

UDC 577.23: 571.27:612.39
doi: 10.15330/jpnu.7.4.7-18

METABOLIC SYNDROME, OBESITY AND *DROSOPHILA*

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Abstract. Metabolic syndrome (MetS) is a cluster of metabolic disturbances increasing a risk of cardiovascular diseases and diabetes 2 type. The main features of MetS include atherogenic dyslipidemia, elevated blood pressure, insulin resistance and elevated glucose levels, a pro-thrombotic state, pro-oxidant and pro-inflammatory states. Excessive consumption of high caloric food and sedentary lifestyle followed by overweight and obesity, as well as aging and stresses are major contributing factors to the MetS development. MeS affects between 10 and 84% of adults depending on the used MetS criteria and increases significantly a risk of cardiovascular diseases, diabetes 2 type and kidney diseases. Patients with metabolic disorders like obesity, diabetes, cardiovascular, and liver disease may have a higher risk of infection of COVID-19 with significantly worse prognosis and outcomes in these patients. In recent years, the fruit fly, *Drosophila melanogaster*, has been actively used to study human metabolic disorders as a cost-effective and expedient model. *Drosophila* belongs to insects with full metamorphosis and its life cycle includes four developmental stages: embryo, larva, pupa, and adult flies. Each developmental stage has its own specific advantages and can be used to study metabolic homeostasis. Studies of metabolic disturbances in *Drosophila* and mammalian models along with humans have demonstrated that flies and small mammalian models have many similarities with humans in basic metabolic functions and share many molecular mechanisms which regulate these metabolic processes. In this paper, we describe the advantages and limitations of *Drosophila* models of metabolic syndrome and obesity in light of physiological and biochemical similarities and differences between insects and mammals.

Keywords: fruit fly, metabolic syndrome, triacylglycerides, fasting glucose, fat body.

Abbreviations: HDL, high density lipoproteins; LDL, low-density lipoproteins; MetS, metabolic syndrome; TAG, triacylglycerides; ROS, reactive oxygen species.

1. INTRODUCTION

Metabolic syndrome (MetS) is a group of medical conditions and symptoms, which together increase a risk of development of cardiovascular diseases and type 2 diabetes. In addition to the reduction of the quality of life of people, the MetS has a significant economic impact on public health expenditure due to the high morbidity rates generated because cardiovascular diseases are the leading cause of mortality worldwide [1, 2].

The pathogenesis and causes of MetS have been studied for over 100 years [3]. This dysmetabolic phenotype was first described by a Swedish physician Eskil Kylin in the 1920s. Kylin determined it as the cluster of signs such as hypertension, hyperglycemia, and gout [3, 4]. In 1947, Dr. Vague defined upper body adiposity as the obesity phenotype that was commonly associated with metabolic abnormalities related to cardiovascular diseases and type 2 diabetes [5]. The increased interest in MetS has been recorded in the last 30 years. During this period, several definitions were introduced to explain the combination of risk factors that increase the incidence of cardiovascular diseases. In particular, MetS was defined with such terms as syndrome X, dysmetabolic syndrome X, plurimetabolic syndrome, and insulin resistance syndrome. Despite the various labels, the term MetS is now used universally [3, 6]. At present time, MetS is defined by World Health Organization (WHO) as a pathologic multifaceted condition characterized by abdominal obesity, insulin resistance, hypertension, and hyperlipidemia [7]. Nonetheless, there is still debate regarding the etiology and pathogenesis of MetS, because a single mechanism underlying this dysmetabolic phenotype is unknown [8-10].

A number of studies support the idea that a complex of factors, which act synergistically contribute to the increasing rates of MetS incidence; these include biological, nutritional, genetic, behavioral, social, and psychological factors [3, 7, 11-13]. In the recent years, a tight link between MetS and obesity was established, but obesity is not always synonymous with MetS. Not all obese people develop MetS symptoms and not all patients with the syndrome suffer from obesity. For example, Asian Americans have higher rates of metabolic syndrome despite lower body mass index and lower prevalence of overweight/obesity as compared with non-Hispanic Whites having lower MetS incidence at higher body mass index [11]. According to this, it was established that there are so-called metabolically healthy obese individuals who have high level of insulin sensitivity and no hypertension and hyperlipidemia and other features of MetS [7].

Mammals, including mice and rats, are thought to be the best models to study obesity and related complications as they relate to human concerns. At the same time, mammalian models are very expensive and restricted by ethical constraints. Recent studies have demonstrated that invertebrate models like fruit flies can be utilized efficiently for research of obesity and associated metabolic complications. The fruit fly *Drosophila melanogaster* is a widely used model object in genetics, but the similarity of physiological and metabolic processes with humans, a short lifespan and ease of cultivation make it a convenient model to study metabolism and metabolic diseases [14-24]. A number of studies revealed that *Drosophila* possesses the conserved regulatory mechanisms involved in metabolic homeostasis and can develop human diseases phenotypes [14, 19, 20, 23-27]. In particular, obese flies accumulate storage lipids (triacylglycerides) in the fat body, an organ with functions of mammalian adipose tissue, and exhibit metabolic complications including redox disturbance, hyperglycemia, and heart dysfunction similarly as it is observed in obese humans.

In this paper, we describe current knowledge on MetS pathogenesis, advantages and limitations of *D. melanogaster* as a model for studying obesity, MetS and related metabolic disorders.

2. EPIDEMIOLOGY, CAUSES AND COMPLICATIONS OF METS

MetS is recognized a disorder, which include such clinical signs as dyslipidemia, hyperglycemia and hypertension, insulin resistance, and increased incidence of oxidative stress [6, 8, 9, 10, 28]. The reported prevalence of MetS differs depending on age, sex, socioeconomic status, the ethnic background of study cohorts, as well as the diagnostic criteria used [7, 10, 29, 30]. According to International Diabetes Federation diagnostic criteria, European MetS prevalence was established as 41% in men and 38% in women [29]. In the United States, survey data of National Health and Nutrition Examination reported that 35% of adults, and as much as 50% of the over-60 population, had a diagnosis of MetS (30.3% in men and 35.6% in women) in 2003-2012 [31]. Mexican American women was reported to have the highest MetS incidence [32].

Overeating and low physical activity are the main causes for world growth of obesity and MetS. We obtain energy from proteins, lipids, and carbohydrates, which are the main fuel macromolecules in the

food that we consume. When we consume more food than we need, excess of nutrients may be converted to reserve carbohydrate or fats for use later under adverse conditions such as starvation, infection, or stress [18]. However, chronic overeating, especially predominant consumption of easily digestible carbohydrate-rich food, combined with a sedentary lifestyle, results in intensive accumulation of storage fats in both adults and children, that can lead finally to overweighting and obesity. Importantly, obese children and adolescents have a high probability of remaining obese as adults [12, 30, 33-35].

Genetic prerequisites, disrupted sleep, chronic emotional stress and other stresses like xenobiotic exposure, unbalanced food, alterations in gut microbiota composition, and getting older are among other important risk factors involved in MetS development and progression and are deeply described elsewhere [2, 6, 9, 12, 30, 35-37]. In addition, a number of studies suggest that parental obesity can lead to fetal metabolic reprogramming and to development of obesity and metabolic dysfunction in the offspring [33, 34, 38, 39]. Newborns from obese fathers had altered DNA methylation overall and significant hypomethylation at the insulin-like growth factor 2 (IGF2) gene. High maternal pre-pregnancy body mass index also led to altered DNA methylation levels in offspring [38]. This indicates the involvement of epigenetic mechanisms in the MetS development; and this issue is actively studied now [30, 40].

Individuals with the MetS are at least twice the risk for cardiovascular diseases compared with the healthy ones. In addition, MetS raises the risk for type 2 diabetes by about 5-fold. Although some investigators favor keeping risk factors separate for purposes of clinical management, others suspect that identification of persons with an aggregation of risk factors can provide useful information for guiding clinical management [41].

Metabolic syndrome is a serious health problem that affects between 10 and 84% of adults depending on the used MetS criteria and increases significantly a risk of cardiovascular diseases, especially atherosclerosis, coronary heart diseases, and heart attack. Due to the increased blood glucose levels, persons with MetS are at higher risk of diabetes 2 type and kidney diseases [1, 7, 42]. Metabolic syndrome is a cluster of metabolic disorders. When a patient has these conditions together, the chances for future cardiovascular disease is greater than any one factor having alone. For example, high blood pressure alone is a serious problem, but when a patient has high blood pressure along with high fasting glucose levels and abdominal obesity, this patient may be diagnosed with MetS. In this case, it is a higher possibility that this patient will have cardiovascular problems because of the combination of risk factors [1]. Recent data on viral pandemic caused by Sars-Cov-2 virus indicate that MetS is a risk factor that influences COVID-19 progression and prognosis. The prevalence of obese, diabetic, hypertensive or liver damage patients with severe cases of COVID-19, in multiple countries, demonstrates the importance of the care with this risk group, in prophylaxis, monitoring and treatment [43].

3. CRITERIA OF METS IN HUMANS

Several attempts have been made to determine the clinical criteria for the syndrome. To date, the consensus criteria presented in Table 1 are the most commonly accepted for diagnostic of MetS. They include abdominal or central obesity, elevated levels of serum triacylglycerides (TAG) and glucose, increased blood pressure, and low levels of high-density lipoproteins (HDL, or so-called "good" cholesterol). When patients have three or more of these signs, they are diagnosed with metabolic syndrome [6, 7]. Metabolic syndrome occurs when a person has three or more of the listed measurements. Additional signs of metabolic syndrome include high levels of low-density lipoproteins (LDL, or so-called "bad" cholesterol), impaired fasting glucose, insulin resistance, fatty liver, hyperuricemia, pro-inflammatory state, and oxidative stress development [1, 28, 44]. The WHO definition of the metabolic syndrome, in contrast to the NCEP ATP 3 criteria, is more etiologically oriented and requires insulin resistance and/or impaired glucose tolerance as a prerequisite criterion, along with two of the following: obesity; hypertriglyceridemia; low HDL-cholesterol; hypertension; or microalbuminuria [1].

4. MECHANISMS OF METS: OVERVIEW

Obesity, especially abdominal obesity, is considered as a major underlying abnormality frequently observed in the MetS. There is still debate about causal relationship between obesity and MetS: whether obesity causes MetS or the syndrome causes obesity. Nevertheless, one can be stated clearly, that obesity is a factor provoking metabolic disturbances. Many studies suggest that excessive fat accumulation, especially of visceral fat, increases the risk of MetS and its various complications, including dyslipidemia, hypertension, type 2 diabetes, atherosclerosis, and cancer [28, 44-46].

Parameters	Values of the parameters		
	WHO 1999	NCEP (National Cholesterol Education Program) ATP3 2005	IDF (International Diabetes Federation) 2006
Abdominal obesity	Waist/hip ratio > 0.9 (men) or > 0.85 (women) or BMI >30 kg/m ²	waist > 102cm (men) or > 88cm (women)	waist > 94 cm (men) or > 80 cm (women)
Elevated blood TAG	> 1.7 mmol/l (150 mg/dl)	> 1.7 mmol/l (150 mg/dl) or drug treatment for elevated triglycerides	> 1.7 mmol/l (150 mg/dl) or drug treatment for elevated triglycerides
Elevated blood pressure	> 140/90 mmHg	> 130/85 mmHg or drug treatment for hypertension	> 130/85 mmHg or drug treatment for hypertension
Evaluated fasting glucose	> 6.1 mmol/l (110 mg/dl), 2 h glucose > 7.8 mmol (140 mg/dl)	> 5.6 mmol/l (100 mg/dl) or drug treatment for elevated blood glucose	> 5.6 mmol/l (100 mg/dl) or diagnosed diabetes
Reduced HDL cholesterol (HDL-C)	< 0.9 mmol/l (35 mg/dl) in men, < 1.0 mmol/l (40 mg/dl) in women	< 1.0 mmol/l (40 mg/dl) in men, < 1.3 mmol/l (50 mg/dl) in women or drug treatment for low HDL-C	< 1.0 mmol/l (40 mg/dl) in men, < 1.3 mmol/l (50 mg/dl) in women or drug treatment for low HDL-C

Tab. 1. Clinical criteria for diagnostic of MetS, modified from [7].

Adipose tissue is the main depot for storage fats; it synthesizes and stores fats in the form of TAG. In addition to accumulation of energy reserves, adipose tissue functions as an important endocrine organ, which consists of adipocytes and many immune cells, and produces a variety of compounds involved in immune reactions and inflammation. The compounds include adipokines, chemokines, and pro-inflammatory cytokines [13, 47-49]. Upon the accumulation of excessive fat, the adipocyte size/number is increased and the profile of adipokine secretion (increase in leptin and decrease in adiponectin) is altered, leading to a low-grade chronic systemic inflammation in adipose tissue [48-52]. In addition, adipocytes secrete various chemokines such as monocyte chemoattractant protein (MCP-1) leading to the recruitment of pro-inflammatory M1 macrophages into adipose tissue. Enlarged adipocytes and infiltrated immune cells increase the production of pro-inflammatory cytokines (tumor necrosis factor alpha (TNF- α), plasminogen activator inhibitor (PAI-1), interleukins 1 β (IL-1 β), and 6 (IL-6)) and chemokines, resulting in systemic inflammatory status [52]. Inflammation increases production of reactive oxygen species (ROS) by the immune cells as a part of the immune response [15, 44]. Since ROS are highly reactive compounds, increase in their production leads to oxidative stress development with damaging effects on cellular constituents. Moreover, oxidative stress may also promote further production of pro-inflammatory cytokines, which, in turn, aggravate ROS production

in a “vicious cycle” [13, 15, 28, 51]. Thus, chronic oxidative stress and chronic inflammation should be considered as two main interconnecting players in obesity-related metabolic complications.

In visceral adipose tissue, the Toll-like receptors (TLRs) present in plasma membrane of adipocytes can be involved in the induction of inflammatory responses [53]. Normally, bacterial lipoproteins and lipopolysaccharides are main activators of these TLRs (TLR2 and TLR4 [54]). It was shown that TLRs receptors might be activated by specific types of lipids, in particular by saturated fatty acids, whereas unsaturated fatty acids was found to inhibit TLR-mediated signaling [55]. Enlarged adipocytes able to release free fatty acids with increasing their levels in the blood and adipose tissue microenvironment. Therefore, blood free fatty acids can contribute to a low-grade inflammation in adipose tissue. The activation of TLRs triggers a signaling cascade leading to translocation of nuclear factor κ B (NF- κ B) to the nucleus. NF- κ B protein is a main transcriptional regulator of adaptive immune response with triggers a synthesis and release by adipose tissue of several pro-inflammatory cytokines, chemokines, and adhesion molecules [28, 52].

Besides hyperlipidemia, high glucose levels can also activate inflammatory pathways. Hyperglycemia may lead to the non-enzymatic glycation of proteins and lipids with formation of advanced glycation end products, which stimulate the activation of the pattern recognition receptor RAGE (receptor for advanced glycation endproducts), and elicit an immune response through the activation of NF- κ B [50, 56]. In obesity-related pro-inflammatory states, the increased size of adipocytes plays a decisive role, because it aggravates the production of adipocytokines followed by triggering a number of inflammation-related pathophysiological processes [52]. The pro-inflammatory mediators aggravate macrophage recruitment and infiltration in adipose tissue. Macrophages increase release of ROS as a part of the immune response; therefore, their functioning leads to overproduction of ROS in adipose tissue resulting in oxidative stress. In turn, oxidative stress causes further increase in pro-inflammatory cytokine synthesis and macrophage infiltration leading inflammation to become chronic [28, 54, 57]. Circulating inflammatory biomarkers including C-reactive protein, fibrinogen, serum amyloid A, cytokines, and chemokines have also been linked to the pathogenesis of MetS. It was identified the significance of C-reactive protein levels in predicting future complications of MetS such as atherosclerotic cardiovascular disease. Mast cells in subcutaneous adipose tissue promote both inflammation and fibrosis. Thus, both adipose tissue and macrophage activity define MetS as an inflammatory disorder [58].

5. DROSOPHILA AS OBESITY MODEL

The fruit fly *D. melanogaster* is an especially useful model for studying obesity and metabolic diseases for a number of reasons. First, flies contain tissues, organs and systems that are analogous to all those involved in human obesity and associated metabolic diseases. In addition, *Drosophila* develop obesity and its associated complications during overconsumption of high caloric food, similar to humans. Furthermore, most genes known to function in metabolic diseases are conserved between flies and humans [ref. from 22]. As in mammalian models, two approaches are developed to study human metabolic disorders in *Drosophila*. One is the genetic approach based on manipulation with genes whose products are involved in nutrient sensing and regulation of metabolism [26, 59-65]. The other is manipulations of diet composition by altering amounts of sugars, fats, or proteins in diet [19, 20, 25, 39, 66-70]. Both approaches allow development of fly phenotypes that display features of human obesity and related metabolic complications (Table 2).

The main advantages of *Drosophila* as a model are high rate of reproduction, short life cycle, short lifespan, and the easiness and low cost of maintenance of flies in the laboratory. Moreover, the composition of fly media can be easily manipulated, that allow researchers to monitor changes in development, behavior and metabolism at different developmental stages [15, 19, 20, 69].

Drosophila belongs to insects with full metamorphosis and its life cycle includes four developmental stages: embryo, larva, pupa, and adult flies. At room temperature, one pair of mating adult flies can

generate hundreds of offspring within ~10-12 days. Each developmental stage has its own specific advantages and can be used to study metabolic homeostasis [26]. Larval and adult stages are used the most frequently in metabolic research [14, 19, 20, 22].

Insect digestive and neuroendocrine systems have many similarities with those in higher vertebrates. In *Drosophila*, food is digested and absorbed in the crop and midgut, which are analogous to human stomach and intestine, respectively. The key metabolic regulating organs in flies are fat body (functions as white fat tissue and liver), oenocytes (functions as hepatocyte-like cells), Malpighian tubules (functions as kidneys), and pars intercerebralis–corpora cardiac system (functions as the hypothalamus–pituitary system) [22, 26, 59]. Details on the functions and regulation of *Drosophila* organs are available in several recent reviews [15, 16, 17, 18, 22 26].

Parameters	Genetic approaches: up-regulation of genes boosting obesity and down-regulation of genes preventing obesity [26, 59; 60, 61, 62, 63, 65]	Diet-induced obesity: high amounts of carbohydrates, protein or fats in the diets [19, 20, 22, 25, 39, 66-70]
Fat accumulation	Increase	Increase
Body weight	Increase	Increase
Glucose	Not determined	Increase
Insulin resistance	Not determined	Increase
Climbing activity based on negative geotaxis	No changes	Decrease
Heart dysfunction	Not determined	Yes
Food intake	Increase	Increase
Lifespan	Shortening	Shortening

Tab. 2. Characteristics of diet and genetic-induced obesity in *D. melanogaster* models, modified from [26].

The biochemical pathways, which involved in regulation of carbohydrate and fat storage, are also very similar to those in humans. In particular, *Drosophila* fat body, which combines functions of adipose and liver tissues, accumulate excess metabolic fuels in the form of storage lipids (TAG) and carbohydrates (glycogen). The accumulation of energy reserves in fly body is regulated by neurosecretory hormones, especially insulin-like peptides and adipokinetic hormone (AKH). They are analogs of human insulin and glucagon, respectively [18, 21]. In flies, insulin-like peptides are produced by insulin producing cells in the median neurosecretory region of the fly brain, which are functional homologues of human pancreatic β -cells. Corpora cardiaca cells located in the ring gland neuroendocrine organ secrete AKH and functions as α -cells of the mammalian pancreas [18, 21, 22]. The fly pars intercerebralis-corpora cardiaca system receives information on the internal metabolic status and coordinates the physiological and behavioral activities of various peripheral organs. Thus, *Drosophila* can be used to investigate various aspects of energy balance including feeding control, food perception, energy expenditure, and metabolism of lipids and carbohydrates [26].

6. CRITERIA OF METS IN *DROSOPHILA*

As it was described above, a defined set of clinical criteria is established to diagnose human patients with MetS [6, 7]. What criteria can be used for a diagnosis of MetS in *Drosophila*? Not all parameters which are used to establish MetS in humans, can be applied to fruit flies (Table 3). This is

due to the peculiarities of the external and internal structure of *Drosophila* body as well as the peculiarities of metabolic and physiological processes in flies as cold-blooded animals with an open circulatory system. For humans, there are already established exact values of clinical parameters that allow distinguishing a healthy person from sick ones. Unfortunately, for *Drosophila*, there are no such indicators developed to date. Conducting experiments, researchers are guided only by the concepts of the control group and experimental one.

Human criteria	Using in <i>Drosophila</i>	Explanation
High waist circumference	No	Exoskeleton (cuticle), which does not allow changing significantly form of the body. Insects have three body parts (head, thorax and abdomen). In humans, adipose tissue is predominantly accumulated around waist in men and around waist and hips in females. In <i>Drosophila</i> , a storage fat is accumulated in the fat body which is located on dorsal side of the insect body and extends through the whole body - from thorax to abdomen [26]
High blood pressure	No	Insects have open circulatory system, no veins and arteries, no blood pressure in hemolymph
High blood TAG	No	The transport form of lipids in <i>Drosophila</i> are diacylglycerols [71]
Low blood HDL	Yes	<i>Drosophila</i> contains specific lipoproteins, which are used for transport of lipids. These lipoproteins are divided into 3 groups based on their density and contain phospholipids, specific apoproteins and diacylglycerols mostly; minor amounts are sterols and triglycerides. More than 95% of hemolymph lipids form fraction with lipoproteins of low density [71]
High blood glucose	Yes	Hemolymph contains free glucose and trehalose

Tab. 3. Criteria of MetS in *D. melanogaster* models.

However, in many cases, it is difficult to clearly predict that the control group is the healthy group with normal parameters. In addition, it is difficult to establish whether the parameters of the experimental fly group were beyond the healthy parameters or maybe they were in the range of the norm. In addition to measuring hemolymph glucose and TAG levels, assessment of postprandial glycaemic response on consumption of food with different carbohydrates (glucose, fructose, or sucrose) was proposed as a marker of metabolic disturbances in flies [19]. Insect postprandial glycaemic response is based on measurement of time-course of glucose levels in hemolymph in staved flies after administration with high carbohydrate food (20% carbohydrates). This test is equivalent to glucose tolerance test in humans [19] and have perspectives to be used as additional marker of insulin resistance in *Drosophila*.

7. CONCLUSION

Metabolic syndrome along with obesity becomes an increasingly common cause of cardiovascular diseases and mortality worldwide and is associated with many risk factors as well as numerous pathophysiologic mechanisms. The most described MetS mechanisms lead to development of insulin resistance, a low-grade inflammation, and oxidative stress. In recent years, *D. melanogaster* has become used as a model to contribute to pathogenesis research to identify more novel players underlying

energy homeostasis, as well as also provide a useful *in vivo* model to develop effective therapeutics. Although *Drosophila* model provides rapid results *in vivo*, it is still important to combine mammalian models and human studies to understand fully obesity and its co-morbidities and to combat eventually them. To confirm the obese state in flies is somewhat difficult due to high variability in values of the tested parameters. In many cases, we can just say about the direction of changes but not on a true obesity. Furthermore, the variability in values of certain parameters can be connected with the absence of standardization. Postprandial response to glucose in hemolymph may be used as a marker of MetS in *Drosophila* model but these procedures need also standardization.

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Received: 10.11.2020; **revised:** 25.12.2020.

Байляк Марія. Метаболічний синдром, ожиріння і *Drosophila*. Журнал Прикарпатського університету імені Василя Стефаника, 7 (4) (2020), 7–18.

Метаболічний синдром – це сукупність метаболічних порушень, які підвищують ризик розвитку серцево-судинних захворювань та діабету 2 типу. Основні складові метаболічного синдрому – атерогенна дисліпідемія, підвищений артеріальний тиск, резистентність до інсуліну та підвищений рівень глюкози, протромботичний стан, прооксидантний та прозапальний стани. Надмірне споживання висококалорійної їжі та малорухливий спосіб життя, що супроводжуються надмірною вагою та ожирінням, а також старіння та стреси є основними факторами, що сприяють розвитку метаболічного синдрому. На метаболічний синдром страждає від 10 до 84% дорослого населення залежно від використовуваних критеріїв для діагностики синдрому. Метаболічний синдром значно підвищує ризик серцево-судинних захворювань, діабету 2 типу та захворювань нирок. У пацієнтів із порушеннями обміну речовин, такими як ожиріння, цукровий діабет, серцево-судинні захворювання та захворювання печінки, підвищується ризик інфікування та ускладнень COVID-19, що значно погіршує прогноз та результати у цих пацієнтів. В останні роки плодова муха *Drosophila melanogaster* активно використовується для вивчення метаболічних порушень людини як економічно ефективна та доцільна модель. Дрозофіла належить до комах з повним метаморфозом і її життєвий цикл включає чотири стадії розвитку: ембріон, личинка, лялечка та доросла особина. Кожна стадія розвитку має свої специфічні переваги і може бути використана для вивчення метаболічного гомеостазу. Дослідження метаболічних порушень на дрозофілі та моделях ссавців продемонстрували, що мухи та моделі дрібних ссавців мають спільні риси з людьми за основними метаболічними функціями та мають спільні молекулярні механізми, що регулюють ці метаболічні процеси. У цій роботі ми описуємо переваги та обмеження дрозофіли як моделі метаболічного синдрому та ожиріння у світлі фізіологічних та біохімічних подібностей та відмінностей між комахами та ссавцями.

Ключові слова: плодова муха, метаболічний синдром, триацилгліцериди, глюкоза, жирове тіло.