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# Introductory Chapter: Unbearable Burden of the Diseases - Obesity

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## 1. Obesity definition measurement and classification

Obesity and overweight are commonly accepted as immoderate fat accumulation in human body that increases the risk of almost every disease [1, 2]. Excessive fat accumulation is usually measured by body mass index (BMI), which is calculated with the weight and height proportions (weight relative to square of the height- $w/h^2$ ) [3]. A reliable measurement of the fat accumulation requires elaborated tool such as magnetic resonance imaging (bio-electrical impedance). Because of the Bio-electrical Impedance Analysis is not widely available, BMI is accepted population-based definition and classification tool of obesity and overweight. Twenty percent and above fat accumulation in human body is accepted as normal [3]. If the level exceeds this limit (according to standard age, height, and weight tables), it is defined as overweight and obesity [3, 4]. BMI is a commonly used indicator of obesity and it classifies as follows ( $\text{kg}/\text{m}^2$ ):  $<18.5$  for underweight,  $18.5\text{--}24.9$  for normal weight,  $25\text{--}29.9$  for overweight, and  $\geq 30$  for obese [4]. Sometimes, BMI shows normal limits even in the presence of abdominal (central) obesity. So the central obesity, which is a kind of more risky obesity, could be hidden [4, 5]. It has been reported that almost half of the children and adults with excess body fat are defined as nonobese according to BMI [6, 7]. Underestimates of obesity prevalence could lead to less attention to problems and inadequate prevention and combat. Bioelectrical impedance uses tetra-polar measurements by touch electrodes and the measurements include broad spectrum from visceral fat mass to subcutaneous fat mass and body fat over [8]. Thus, misinterpretation of obesity and overweight can be prevented. Maximum attention is required to correct the evaluation of adipose tissue dissemination and measurements. Health care professionals must be aware of hidden obesity.

## 2. Obesity pathogenesis

The most widely accepted opinion about obesity pathogenesis is that it is the result of balance mechanism between energy intake and expenditure. Recent investigation indicates that obesity pathogenesis is more complex than just an energy imbalance. The mechanism of energy intake and expenditure is a homeostatic process [9, 10–12]. This balance mechanism regulates body weight in “normal” limits. Earlier, obesity was thought just as an extreme calorie consumption than what the body needs. But researches and inventions in medicine show that the mechanism and etiology of obesity are not so simple. Reported studies showed that obesity pathogenesis is not only based on excess energy expenditure but also on the body's urge to fix and maintain the weight at an augmented value [9, 11, 13–15]. This process would explain why obesity does not respond to long-lasting diet and exercise program or why there is no permanent reduction in weight even if there is a response [12].

The main issue is to understand that why human body keep adipose tissue. Extreme fat accumulation actually is a biological defense mechanism of the adipose tissue [13]. Adipose tissue is a storage and endocrine organ needed for energy homeostasis. It is well known that the regulation of energy homeostasis depends on the adipose tissue. Recently, studies revealed that especially the visceral compartment of the adipose tissue not only simply deposits energy but also plays an active role in endocrine metabolism and immune system [12, 14, 16]. To comprehend pathogenesis of obesity needs to understand how adipose tissue works. Adipose tissue (composed of adipocytes-fat cells) has important roles in glucose homeostasis, immune responses, hormonal regulation, and reproductive functions. Fat cells (adipocytes) are derived from mesenchymal stem cells. Preadipocytes become adipocytes through the cell differentiation process called adipogenesis. [11, 13, 16]. It has been shown that adipocytes secrete biologically active molecules called adipocytokines. Some of these are tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), adiponectin, visfatin, omentin, cytokines, resistin, retinol-binding protein 4, and leptin. In case of excessive fat accumulation, pro-inflammatory cytokines such as tumor necrosis factor-alpha, interleukin (IL)-1 $\beta$ , and IL-6 are released. TNF- $\alpha$  is a main cytokine in the inflammatory response. It is well known that the serum TNF- $\alpha$  levels are increased in obesity and decreased in case of weight loss. Because of an active interaction of adipocytokines, obesity is regarded as a chronic inflammation in the human body. It is clearly known that the adipose tissue expands so that the circulating inflammation-related adipocytokines increase. Excessive fat accumulation continually increased oxidative stress (OS) [16–18]. OS leads to inflammatory reaction by triggering acute-phase response. Adipose tissue contains white and brown adipocytes. The white adipose tissue (WAT) is chiefly responsible for fat accumulation. WAT stores energy as triglycerides [11]. The brown adipose tissue (BAT) is an important energy source for the human body. It is responsible for the thermogenic activity. There is plenty of brown adipose tissue during infancy to adolescent surrounding the heart and large vessels. It has been shown that BAT decreases as humans mature. A mature human body has few brown adipose tissues as scattered cells within white fat pads. The brown adipocytes are multilocular and contain less lipid. [19]. Recently, it has been shown that the brown adipose tissue could be increased with the catecholamine discharge. To living in cold area increase brown adipose tissue. This specificity could be used effectively for combating obesity. Biologically, humans are prone to conserve body fat as a defense mechanism in case of starvation and famine. The theory of “thrifty gene” claimed that human genes were predisposed to accumulate adipose tissue for use in case of energy requirement [1, 11, 19]. This mechanism was a key factor for survival once. Over time, the problem of finding food disappeared. However, human biology and physiology failed to adapt to this quick change. Humans’ evolutionary biology remained slow in the face of fast transformation of environmental conditions (easily reachable abundant foods). Human genes which is predisposed to excessive weight gain pursued their tasks in present of the today’s world. Genes are not the only factors that lead to obesity. Excessive fat accumulation is a multifactorial disorder. First of all, it is a disorder of the energy homeostasis system [10–13]. Still, the fundamental reason that leads to obesity is energy imbalance between energy intake and expenditure. Examining all these factors comprehensively could provide an understanding of the pathogenesis of obesity. The main factors in the pathogenesis of obesity were claimed to be behavioral, biological, environmental, and molecular [1, 12]. All these factors contribute to fat accumulation on a different level. Generally, personal traits (binge eating-drinking, laziness, lack of will power, insufficient sleep, sedentary life style, and self-indulgence) were accused as behavioral factors that lead to excess weight gain [9, 10]. Biological factors include disability, gut microbiota, comorbidity, and prenatal, neural, and endocrine conditions

besides genetics. Environmental factors that lead to obesity are socioeconomic status, cultural reasons, and environmental pollutions. On a molecular level, the pathogenesis of obesity is based on the impairment of electron transport chain. Low education and socioeconomic level, family lifestyle, eating habits, and inactivity play important roles in obesity [1–3, 9, 10]. When the interaction of biological factors with behavioral and/or environmental factors is abundant, excessive fat accumulation is inevitable. Until now, the pathogenesis of obesity is not clarified entirely, but it is believed to be a disorder with multiple causes. The current opinion is that obesity is a multifactorial and heterogeneous disease that leads to adverse cellular and metabolic effects.

### **3. How obesity causes diseases**

It is well known that obesity and overweight are major risk factors for many acute and chronic diseases from metabolic and mental to cancer [20–23]. There is no doubt that obesity is a disease of its own, but it is also the main cause of various diseases in human body. The underlying mechanisms of obesity-related diseases are not well understood, but many evidences have pointed to cellular oxidative stress, following oxidative damage [16–18]. The expansion of the adipose tissue especially increases the visceral fat accumulation. Excessive energy mainly deposit in adipose tissue as triglyceride. It has been shown that visceral fat accumulation leads to many chronic diseases. Blood glucose and lipids are continually high in obesity. Increased levels of glucose and lipids in circulation lead to excessive energy substrates in adipose tissue, which increase the production of reactive oxygen species (ROS) [1, 12, 16–18]. It is well known that ROS is related to chronic inflammation in the human body. Excessive production of ROS can lead to cellular damage and cellular dysfunction by disrupting the structures of proteins, lipids, and nucleic acids. Obesity is a chronic systemic inflammation of the adipose tissue. Long-lasting obesity eventually activates the innate immune system in adipose tissue. Excessive fat accumulation continually increases oxidative stress (OS), which leads to inflammatory reaction by triggering acute-phase response. OS-activated immune cells generate free radicals. Adipokines also induce the production of reactive oxygen species in the same way. The inflammation of adipose tissue plays a critical role in obesity-related diseases. Intensive and permanent oxidative stress damages cellular structures. Additionally, in case of insufficient antioxidant capacity, obesity-related complications easily emerge [1, 12, 16–18]. Thus obesity led to the cardiovascular, gastrointestinal, genitourinary, metabolic/endocrine, musculoskeletal/orthopedic, neurological and central nervous, obstetric and perinatal, skin, psychological, respiratory/pulmonary, and reproductive systems [1–3]. Studies to understand the pathogenesis of obesity and obesity-related diseases are continuing rapidly. Fully comprehending the molecular mechanisms of obesity and obesity-related diseases would lead to the discovery of new therapies and preventive methods.

### **4. Obesity prevalence**

Obesity was not common until the twentieth century. The World Health Organization (WHO) formally accepted obesity as a global epidemic in 1997 [2]. Obesity prevalence steadily increased in the following years. The prevalence of obesity doubled in the last 40 years. Almost 50% of the adults are overweight and obese in many countries. WHO has reported obesity prevalence (2015) in some countries such as: Cook Island: 89%, Qatar: 42%, UAE: 37%, Saudi Arabia: 34%, Turkey: 29%,



Egypt: 28%, Australia: 28%, UK: 28%, France: 23%, Italy: 21%, Sweden: 20%, Germany: 20%, Brazil: 20%, and Japan: 3%. [2, 3, 24]. The worst is that obesity affects children all around the world. It has been reported that a quarter of children around the world are obese. The prevalence of obesity is especially high in industrialized countries, but it is also dramatically increasing in developing countries. Obesity prevalence is higher in low-income and low-educated people and the rate of obesity increases with age. Female gender is a risk factor for obesity especially in developing countries. [2, 3, 7, 24].

## **5. Combating obesity**

As the prevalence of obesity increases, the morbidity and mortality from obesity-related diseases (mainly cardiovascular diseases, diabetes, and various cancers) also increase [9, 24–26]. Obesity and obesity-related morbidities require careful clinical assessment. Obesity-related health problems also increase the treatment cost and lead to financial and labor loss in society [27]. As an effective strategy to combating obesity, population-based, preventive, and sustainable public health approaches are necessary. Specialized public health strategies for the risk groups such as children, adolescent, low-educated, and disabled people are also important to reduce and prevent obesity. Obesity is preventable and health care professionals have an important role in preventing it. Aim of the health care policies is to increase the life expectancy and more qualified life span for human beings all over the world. Healthy and supportive environments are indispensable to combat obesity [28, 29]. Education is the most important step for challenging obesity. The training program should be implemented at every opportunity to effect people's choices, by making basic healthy life-style choices (i.e., regular physical activity, healthier foods, etc.). Lifestyle factors and personal responsibility are efficient to some degree to decrease the prevalence of obesity. However, more effective measures are required to cope with an epidemic of obesity at the societal level. Parents must be enlightened about obesity to prevent childhood obesity. Obese children mostly become obese adults. Healthier food choices are provided in school. Obesity awareness must be increased by educative programs (such as school-based education about nutrition and dietary guidelines) [30, 31]. The fundamental issue is to produce healthier and unprocessed foods for the growing world population. Food marketing and pricing policies should be changed in favor of the people, not the industry.

## **6. Key points**

- Obesity could be defined as a multifactorial and heterogeneous disease.
- Obesity is a chronic systemic inflammation of the adipose tissue.
- Adipose tissue is a storage and endocrine organ needed for energy homeostasis.
- Fat accumulation actually is a biological defense mechanism of the adipose tissue.
- Excessive fat accumulation continually increases oxidative stress.
- The underlying mechanisms of obesity-related diseases are associated with the oxidative damage.


- The main factors in the pathogenesis of obesity are claimed to be genetic, biological, behavioral, environmental, and molecular.
- Obesity is a major risk factor for several diseases from metabolic and mental to cancer.
- Obesity reduces life expectancy and increases the risk of mortality.
- Almost 50% of adults are overweight and obese in many countries.
- A quarter of children around the world are obese and obese children mostly become obese adults.
- BMI is a commonly used indicator for obesity, but central obesity could be hidden.
- Obesity-related health problems also increase the treatment cost and lead to financial and labor loss in society.
- The prevalence of obesity is higher in low-income and low-educated people and the rate of obesity increases with age. Female gender is a risk factor for obesity especially in developing countries.
- Obesity is preventable. Population-based, preventive, and sustainable public health approaches are necessary to combat an epidemic of obesity.

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## References

- [1] Haslam DW, James WP. Obesity. *Lancet*. 2005;**366**:1197-1209. DOI: 10.1111/j.1467-789X.2009.00708
- [2] WHO. Global Strategy on Diet, Physical Activity and Health Solarex. [Internet]. 2015. Available from: <http://www.who.int/features/factfiles/obesity/en/> [Accessed: 12-11-2018]
- [3] World Health Organization Technical Report Series 854. Physical Status: The Use and Interpretation of Anthropometry. Geneva 1995. pp. 312-17. [Internet]. Available from: [http://www.euro.who.int/data/assets/pdf\\_file/0011/199532/Health2020-Long.pdf/0010/74746/E90711.pdf](http://www.euro.who.int/data/assets/pdf_file/0011/199532/Health2020-Long.pdf/0010/74746/E90711.pdf). [Accessed: 12-10-2018]
- [4] Croghan IT, Phelan SM, Bradley DP, Bauer KW, Seung-Young-Han, Bradley SM, et al. Needs assessment for weight management: The learning health system network experience. *Mayo Clinic Proceedings: Innovations, Quality & Outcomes*. 2018;**2**(4):324-335. DOI: 10.1016/j.mayocpiqo.2018.08.001
- [5] Barberio AM, Alareeki A, Viner B, Pader J, Vena JE, Arora P, et al. Central body fatness is a stronger predictor of cancer risk than overall body size. *Nature Communications*. 2019;**10**(1):383. DOI: 10.1038/s41467-018-08159-w
- [6] Javed A, Jumeau M, Murad MH. Diagnostic performance of body mass index to identify obesity as defined by body adiposity in children and adolescents: A systematic review and meta-analysis. *Pediatric Obesity*. 2015;**10**:234-244
- [7] Reilly JJ, Kelly J, Wilson DC. Accuracy of simple clinical and epidemiological definitions of childhood obesity: Systematic review and evidence appraisal. *Obesity Reviews*. 2010;**11**:645-655
- [8] Metcalf B, Rabkin RA, Rabkin JM, Metcalf LJ, Lehman-Becker LB. Weight loss composition: The effects of exercise following obesity surgery as measured by bioelectrical impedance analysis. *Obesity Surgery*. 2005;**15**(2):183-186
- [9] Mathus-Vliegen EMH, Basdevant A, Finer N, Hainer V, Hauner H, Micic D, et al. Prevalence, pathophysiology, health consequences and treatment options of obesity in the elderly: A guideline. *Obesity Facts*. 2012;**5**: 460-483. DOI: 10.1159/000341193
- [10] Wolin KY, Petrelli JM. Obesity. Santa Barbara, CA: Greenwood Press; 2009. ISBN 10: 0313352763/ISBN 13: 9780313352768
- [11] Rosen ED, Spiegelman BM. Adipocytes as regulators of energy balance and glucose homeostasis. *Nature*. 2006;**444**:847-853. DOI: 10.1038/nature05483
- [12] Trayhurn P. The biology of obesity. *The Proceedings of the Nutrition Society*. 2005;**64**(1):31-38
- [13] Lefterova MI, Lazar MA. New developments in adipogenesis. *Trends in Endocrinology and Metabolism*. 2009;**20**:107-114. DOI: 10.1016/j.tem.2008.11.005
- [14] Blaszczak AM, Jalilvand A, Liu J, Wright VP, Suzo A, Needleman B, et al. Human visceral adipose tissue macrophages are not adequately defined by standard methods of characterization. *Journal Diabetes Research*. 2019:8124563. DOI: 10.1155/2019/8124563
- [15] Sharon MF. Obesity: Risk factors, complications, and strategies for sustainable longterm weight management. *Journal of the American Association of Nurse Practitioners*. 2017;**29**(Suppl 1):3-14

- [16] Marseglia L, Manti S, D'Angelo G, Nicotera A, Parisi E, Di Rosa G, et al. Oxidative stress in obesity: A critical component in human diseases. *International Journal of Molecular Sciences*. 2015;**16**(1):378-400
- [17] Kern L, Mittenbühler MJ, Vesting AJ, Ostermann AL, Wunderlich CM, Wunderlich FT. Obesity-induced TNF $\alpha$  and IL-6 signaling: The missing link between obesity and inflammation-driven liver and colorectal cancers. *Cancers (Basel)*. 2018;**11**(1). pii: E24. DOI: 10.3390/cancers11010024
- [18] Furukawa S, Fujita T, Shimabukuro M, Iwaki M, Yamada Y, Nakajima Y, et al. Increased oxidative stress in obesity and its impact on metabolic syndrome. *The Journal of Clinical Investigation*. 2004;**114**(12):1752-1761. DOI: 10.1172/JCI200421625
- [19] Sellayah D. The impact of early human migration on Brown adipose Tissue Evolution and its relevance to the modern obesity pandemic. *Journal of the Endocrine Society*. 2018;**3**(2): 372-386. DOI: 10.1210/js.2018-00363
- [20] Landsberg L, Aronne LJ, Beilin LJ, Burke V, Igel LI, Lloyd-Jones D, et al. Obesity related hypertension: Pathogenesis, cardiovascular risk, and treatment-a position paper of the Obesity Society and the American Society of Hypertension. *Obesity*. 2013;**(1)**:8-24. DOI: 10.1002/oby.20181
- [21] Çakmur H, Ardiç S. Investigation of obesity and depression in a sample of Turkish elderly adults. *Turkish Journal of Geriatrics*. 2015;**2**:115-122
- [22] Roberts DL, Dive C, Renehan AG. Biological mechanisms linking obesity and cancer risk: New perspectives. *Annual Review of Medicine*. 2010;**61**:301-316. DOI: 10.1146/annurev.med.080708.082713
- [23] Xu YXZ, Mishra S. Obesity-linked cancers: Current knowledge, challenges and limitations in mechanistic studies and rodent models. *Cancers (Basel)* 2018;**18**(12):10. pii: E523. DOI: 10.3390/cancers10120523
- [24] Berghöfer A, Pischon T, Reinhold T, Apovian CM, Sharma AM, Willich SN. Obesity prevalence from a European perspective: A systematic review. *BMC Public Health*. 2008;**8**:200. DOI: 10.1186/1471-2458-8-200
- [25] Roberto CA, Swinburn B, Hawkes C, Huang TT, Costa SA, Ashe M, et al. Patchy progress on obesity prevention: Emerging examples, entrenched barriers, and new thinking. *Lancet*. 2015;**385**(9985):2400-2409. DOI: 10.1016/S0140-6736(14)61744-X
- [26] Montgomery MK, De Nardo W, Watt MJ. Impact of lipotoxicity on tissue “cross talk” and metabolic regulation. *Physiology (Bethesda, Md.)*. 2019;**34**(2):134-149. DOI: 10.1152/physiol.00037.2018
- [27] Withrow DA, Alter D. The economic burden of obesity worldwide: A systematic review of the direct costs of obesity. *Obesity Reviews*. 2011;**12**:131-141. DOI: 10.1111/j.1467-789X.2009.00712
- [28] LeBlanc-Morales N. Culinary medicine: Patient education for therapeutic lifestyle changes. *Critical Care Nursing Clinics of North America*. 2019;**31**(1):109-123. DOI: 10.1016/j.cnc.2018.11.009. Epub 2018 Dec 24
- [29] Volger S, Rigassio Radler D, Rothpletz-Puglia P. Early childhood obesity prevention efforts through a life course health development perspective: A scoping review. *PLoS One*. 2018;**13**(12):e0209787. DOI: 10.1371/journal.pone.0209787
- [30] Bramante CT, Thornton RLJ, Bennett WL, Zhang A, Wilson RF, Bass EB, et al. Systematic review of natural experiments for childhood



obesity prevention and control.  
American Journal of Preventive  
Medicine. 2019;**56**(1):147-158. DOI:  
10.1016/j.amepre.2018.08.023

[31] Stoner L, Beets MW, Brazendale K,  
Moore JB, Weaver RG. Exercise dose  
and weight loss in adolescents with  
overweight-obesity: A meta-regression.  
Sports Medicine. 2019;**49**(1):83-94.  
DOI: 10.1007/s40279-018-01040-2

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