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Fat in human body is stored in

How fat is stored in body. The most prominent (major) type of fat stored in food and in the human body is.

loose connective tissue consisting mainly of adipocytes adipose redirection here. for the imaginary creature of the doctor who, see list of doctor who universe creatures and aliens (0 ât "9, adipose. see also: fat adipose tissue is one of the main types of connective tissue. Morphology of three different classes of adipocytes. Pronunciation / ë |déaëœpoêšs / (hearth) identifiedrimeshd000273fma20110 terminologynogia [edified on wikidata] adipose tissue composed mainly of adipocytes, adipose tissue connective tissue composed mainly of adipocytes, fibroblasts, vascular endothelial cells and a variety of immune cells such as the macrophages of adipose tissue is derived from preadipocytes. Its main role is to store energy in the form of lipids, although pillows and also isolates the body. far from being ormonally inert, adipose tissue has, in recent years has been recognized as an important endocrine organ, [2] while producing hormones such as leptin, estrogen, resistia and cytokin (in particular TNFα). the two types of adipose tissue are white adipose tissue seems to be partly controlled by the adipose tissue after the most specifically brown adipose tissue was first identified by the Swiss naturalist conrad gesner in 1551. [3] anatomical characteristics distribution of white adipose in the human body, is the adipose tissue: under the skin (subcutaneous fat,) around the internal organs (visceral fat,) in bone marrow (yellow bone marrow,) intermuscular (muscle system) and in the breast (mommy tissue.) the adipose tissue is found in specific places, which are indicated as adipose deposits. In addition to adipose tissue, there are other types of cells, collectively stromal vascular fraction (svf) of cells. svf includes methiocytes, fibroblasts, adipose tissue macrophages and endothelial cells. adipose tissue contains many small blood vessels. in the integumentary system, which includes the skin, accumulates in deeper level, the subcutaneous layer, providing insulation from heat and cold. around organs, provides protective padding. However, its main function is to be a reserve of lipids, which can be oxidized to meet the energy needs of the body and protect it from excess glucose by preserving triglycerides produced by the liver from sugars, although some evidence suggests that most fat synthesis from carbohydrates occurs in the adipose tissue itself. [4] The deposits of adiposes in different parts of the body have different biochemical profiles. under normal conditions, it provides feedback for hunger and diet to the brain. mice the obese mouse on the left has large shops of adipose tissue on the right mice have eight main adipose deposits, four of which are within the abdominal cavity. [1] coupled gonadal deposits are attached to the uterus and ovaries in females and epididymis and testicles in males; The coupled retroperitoneal deposits are located along the dorsal wall of the abdomen, surrounding the kidney, and, when massive, extend into the basin. the mesenteric deposit forms a web similar to the glue that supports the intestine and the spleen) and - when massive - extends into ventral abdomen. Both mesenteric deposits and Omental incorporates a very lymphoo fabric with lymph nodes and dumpling points, respectively. The two superficial deposits are the coupled inguinal deposits, which are found prior to the upper rear limb segment (under the skin) leather) subscapular deposits, which are located under the skin between the dorsal ridges of the scapulates. The layer of brown adipose tissue in this deposit is often covered by a "frosting" of white adipose fabric; Sometimes these two types of fat (brown and white) are difficult to distinguish. Inguinal deposits include pericardial, which surrounds the heart, and coupled poplite deposits, among the main muscles behind the knees, each containing a large lymph node. [5] Of all deposits in the mouse, gonadal deposits are the largest and most easily dissected, [6] which includes about 30% of the disable fat. [7] Obesity In an obese person, the excess adipose tissue that hangs down from the abdomen is indicated as a diaper. A diaper complicates the surgical intervention of the obese individual. It can remain as a literal "skin horn" if a severely obese person quickly loses large amounts of fat (a common result of gastric bypass surgery). Obesity is treated through exercise, diet and behavioral therapy. Reconstructive surgery is a method of treatment. [8] Visceral fat abdominal obesity in men ("beer belly") See also: Abdominal Obesity Visceral fat or abdominal fat[9] (also known as organ fat or intra-addominal fat) is located within the abdominal fat is different from subcutaneous fat under the skin, and interspersed intramuscular fat in skeletal muscles. Fat in the lower body, such as in the thighs and buttocks, is subcutaneous and is not constantly detached tissue, while fat in the abdomen is mostly visceral fat general fat general fat is composed of several fat general fat gene excess of visceral fat is known as central obesity, or "blind fat", in which the abdominal volume and abdominal fat. Excessive visceral fat is also related to type 2 diabetes,[12] insulin resistance,[13] inflammatory diseases,[14] and other obesity-related diseases. [15] Similarly, the accumulation of fat in the neck (or cervical adipose tissue) was shown to be associated with mortality. [16] Several studies have suggested that visceral fat can be predicted by simple anthropometric measures, [17] and predicts mortality more accurately than the body mass index or the circumference of life. [18] Men are more likely to have the fat stored in the abdomen due to differences in sex hormone. Female sex hormone causes fat to be stored in buttocks, thighs and thighs to life;[21] the next fat is stored in the abdomen. [10] Visceral fat can be caused by excess cortisol levels. [22] At least 10 MET-hours per week of aerobic exercise leads to reduced visceral fat in those without metabolic disorders. [23] Strength training and calorie restriction also reduce visceral fat, although their effect may not be cumulative. [24] Both physical exercise and hypocaloric diet cause visceral fat loss, but exercise has a greater effect on visceral fat than fatThe high intensity exercise will reduce the total body fat and the relationship between visceral adipose tissue to subcutaneous adipose tissue, suggesting a preferential mobilization for visceral grease over the subcutaneous fat. [28] Epicardial components have been observed in comparing EAT with subcutaneous fat, suggesting a specific effect of the storage of fatty acids stored on the function and metabolism of adipocy. [30] Subcutaneous Fat See also: Body Fat percentage Micro-Anatomy of Subcutaneous Fat Most of the remaining non-visceral fat is located just below the skin in a region called ipodermis.[31] This subcutaneous fat is not related to many of the pathologies related to classical obesity, such as heart disease, cancer and stroke, and some evidence also suggests that it could be protective. [32] The typically female (or gynecoid) model of the distribution of body fat around the hips, thighs and buttocks is subcutaneous fat, and therefore poses less than a health risk than visceral fat.[33][34] Like all other fatty organs, subcutaneous fat is an active part of the endocrine system, secreted leptin hormones and resistin. [31] The relationship between the subcutaneous fat is an active part of the endocrine system, secreted leptin hormones and resistin. [31] The relationship between the subcutaneous fat is an active part of the endocrine system, secreted leptin hormones and resistin. these equations was formed by Durnin and Wormersley, who rigorously tested many skinfold types, and consequently created two formulas to calculate the body density—as the sum of skin folds increases, the body density decreases. [35] Factors such as sex, age, population size or other variables can make equations invalid and unusable, and, starting from 2012[update], the equations of Durnin and Wormersley remain only estimates of a person's real fat level. New formulas are still being created. [35] Marrow's fat, also known as adipose tissue of marrow (MAT), is an unknown fat deposit that resides in the bone and is interspersed with hematopoietic cells as well as bone elements. Adipocytes in this deposit are derived from mesenchymal stem cells (MSC) that can give rise to fat cells, bone cells and other types of cells. The fact that MAT increases in the regulation of the caloric/anorexia restriction is a feature that distinguishes this deposit from other fat deposits. [36][37] The exercise regulates MAT, decreasing the amount of MAT and decreasing the size of the adipocytes of marrow. [39][41] The exercise regulation of marrow fat suggests that it brings a certain physiological resemblance to other white fat deposits. In addition, the increase of MAT in obesity further suggests a similarity with white fat deposits. [39] Ectopic fat is the preservation of triglycerides in tissues other than adipose tissue, which should contain only small amounts of fat, such as liver, skeletal muscle, heart and pancreas. [1] This can interfere with cell functions and therefore organ function and is associated with insulin resistance in type-2 diabetes. [42] It is stored in relatively high quantities around the organs of the abdominal cavity, but it should not be confused with visceral fat. The specific cause for the accumulation of genetic, environmental and behavioral factors that are involved in excess of energy intake and decreased physical activity. Substantial weight loss can reduce ectopic fat deposits in all organs and this is associated with an improvement of the function of that organ. [42] In the latter case, non-invasive weight loss interventions such as diet or exercise can decrease fat(particularly in the heart and liver) in obese or overweight children and adults. [43][44] Free fatty acids of physiology (FFA) are released from lipoproteins from lipoprotein from lipoproteins from lipoproteins from lipoproteins from lipoproteins from lipoproteins from lipoproteins from lipoprotein from lipoproteins from lipoprotein from lipoproteins fro a net internal flow of FFA, and only when the insulin is low can FFA leave the fat tissue. The secretion of insulin is stimulated by high blood sugar, which results from the consumption of carbohydrates. [46] In humans, lipolysis (hydrolysis of triglycerides to free fatty acids) is controlled by balanced control of lipolytic B-adrenergic receptors and intermediate antilipolysis of the a2A-adrenergic receptor. Fat cells play an important physiological role in maintaining the levels of triglycerides and free fatty acids, as well as determining insulin resistance. This largely explains why central obesity is a marker of impaired glucose tolerance and is an independent risk factor for cardiovascular disease (even in the absence of diabetes mellitus and hypertension). [47] Studies of female monkeys at Wake Forest University (2009) found that people suffering from higher stress have higher levels of visceral fat in their bodies. This suggests a possible cause and-effect connection between the two, where stress promotes the accumulation of visceral fat, which in turn causes hormonal and metabolic changes that contribute to heart disease and other health problems. [48] Recent advances in biotechnology have allowed the collection of adult stem cells from adipose tissue, allowing the stimulation of tissue regrowth using the patient's own cells. In addition, adipose-derived stem cells derived from both humans and animals reportedly can be efficiently reprogrammed into induced pluripotent stem cells without the need for feeding cells. [49] The use of a patient's own cells reduces the possibility of tissue rejection and avoids ethical problems associated with the use of human embryonic stem cells. [50] A growing body of evidence also suggests that different fat deposits (i.e. abdominal, omental, pericardial) produce adipose-derived stem cells with different characteristics include proliferation rate, immunophenotype, differentiation potential, gene expression, as well as sensitivity to hypoxic culture conditions. [52] Oxygen levels appear to play an important role on the metabolism and function of estradiol. [54] Adipose derived hormones include: Adiponectin Resistin Plasminogen activator inhibitor-1 (PAI-1) TNFα IL-6 Leptin Estradiol (E2) Adipose tissues also secrete a type of cytokines (cell-to-cell that signals proteins) called adipokines (adipokines such as adiponectin Resistin Plasminogen activator inhibitor-1 (PAI-1) TNFα IL-6 Leptin Estradiol (E2) Adipose tissues also secrete a type of cytokines (adipokines), which play a role in the complications of obesity -associated. Perivascular adipose tissue releases adipokines such as adiponectin Resistin Plasminogen activator inhibitor-1 (PAI-1) TNFα IL-6 Leptin Estradiol (E2) Adipose tissues also secrete a type of cytokines (adipokines), which play a role in the complications of obesity -associated. Perivascular adipose tissue releases adipokines (adipokines), which play a role in the complications of obesity -associated. which affects the contractile function of the surrounding vessels. [1][55] Fat Brown Fat Cell Main Article: Fat Brown Fat or Brown Fat Organization of the surrounding vessels. [1][55] Fat Organization of the surrounding vessels. [phosphorylation within the mitochondria through the specific expression of non-compacting protein 1 (UCP1). [56] BAT is mainly found around the neck and large blood vessels of the chest, where it can actually act in heat exchange. BAT is robustly activated to cold exposure by the release of catecholamines from sympathetic nerves which results in UCP1 activation. The activation of BAT can also occur in response to surplus. [57] UCP1 activity is stimulated by long chain fatty acids that are subsequently produced to the activation of the î²-adrenergic receptor. [58] UCP1 is proposed to function as a fatty acid proton symbol, even if the exact mechanism has yet to be hallucts. [58] On the contrary UCP1 is inhibited by ATP, ADP and GTP. [59] Attempts to simulate this pharmacological process so far have not been successful. success. Manipulating the differentiation of "brown fat" could become a mechanism for weight loss therapy in the future, encouraging fabric growth with this specialized metabolism without inducing other organs. A review on the possible therapeutic destination of Bruno fat to treat the human obesity was published by Samuelson and Vidal-Puig in 2020. [60] Until recently, the brown adipose tissue was designed to be mainly limited At newborns in humans, but the new tests have now turned upside down this belief. The metabolically active tissue with temperature reactions similar to the brown adipose ones has been reported for the first time in the reck and in the trunk of some human adults in 2007, [61] and the presence of brown adipose in human adults was subsequently verified Histologically in the same anatomical regions. [62] [63] [64 Beige Fat and Wat Browning Wat Browning, also indicated as Beiging, occurs when the adipocytes features are developed within WAT DEPOT. Beige adipocytes take on a multi-channuish appearance (containing different drops of lipids) and increase the expression of non-Accoupling 1 (UCP1) protein. [65] In this way, these adipocytes normally of energy storage become energy release adipocytes. The capacity to burn brown and beige fat calories was widely studied as research efforts focus on therapies aimed at treating obesity and diabetes. The 2.4-dinirlhenol drug also acts as a chemical accoupler similar to UCP1, was used for weight loss in the 1930s. However, it was quickly interrupted when excessive dosage has led to negative side effects, including hyperthermia and death. [65] Agonists Î²3, as CL316,243 were also developed and tested in humans. However, the use of these drugs has proved largely unsuccessful due to different challenges, including specifics of the species receptor and poor oral bioavailability. [66] Cold is a primary regulator of BAT processes and induces wat brunette. Brunette in response to chronic cold exposure was well documented and is a reversible process. A study in mice has shown that cold-induced browning can be completely reversed in 21 days, with measurable decreases in UCP1 seen within a period of 24 hours. [67] A study by Rosenwald et al. It revealed that when animals are re-exposed to a cold environment, the same adipocytes adopt beige phenotype, suggesting that beige adipocytes are maintained. [68] Transcription regulators, as well as an increasing number of other factors, adjust beige fat induction. Four transcription regulators are central to brunette wat and serve as goals for many of the molecules known to influence this process. [69] These include the Prior Domain of the Active Receptor Peroxis Gamma Proliferator (PParî³,) containing 16 (PRDM16), [70] the coactivator Range of the receptor peroxiser 1 alpha (PGC-1α,) and the factor B -CELL-2 (EBF2) [71] [73] [73] The list of molecules that constantly affect the acquired popularity has grown in direct proportion of the subject to the one that has grown in proportion of more grew Evolving is knowledge. Among these molecules there are irisin and fibroblasts growth factor 21 (FGF21,) that have been well studied and are considered important brunette regulators. Irisin is secreted by muscle in response to exercise and has been shown to increase brunette by acting on beige prees. [74] FGF21, a hormone secreted primarily by the liver, earned a great interest after being identified as a powerful glucose absorption stimulator and is thought to help in diet-induced obesity resistance [75] FGF21 can also be secreted in response to exercise and a low protein diet, even if this' Last was not carefully investigated. [76] [77 The data of these studies suggest that environmental factors such as diet and physical exercise can be important brunette mediators. In mice, it was discovered that aging can occur through the production of methionine-enkefalin peptides by type 2 2 Lymphoid cells in response to interleukin 33.[78] Genomic and bioinformatic tools to study browning Due to the complex nature of adipose tissue and a growing list of browning regulation molecules, there is great potential for the use of bioinformatic tools to improve study within this field. WAT's browning studies have greatly benefited from advances in these techniques, as beige fat is rapidly gaining popularity as a therapeutic goal for treating obesity and diabetes. DNA microarray is a bioinformatic tool used to quantify expression levels of various genes at the same time, and has been widely used in the study of adipose tissue. A study of this type used microarray analysis in combination with Ingenuity IPA software to examine changes in gene expression WAT and BAT when mice were exposed to temperatures of 28 and 6 °C. The most significantly high and low genes were then identified and used for the analysis of differently expressed paths. It has been discovered that many of the upregulated ways in WAT after cold exposure are also highly expressed in BAT, such as oxidative phosphorylation, fatty acid metabolism and pyruvate metabolism and pyruvate metabolism and pyruvate metabolism. [79] This suggests that some of the adipocytes passed to a beige phenotype at 6 °C. Mössenböck et al. also used microarray analysis to prove that insulin deficiency inhibits the differentiation of beige adipocytes but does not disturb their browning ability. [80] These two studies demonstrate the potential for the use of microarray in the study of WAT burn. RNA sequencing (RNA-Seq) is a powerful computational tool that allows quantification of RNA expression for all genes within a sample. By incorporating RNA-Seq into browning studies is of great value, as it offers better specificity, sensitivity and a more complete view of gene expression than other methods. RNA-Seq was used both in human studies and to identify potential therapeutic molecules that can induce beige phenotype. A study of this type used RNA-Seq to compare the gene expression profiles of WAT from wild type mice (WT) and those that overexpress the B-Cell Factor-2 (EBF2). The WAT of transgenic animals showed a brown fat gene program and had decreased the specific expression of WAT compared to WT mice. [81] Thus, EBF2 was identified as a potential therapeutic molecule to induce beiging. Chromatic immunoprocession with sequencing (ChIP-seq) is a method used to identify protein binding sites on DNA and evaluate histone changes. This tool allowed the epigenetic regulation examination of beige adipocytes. Studies observing the chromatic landscapes of beige adipocytes found that the adipogenesis of these cells derives from the formation of specific color landscapes of the cells, which regulate the transcriptional program and, finally, the differentiation of specific color landscapes of these cells derives from the formation of specific color landscapes of the cells, which regulate the transcriptional program and, finally, the differentiation of specific color landscapes of the cells, which regulate the transcriptional program and, finally, the differentiation of specific color landscapes of the cells, which regulate the transcriptional program and, finally, the differentiation of specific color landscapes of the cells, which regulate the transcriptional program and, finally, the differentiation of specific color landscapes of the cells, which regulate the transcriptional program and, finally, the differentiation of specific color landscapes of the cells, which regulate the transcriptional program and the cells are considered to the cells are considered to the cells are cells as a constant of the cells are considered to the cells are cells as a cell a transcriptional and epigenetic factors that influence the development of beige adipocytes. [81] Genetics main article: Genetics of obesity § Genes Genes The gene hypothesis (also called the hypothesis of famine) states that in some populations the body would be more efficient to maintain fat in times of abundance, thus guaranteeing greater starvation in times of food scarcity. This hypothesis, originally advanced in the context of glucose and resistance to insulin, it was discredited by physical anthropologists, physiologists, and the original supporter of the idea itself compared to that contexts. [82] [83] In 1995, Jeffrey Friedman, in the residence of him at the Rockefeller University, Rockefeller, With Rudolph Leibel, Douglas Coleman et al. discovered the protein leptina is produced in white adipose tissue and signs for hypothalamus. When leptin levels decrease, the body interprets this as a loss of energy and hunger increases. The rats without this protein eat until they are four times their normal size. Leptina, however, plays a different role in obesity induced by diet in rodents and humans. Because adipocytes produce leptin, leptin levels are elevated in obeses. However, hunger remains, and - when leptine levels decrease due to weight loss, hunger increases. The drop of leptina is seen better as a signal of hunger than the rise of leptina is seen better as a signal of satiety. [87] However, the high leptina in obesity are currently at the centre of obesity research. [88] Gene defects in the leptin gene (OB) are rare in human obesity. [89] Since July 2010 [Update], only 14 individuals of five families have been identified worldwide who carry a mutative gene of the ob (one of which was the first ever identified worldwide who carry a mutative gene of the ob (one of which was the first ever identified worldwide who carry a mutative gene of the ob (one of which was the first ever identified worldwide who carry a mutative gene of the ob (one of which was the first ever identified worldwide who carry a mutative gene of the ob (one of which was the first ever identified worldwide who carry a mutative gene of the ob (one of which was the first ever identified worldwide who carry a mutative gene of the ob (one of which was the first ever identified worldwide). United Kingdom, a family living in Turkey, one in Egypt, and one in Austria [90] [91] [92] [95] [96] Others have been identified as genetically partially lacking in leptin, and, in these individuals, leptin levels on the lower range of normal range can predict obesity. [97] Several mutations in genes involving melanocortins (used in brain signalling associated with appetite) and their receptors have been identified as causing obesity in a larger portion of the population than leptin mutations [98]. Physical Properties The fatty tissue will galley more easily than a person of the same weight with more muscular tissue, since the muscle tissue has a density of 1,06 g/ml. [100] Body fat meter See also: Analysis of bioelectric impedance A body fat meter is a tool used to measure body fat meter see also: Analysis of bioelectric impedance A body fat meter is a tool used to measure body fat meter see also: Analysis of bioelectric impedance A body fat meter see al relatively inexpensive body fat meter uses the principle of bioelectric impedance analysis (BIA) to determine the person to calculate an water was drunk before analysis. Before bioelectric impedance analysis machines were developed, there were many different ways in the analysis of body composition such as skin bending methods using calibers, underwater weighing, plethismography of displacement of the whole air (ADP) and DXA. Animal studies Inside the fabric(Adiposo) of CRC2 deficient mice, there is a greater number of eosinophils, greater activation of alternative macrofuses and a propensity towards the expression of type 2 cytokines. Furthermore, this effect was exaggerated when mice became obese from a high fat diet. [101] Gallery Diagramatic Section view of the skin (enlarged). White adipose fabric in paraffin percentage Cellulite Lipodistrophy Lipodistrophy Human fat used as a pharmaceutical in traditional medicine Obesity Starvation of obesity Classification of obesity Classification of childhood obesity EPODE International Network, the world's largest obesity prevention network World Fit A program of the U.S. Olympic Committee (USOC), and the United States Association of Olympics and Paralympics (USOP) Obesbration and walking Social Stigma of Obesity 22 (16): 2298-314. doi:10.1089/scd.2012.0647. PMC 3730538. PMID 23517218. "He laid the fabric as an endocrine organ. 89 (6): 2548-56. doi:10.1210/jc.2004-0395. PMID 15181022. Cannon B, Nedergaard J (August 2008). "Biology of development: neither fat nor meat". Nature. 454 (7207): 947-48. Bibcode:2008Natur.454..947C. doi:10.1038/454947a. SMED 18719573. 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