisms dominate over stimulants. Interestingly, the hedonistic effect of food consumption may prolong feeding. The mesolimbic dopamine pathway may be responsible for this action; tasty foods may release dopamine into the nucleus accumbens [28 - 31]. End of Meal The digestion process initiates in the stomach, where pepsinogen is converted to pepsin by hydrochloric acid, and proceeds to the intestine via the pyloric sphincter. The nervous system and various digestive system hormones [e. The gastrointestinal system is equipped with specialised chemo- and mechano-receptors that monitor physiological activity and transfer information to the brain mainly via the vagus nerve [1 , 28]. Among the factors that influence this process during the meal are amino acid levels in the circulation amino acids are derived from both protein catabolism and their intestinal absorption. According to the aminostatic hypothesis, amino acids act as peripheral signals to the brain in order to maintain the long-term balance between energy intake and energy expenditure as well as body fat mass over days or weeks [24 , 32]. In this context, increased muscle catabolism and elevation of amino acids levels leads to feeding, whereas postprandial uptake of amino acids from the plasma into the muscles results in the cessation of feeding and a period of satiety [24 , 33]. The absorption rates of amino acids are highly dependent on their protein source. Several amino acids derive from the catabolism of soy protein and milk; however specific milk proteins such as beta-lactoglobulin and casein have different digestibility [24 , 33 - 35]. Whey has been found to reduce food intake at 90 min, whereas casein exerts a stronger effect later at min [33 , 36]. Furthermore, there is considerable evidence that the effect of whey proteins on satiety and food intake is mediated via the release of certain satiety hormones such as CCK, glucagon like peptide-1 GLP-1 , gastric inhibitory peptide GIP , peptide YY and ghrelin. Indeed, more than 20 different regulatory peptide hormones are released in the gastrointestinal system and many of them are involved in the regulation of food intake [36 , 37]. Furthermore, peripheral opioid and CCK-A receptors can be activated by casein ingestion; blocking these receptors with antagonists reduces their effect on food intake [36 - 39]. Dietary Choices Dietary preference is defined as an option when all kinds of food are available. Practically all types of food are never available at one time, therefore we choose the food we like best or the food that is easier to have at that time. Appetite is characterized by the preference of a particular food over another whose consumption may bring pleasure and taste. Dietary choices are determined by both biological genetic, hereditary factors and environmental factors. Scientific data indicate that appetite or distaste for certain kinds of food is incorporated in our genetic code. We are born with unknown predispositions for sweet or bitter tastes, sour or salty tastes [3 , 4 , 7 , 9 , 16 , 40]. Other factors responsible for dietary choices involve congenital conditions; a plurality of environmental factors determines our dietary preferences including imitation, social and emotional effects, physiological needs, industrial communication policies i. In obese people the situation is slightly different because the amount of food intake may be increased following its beneficial effect on emotional status i. Interestingly, there is a tendency towards the consumption of foods rich in carbohydrates whenever obese individuals are emotionally distressed [11 , 41 , 43]. It should also be noted that exogenous factors such as food taste, smell and hedonic effect may have a greater influence on obese people than endogenous factors such as hunger, energy needs and satiation [3 , 4 , 11 , 41]. Experimental data have indicated the presence of several peptides with their receptors in the hypothalamus and other parts of the CNS that may affect the quantity and quality of food intake [7 , 15 , 44 , 45]. These peptides act as sensors that transfer signals from the periphery and stimulate or inhibit appetite and food intake accordingly in order to maintain energy homeostasis; not only they regulate the amount of each meal but also long-term energy reserves i. The main hormones involved in this process are insulin and leptin [45 , 47]. It should be noted that abdominal fat is the one related to increased cardiovascular CVD risk in both genders, as reported in several meta-analyses [48 - 50]. Consequently, central obesity is included in all definitions of the metabolic syndrome [51 - 55] and should always be taken into consideration when assessing CVD risk. Furthermore, epicardial fat has been recently associated with coronary artery disease prevalence and severity [56 , 57]. There is also data that correlate epicardial fat thickness with leptin and ghrelin concentrations [58]. Leptin, Insulin and Obesity Leptin, one of the most important adipose-tissue

derived hormones, plays a major role in the regulation of energy intake and energy expenditure in terms of appetite and metabolism control [59 , 60]. Leptin is a amino acid protein primarily produced in white adipose tissue. Circulating leptin levels are directly proportional to the total amount of fat in the body. Leptin acts on specific hypothalamic receptors and inhibits appetite by counteracting the effects of the orexigenic neuropeptide Y NPY [47 , 61 - 63]. The absence of leptin or its receptor leads to uncontrolled food intake, resulting in obesity [65]. Insulin, a hormone composed of 51 amino acids, is produced by the islets of Langerhans in the pancreas; it can regulate carbohydrate and fat metabolism in the body via glucose utilization in the periphery e. In the absence of insulin, glucose uptake from peripheral tissues is inhibited; thus hyperglycemia occurs, leading to diabetes, and fat becomes the energy source of the organism via gluconeogenesis [65]. Similarly, in cases of insulin resistance, both obesity and diabetes may arise. Leptin and insulin were shown to proportionally increase the quantity of body fat after eating [64]. Furthermore, these hormones are able to influence meal duration, metabolic activity and energy intake for a prolonged period of time through interactions with the CNS [44 , 45 , 59]. Certain characteristics of leptin and insulin secretion and action explain why they are regarded as regulators of food intake and energy homeostasis [47 , 66 - 68]. For example, their levels in the circulation are proportional to adipose tissue mass, they penetrate the blood-brain barrier into the CNS at rates that depend on their plasma concentrations and specific receptors for leptin and insulin have been identified in neurons that control metabolic activity [64 , 66 , 69]. Furthermore, their administration directly into the CNS was reported to inhibit food intake in animal models [64 , 69]. Interestingly, both hyperinsulinemia and insulin resistance exist in obese individuals, probably as a balancing mechanism to inhibit further increases in body weight [59 , 62 , 70]. Leptin effects on food intake mechanisms seem to be stronger than those of insulin. It has been shown that the absence of leptin in the human body can cause severe obesity, even though insulin levels are high [65]. The interactions between leptin and insulin are complicated while their effects on the endocrine system differ [71 - 73]. Leptin acts as a negative signal for the brain and suppresses food consumption. On the other hand, insulin promotes glucose uptake from peripheral tissues in a rate proportional to serum leptin levels. Insulin secretion is adjusted in response to acute metabolic changes; insulin levels increase during meals or when glucose is elevated for another reason and decrease during stress and exercise [64 , 65 , 73]. Leptin is secreted from adipocytes in an amount proportional to the metabolic action of fat cells; thus plasma leptin levels are a reliable marker of body fat [47 , 74]. Low leptin levels indicate depleted fat stores and inhibit functions that require adequate energy stores e. Plasma insulin levels have a direct link to body weight and body adiposity; they also reflect acute changes in energy homeostasis [64 , 74]. Of note, insulin secretion reflects the amount of visceral white adipose tissue, whereas leptin secretion reflects total fat mass and especially subcutaneous fat mass [64 , 75]. Of note, in obesity, small quantities of food consumption may cause greater reductions in insulin and leptin levels compared with the expected in terms of the increased adipose tissue mass [64]. In response to body fat increase, leptin and insulin levels are also increased [64].

5: Selfish brain theory - Wikipedia

A list of Citizendium articles, and planned articles, about Glucostatic theory of appetite control. See also changes related to Glucostatic theory of appetite control, or pages that link to Glucostatic theory of appetite control or to this page or whose text contains "Glucostatic theory of appetite control".

6: What is GLUCOSTATIC THEORY? definition of GLUCOSTATIC THEORY (Psychology Dictionary)

Metabolism, Metabolic Rate, Nutrition. STUDY. What is the glucostatic theory of appetite control? What is the lipostatic theory? Hypothalamic control - Feeding center.

7: Motivation and emotion/Book//Self-regulation of overeating - Wikiversity

GLUCOSTATIC THEORY OF APPETITE CONTROL pdf

The glucostatic theory maintains that food consumption is activated because of a decrease in blood glucose []. The lipostatic theory specifies that body fat is the answer to regulate the feeding behaviour [22, 23].

8: The glucostatic theory of appetite control and the risk of obesity and diabetes.

Attributed to Keesey et al, this theory is considered a "long term" regulation theory go hunger that assumes that there is a "set-point" that defines the baseline weight of a person. The lipostatic theory thus describes the homeostatic process by which the body achieves this balance.

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