DOI: https://doi.org/10.21323/2414-438X-2025-10-1-11-31
Received 01.11.2024
Accepted in revised 25.02.2025
Accepted for publication 03.03.2025
Available online at https://www.meatjournal.ru/jour
Review article
Open Access

FAST FOOD CONSUMPTION HAS A GREAT IMPACT ON THE AGING PROCESS — A REVIEW

Farhang H. Awlqadr[1], Ammar B. Altemimi[2, 3] *, Syamand Ahmed Qadir[4], Zina T. Alkanan[2], Aryan Mahmood Faraj[5], Qausar Hamed ALKaisy[6], Tarek Gamal Abedelmaksoud[7] *

[1] Food Science and Quality Control, Halabja Technical College, Sulaimani Polytechnic University, Sulaymaniyah, Iraq
[2] Department of Food Science, College of Agriculture, University of Basrah, Basrah, Iraq
[3] College of Medicine, University of Warith Al-Anbiyaa, Karbala, Iraq
[4] Medical Laboratory Techniques Department, Halabja Technical Institute, Research center, Sulaimani Polytechnic University, Sulaymaniyah, Iraq
[5] Medical Laboratory Science Department, Halabja Technical College of Applied Sciences, Sulaimani Polytechnic University Sulaymaniyah, Iraq
[6] Department of Dairy Science and Technology, College of Food Sciences, University of AL-Qasim Green, Al Qasim, Iraq
[7] Food Science Department, Faculty of Agriculture, Cairo University, Giza, Egypt

Keywords: *fast food, aging process, nutritional quality, metabolic diseases, obesity*

Abstract

This review examines the impact of fast food consumption on health and its role in the development of chronic diseases that accelerate the aging process. A comprehensive literature review was conducted to explore the relationship between fast food intake and the onset of cardiovascular diseases, diabetes, cancer, and obesity, all of which are associated with premature aging. The findings indicate that fast foods high in fat and sugar contribute to chronic inflammation — a phenomenon referred to as "inflammageing", which damages the vascular system and heightens the risk of cardiovascular diseases, including atherosclerosis and heart failure. Additionally, metabolic disorders such as insulin resistance and dyslipidemia disrupt glucose homeostasis, further exacerbating chronic inflammation and promoting accelerated aging. Moreover, fast food consumption is linked to an increased risk of cancer, largely due to the presence of carcinogenic compounds in processed meats and the obesity epidemic, which amplifies aging through mechanisms involving chronic inflammation, oxidative stress, and hormonal imbalances. Collectively, these factors impair immune function and elevate the risk of neurodegenerative diseases. Consequently, fast food consumption significantly contributes to premature aging by fostering chronic inflammation, metabolic disorders, and oxidative stress. Urgent public health interventions are necessary to mitigate these adverse effects and promote healthier dietary patterns, to enhance quality of life and longevity.

For citation: Awlqadr, F. H., Altemimi, A. B., Qadir, S. A., Alkanan, Z. T., Faraj, A. M., ALKaisy, Q. H. et al. (2025). Fast food consumption has a great impact on the aging process — A review. *Theory and Practice of Meat Processing*, 10(1), 11–31. https://doi.org/10.21323/2414-438X-2024-10-1-11-31

Introduction

These days fast food does not seem only as a dietary pattern in the USA but also as a diet used globally. Fast foods are convenient, affordable, and have the flavor most people like. However, they are energy-dense and fatty. Such foods have high levels of trans fats, sugars, and sodium, but contain low levels of essential nutrients, such as vitamins and minerals, as well as fiber. Thus, the fast food eating pattern has concerning public health outcomes, especially regarding the long-term effects of growing old. High calorie content, unhealthy trans fats from fast food, and insufficient amounts of essential nutrients may be a cause of many diseases. Nutrients are necessary for the body for proving energy, building and repairing tissues, regulating metabolism and maintaining homeostasis [1,2]. But excessive calorie intake can lead to obesity and other metabolic malfunctions that are key factors for the speed of aging among people [3].

The surge in obesity rates is the major consequence of fast food eating. Obesity is primarily a recognized risk factor for several chronic diseases, including CVD, diabetes type 2, and certain types of cancer. As a result, it tends to decrease life expectancy and speed up the aging process [4,5]. Moreover, the caloric content of overly consumed fast foods equals weight gain and obesity, leading to chronic inflammation, oxidative stress, and hormonal imbalances that promote accelerated aging [6,7]. The accumulation of bad fats and too much sodium in fast foods harms heart function [8].

Saturated and trans fats raise the level of LDL cholesterol, causing atherosclerosis, a hardening that may even advance to arterial narrowing. In turn, these are critical causes of heart attacks, strokes, and other important cardiovascular events that are leading causes of morbidity and mortality in older adults. These cardiovascular diseases are critical to the aging process because they impair the body's various organs and systems from functioning at total efficiency [9,10]. Moreover, fast food is highly associated with growing metabolic diseases, especially metabolic syndrome and diabetes type 2 [1,11]. Fast foods have a high glycemic load, leading to a quick elevation of blood sugar, and therefore insulin resistance increases

over time [12,13]. More important is the fact that insulin resistance is a determinant of type 2 diabetes and, thus, related to severe complications, such as kidney failure, neuropathy, and cardiovascular diseases — all of which serve to reduce life expectancy and accelerate aging [14]. In addition, fast food usually contains ingredients with pro-inflammatory and oxidative properties. Unhealthy fats and sugar-rich diets increase pro-inflammatory cytokines and reactive oxygen species (ROS) after cellular damage and chronic inflammation. Numerous age-related diseases, such as Parkinson and Alzheimer disease, among other neurodegenerative conditions, are associated with these factors [15,16]. In addition, other emerging reports indicate a negative association between fast food consumption, cognitive function, and mental health [17]. Unhealthy fat and high-sugar diets are associated with reduced memory, cognitive flexibility, and an increase in dementia risk. The neuroinflammatory and oxidative effects of such diets can accelerate cognitive decline, an essential aspect of the aging process [18]. In other words, the overall impact of fast food consumption is general enough to reach most aspects of the aging phenomenon, including both physical and cognitive health [19]. The purpose of this review is to underscore the tremendous impact that fast food has on public health and, further, on aging phenomena. We offer another piece of evidence to raise serious discussion on nutrition education for public intervention and policy changes to shift the balance toward healthy eating and increase quality of life and healthy aging.

Objects and methods

The sources of information were the following scientific databases: ScienceDirect, PubMed, Scopus, ResearchGate, and Google Scholar. The search strategy included the following keywords: fast food, aging process, nutritional quality, metabolic diseases, obesity. The following acceptance criteria for research characterization were considered: the role of fast food consumption in the development of aging processes. The parameters of the publications were as follows: publication from 1977 until 2024 (178 references were selected for this review); language: English. Exclusion criteria: no access to the full text articles. The published and selected research results were analyzed, systematized, summarized, after which conclusions were drawn by sections and a general conclusion.

Types of fast food

Modern diets include fast food, which is prepared and served rapidly in restaurants or for takeaway. These foods are heavy in energy and low in nutrients, frequently containing harmful fats, sugars, and sodium. This section discusses common fast foods, their nutritional value, and their appeal across demographics. The first type of fast food are burgers and sandwiches, which are popular worldwide because of their convenience, price, and taste. A burger often has a beef patty, buns, condiments, cheese, lettuce, and tomatoes. Chicken, fish, and vegetarian sandwiches are available. These foods are unhealthy due to their high caloric content, saturated fats, and sodium [20]. Due to their high fat and calorie content, such meals can raise obesity and cardiovascular disease risk [21]. The second type is fried chicken, which is popular among Asian and young adult population. Chicken chunks are battered and deep-fried for a crispy, delicious exterior. Fried chicken is tasty but heavy in trans fats and cholesterol, which can cause heart disease and other metabolic diseases. KFC's brand influence contributes to its widespread consumption [22]. Fried chicken is linked to obesity and poor lipid profiles, increasing cardiovascular disease risk [23]. In the West, the popularity of the third type of fast food, pizza, is huge. Teens and young adults eat it often because of its convenience and diversity of toppings [24]. A common pizza has a bread base, tomato sauce, cheese, pepperoni, vegetables, and other ingredients. Pizza has calcium and protein from cheese, but also saturated fats, refined carbs, and sodium. The high calorie and sodium content of pizza may contribute to obesity and hypertension [25]. The fourth type are soft drinks, commonly served with fast food, which drastically increase sugar intake. These drinks are high in added sugars and calories but low in nutrients. Sugary drinks can cause insulin resistance, diabetes type 2, and weight gain [26]. Regular consumption of these drinks can also cause tooth issues and metabolic syndrome [27]. The fifth type are French fries, which are a fast food staple commonly served with burgers and sandwiches. They contain harmful fats and sodium from deep-fried potatoes. Due to their high trans fat content, French fries can cause weight gain and cardiovascular illness [21]. The high glycemic index of potatoes causes blood sugar increases and insulin resistance [27]. Figure 1 shows fast food types and their impact on health.

Overview of fast food consumption

Fast-food consumption has become a pervasive aspect of modern diets worldwide, beginning from the mid-20th century and continuing to expand through 2024. Initially popularized in the United States by chains, such as McDonald's and Burger King in the 1950s, fast food quickly became entrenched in the diets of many people due to its convenience, affordability, and palatability. The trend gained momentum in the early 2000s, with significant growth in developing countries experiencing rapid urbanization and economic changes. For instance, the fast-food market in India was projected to be worth 27.57 billion dollars by 2020, highlighting its widespread popularity [28]. Recent studies show that fast food consumption remains high, with 36.5% of U.S. adults consuming fast food on any given day in 2017–2018, and over 55% of young adolescents in low- and middle-income countries (LMICs) consuming fast food at least once a week [29]. Younger individuals, particularly those under 30, and lower-income households are the most frequent consumers, driven by the affordability and accessibility of fast food. Fast food is characterized by high caloric content, unhealthy fats, sugars, and sodium, which contribute to various adverse health outcomes. Frequent

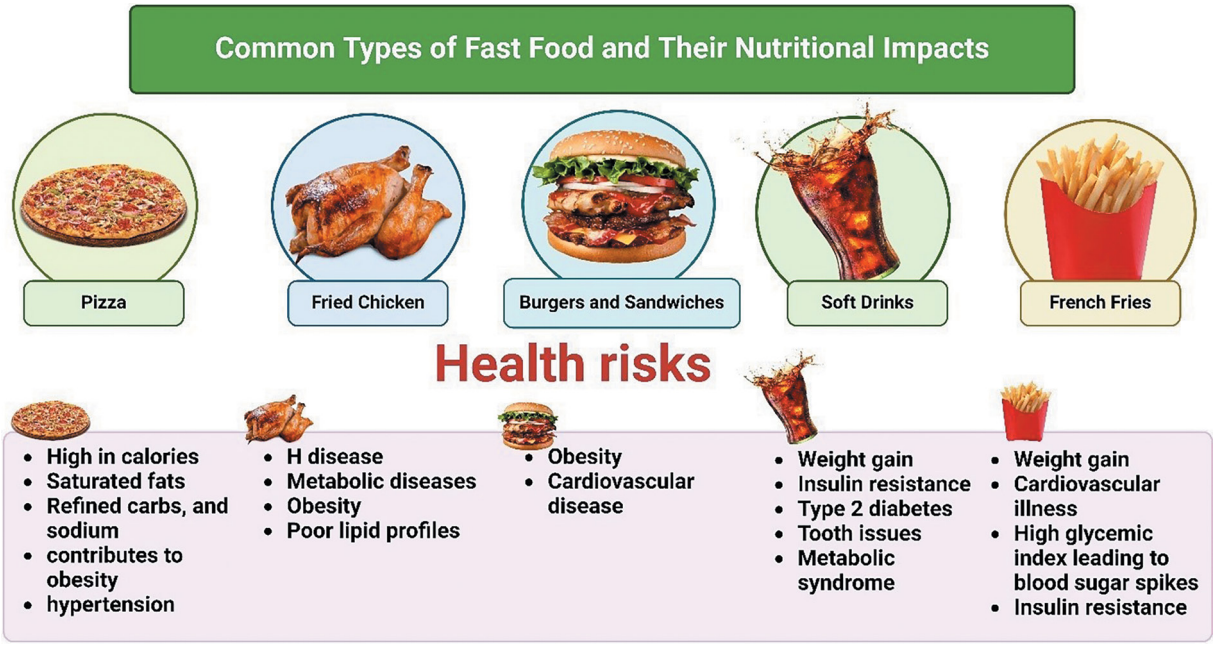

Figure 1. Common types of fast food and their associated health risks

consumption is associated with elevated calorie intake as well as a low-quality diet that contains higher amounts of total fat, saturated fat, sodium, and sugars, which are the essential factors in the onset of obesity and related metabolic disorders [30]. Research studies have continuously revealed that repetitive eating of fast foods can lead to a major increase in weight and insulin resistance. For instance, people who frequently eat fast food take up more kilograms and have more probability of developing insulin resistance as compared to those who do not consume it that often [31]. The level of obesity among people tends to correlate positively with the number of fast-food restaurant visits [32]. In addition, the consumption of fast food endangers cardiovascular health as the existence of trans fats and cholesterol are high leading to risks of increased heart disease and metabolic diseases. Frequent consumption of fried dishes, which is popular in fast foods, has been proven to be connected with bad cholesterol and higher chances of having heart diseases [33]. Fast food is really popular, but its unfavorable health consequences oblige people to continue making efforts to replace their unhealthy eating habits with healthier ones. These efforts may include introducing or extending a range of healthy food alternatives at fast-food restaurants and making nutritional information more understandable to people. However, the efficacy of these interventions is not all that clear. Notwithstanding the willingness of a significant percentage of the populace who claim to prefer healthy alternatives, the data from the questionnaire show that only a small number consult nutrition information [32]. As a result, tackling the increase in fast-food eating and related health issues is the most dominant public health problem, which asks for various measures promoting the production of healthier foods and minimizing the risk of obesity and metabolic diseases. Table 1 provides a detailed overview of global trends in fast food consumption.

Nutrient composition of fast foods

The nutritional profile of fast food has been examined thoroughly, and it was found that fast food is capable of producing a lot of adverse health effects. This is the result of hypercaloric burgers, which contain unhealthy fats (including trans fats), high levels of sodium as well as sugars and low levels of nutrients such as fiber, vitamins, and minerals, which are vital for the body. Saunders and Middleton [41] studied fatty levels, along with the trans-fatty acids and salt, in popular fast foods that are taken out and consumed in the most deprived urban community in the UK. Most of these products were found to exceed the recommended daily allowance for at least one studied parameter, more than 30% and 27% of products exceeded the recommended levels for total fat or SFA and salt, respectively, which means their poor quality. A study conducted by Kök Şan and Gökçay in Turkey [42] examined the nutritional characteristics of commercially available food products for infants and toddlers. The study revealed that a significant number of these products did not meet the recommended nutrition requirements, especially in terms of their sugar and sodium content. This underscores the necessity for more stringent rules to guarantee healthier food choices for children, which might also encompass fast food consumed by older demographics. Jindarattanaporn et al. [43] assessed the nutritional profile of popular menu items available through online food delivery applications in Bangkok, Thailand. The study found that most items were unhealthy, with high sodium and sugar content, suggesting that the convenience of fast food delivery may exacerbate poor dietary choices and related health issues. Bernstein et al. [44] compared the nutritional compositions of foods in the Canadian Community Health Survey to a representative database of branded food products. This comparison highlighted significant differences in the nutrient content, particularly concerning saturated fats and

Table 1. Global trends and opportunities, demographic effects, and the changes in the fast food consumption patterns

Category	Trends and opportunities	Demographic impacts	Changing patterns	References
		Age		
Children [2–19 years]	Fast food consumption has increased.	Global fast food consumption among children is increasing, especially in urban areas.	The USA and Cebu consume more fast food, while China and Russia consume less.	[34]
Adolescents [12–17 years]	Weekly consumption is high.	Adolescents frequently consume fast food and carbonated drinks.	There is significant variability by region, with higher consumption in Latin America and high-income countries.	[35]
Young adults [18–29 years]	The fast food industry is growing.	Convenience and busy lifestyles drive high consumption among young adults.	Urbanization and increased disposable income are major factors.	[22]
Adults [30–50 years]	Increasing trends	Fast food consumption is increasing due to lifestyle changes and time constraints.	The preference for fast food varies by age and increases with urbanization.	[36]
		Gender		
Male	Consumption frequency is higher.	Men consume fast food more frequently than women.	Fast food advertising and marketing have a greater influence.	[37]
Female	Increasing trends	Women are increasingly consuming fast food, driven by urban lifestyles and work commitments.	Consuming fast food has an impact on diet and nutrition.	[38]
		Types of fast food		
Burgers and sandwiches	Popular globally	All age groups widely consume it, particularly in urban areas.	Consumers are driven by increased availability and convenience.	[20]
Fried chicken	High consumption	It is particularly popular in Asian countries and among young adults.	Brand influence (e. g., KFC, McDonald's) plays a significant role.	[39]
Pizza	Increasing popularity	It is consumed frequently among teens and young adults, especially in Western countries.	Fast delivery and online ordering increase consumption.	
Soft drinks	Commonly paired with fast food	Fast food intake rises with high consumption, particularly in adolescents.	Marketing and availability in fast food outlets drive consumption.	

fiber, reflecting the nutritional inadequacies of many fast food products. Marshellina et al. [45] conducted a study on medical students in Indonesia and discovered that the consumption of fast food was widespread. This was mostly due to the convenience and high stress levels experienced by the students. As a result, their protein intake was low while their fat consumption was high. This study emphasizes the necessity for improved nutritional instruction to alleviate the adverse health effects of fast food. Lastly, the study by Rodríguez-Martín et al. [46] compared the nutrient profiles of plant-based and animal-based foods in Spain. They found that plant-based foods, while generally healthier, still posed certain nutritional challenges, such as lower protein content compared to their animal-based counterparts.

Table 2 summarizes research findings on the nutrient composition of fast foods.

Table 2. Nutrient composition of fast foods

Study title	Key findings	Year	Reference
Energy, sodium, sugar, and saturated fat content of New Zealand fast-food products and meal combos in 2020.	Fast food diet increased bile-tolerant microbial genera and decreased fiber-fermenting bacteria, altering gut microbiome and metabolites.	2020	[47]
Availability and nutrient composition of vegetarian items at US fast-food restaurants	Menu items that are low-calorie, vegetarian, and free of gluten typically have healthier nutrient profiles	2021	[48]
Evaluation of the nutritional quality of ultra-processed foods (ready to eat + fast food): Fatty acid composition.	Fast-food products in NZ provided more energy, saturated fat, sugars, and sodium than recommended.	2021	[49]
Nutrient intake and dietary quality among children and adolescents by fast food consumption status: What we eat in America, NHANES2013–2016.	Vegetarian fast-food items generally lower in calories, saturated fat, protein, and sodium, but higher in sugar and non-sugar carbohydrates than non-vegetarian items.	2021	[50]
Nutritional composition of breakfast in children and adolescents with and without celiac disease in Spain-Role of gluten-free commercial products	Ultra-processed foods, including fast food, were high in saturated and monounsaturated fats, with seafood being an exception.	2021	[51]
Nutrient profile of commercially packaged food products in Türkiye	Regular consumption of fast food among children and adolescents is linked to worse diet quality and increased consumption of harmful nutrients.	2020	[52]
Level of knowledge on the effect of fast foods on health among young hypertensive patients in Bangladesh	Gluten-free breakfast products often had less protein and saturated fat but more salt compared to gluten-containing counterparts.	2023	[53]
Fast food and its effects among teenagers in the Municipal of Cachoeiro De Itapemirim-Espirito Santo, Brazil	Snacks had the highest energy and saturated fat, while beverages had the lowest energy, fat, and protein. Confectionaries were high in carbohydrates and sugars.	2023	[54]

Health impacts of fast food consumption

The use of fast food has been increasingly associated with a variety of negative health effects, especially metabolic problems. Studies offer a thorough examination of the diverse effects of fast food on health. A study conducted by Taniim et al. [53] reveals that young hypertensive individuals in Bangladesh possess an inadequate understanding of the adverse health consequences associated with fast food consumption. This underscores the necessity for health education initiatives aimed at enhancing awareness. Parvin et al. [55] demonstrated that there is a correlation between fast food intake and higher BMI among nursing and public health students in London. The study also reveals that females are more likely to be attracted to the flavor of fast food, while males are more inclined towards its convenience. Nyangoya and Attoni [54] found that fast food consumption among teenagers in Brazil is linked to obesity, depression, diabetes, heart disease, and reproductive health issues, highlighting the broad spectrum of health risks associated with fast food. Marshellina et al. [45] identified that medical students at Tanjungpura University have low protein intake and high-fat consumption due to frequent fast food intake, necessitating better nutritional education. Baskati and Pareek [56] discuss the shift towards high-calorie fast foods in India, which has contributed to rising obesity, coronary artery disease, and diabetes mellitus. Wijaya et al. [57] emphasize that junk food, including fast foods, significantly impacts body weight and is a key factor in the global obesity pandemic. Ramadani and Jannah [58] examined the relationship between fast food consumption and obesity among high school students in Indonesia, finding no significant relationship, but noting the need for further education on healthy eating. El-hasry et al. [59] assessed the perception of mothers regarding the effect of fast food on preschool children's health, finding poor knowledge and practices among mothers, highlighting the need for targeted health education programs. Pratheepkumar et al. [60] found a high prevalence of fast food consumption among university students, with significant associations between fast food intake and obesity, emphasizing the influence of peer pressure and convenience. AlTamimi et al. [61] noted that fast food intake is prevalent among middle-aged men in Saudi Arabia, with significant associations with nationality and obesity. Fitrianti et al. [62] identified key factors influencing fast food consumption among adolescents in Jakarta, including knowledge, body image, and promotional influences. Abrahamsson et al. [63] found that exposure to fast food restaurants during childhood and adolescence increases BMI and negatively impacts cognitive ability. Alanazi et al. [64] reviewed the impact of social media on fast food consumption, finding that social media significantly influences poor nutritional habits, particularly among children and adolescents. Pushkar et al. [65] found a high prevalence of fast food consumption among medical students, with significant associations between consumption frequency and BMI. Kasmarini et al. [66] noted that frequent fast food consumption among adolescents is linked to poor sleep quality and higher rates of overweight and obesity. Lestari et al. [67] found a significant relationship between fast food consumption and obesity among adolescents in Kendari, Indonesia. Mendonca discusses how modern food habits, including increased fast food consumption, have led to a rise in health issues such as obesity, diabetes, and cardiovascular diseases [68]. Kagathara et al. [69] found that dietary practices, including frequent consumption of fast food, are associated with mental health issues such as stress, depression, and anxiety among medical students. Finally, Saragih et al. [70] showed that nutritional education using animation media can effectively reduce fast food consumption habits and obesity rates among adolescents. Figure 2 summarizes some diseases linked to fast food consumption.

Obesity and weight gain

There has been a link between the intake of fast foods and obesity, and many studies have pointed to the impact of fast food on weight gain and obesity. This section of the paper discusses how high intakes of fast food cause obesity and analyses the mechanisms through which this occurs and associated health effects. One of the many reasons why fast foods cause obesity is their trend toward high-calorie content. The average fast food meal, for example, hamburgers, fries, and drinks, among others, has more than 1200 calories, which is over 50% of what an ordinary adult requires in a day [69,70].

Such caloric excess easily translates into weight gain if consumed regularly [71]. Furthermore, fast food is typically high in unhealthy fats, particularly saturated and trans fats, and sugars. They are energy-dense nutrients that provide many calories in a small volume of food. Consumption of such fats and sugars contributes to weight gain not only by increasing calories but also by causing metabolic imbalances. For instance, consuming snacks that are high in trans-fatty acids increases abdominal fat, a risk factor for metabolic syndrome and cardiovascular diseases [22,72]. The composition of fast food may also result in low satiety for consumers. Fast food lacks dietary fiber and protein, which contribute to heightened satiety and reduced inter-meal food intake. This way, one will likely go beyond the required intake and eventually gain weight [73]. The combination of fat with a high content of sugar and salt in fast foods makes them highly palatable and, therefore, increases appetite, compelling people to overeat. Furthermore, due to accessibility and ease of use of fast foods, a significant number of people frequently opt for fast food in today's hectic lives. Studies have indicated that eating fast food two or more times each week significantly increases the risk of gaining a substantial amount of weight and becoming obese [14,74]. Furthermore, fast food companies' widespread marketing tools, such as advertising and promotion, encourage consumption, even among children and adolescents [75,76].

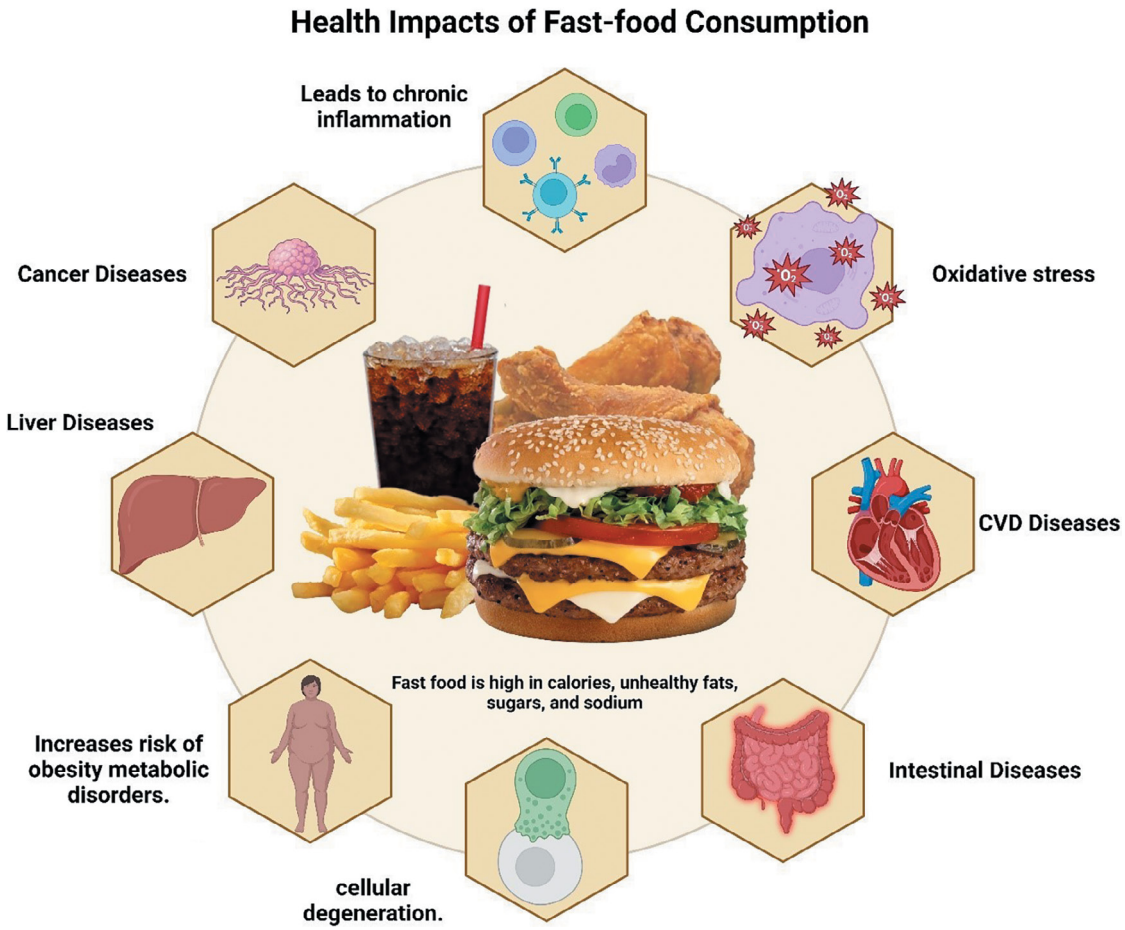

Figure 2. Health impacts of fast food consumption

Simply put, the trend of fast-food consumption correlates with the prevalence of obesity epidemics. Obesity is one of the leading risk factors for several chronic diseases, such as type 2 diabetes, cardiovascular diseases, and specific cancers. The presence of excess body fat, particularly abdominal body mass, has been associated with insulin resistance, dyslipidemia, and hypertension, each of which is related to the pathogenesis of the metabolic syndrome per se [77,78]. Obesity, therefore, poses a severe health threat leading to a reduction in quality and quantity of life expectancy [79]. The CARDIA study showed that frequent fast food consumption is associated with significant weight gain and increased insulin resistance over 15 years, increasing the risk of obesity and type 2 diabetes [51]. In addition, found that fast food consumption in teenagers was associated with a higher BMI and body fat percentage, leading to increased odds of obesity [71]. Moreover, an analysis of U.S. adults showed that fast food consumption results in a diet high in energy density and low in essential micronutrient density, contributing to overweight and obesity [72]. Fast food consumption among children leads to higher caloric intake, more total fat and added sugars, and poorer diet quality, which increases obesity risk [73]. Longitudinal research suggests that higher intake of fast food and skipping breakfast during youth are indicators of weight growth in adulthood, which contributes to obesity [83]. Table 3 summarizes some studies on the effects of fast food on obesity and weight gain.

Mechanisms of obesity and weight gain on aging

Obesity and weight growth have a substantial impact on the aging process due to many biological factors. They lead to faster aging, higher rates of illness, and a shorter lifetime. Chronic inflammation is a significant pathway, in which adipose tissue in obese individuals releases pro-inflammatory cytokines such as TNF-alpha, IL-6, and CRP. This leads to systemic inflammation, which speeds up cellular aging and contributes to age-related illnesses such as cardiovascular disease, type 2 diabetes, and Alzheimer's disease [84]. Another important mechanism is oxidative stress. Increased adiposity causes an elevated production of reactive oxygen species (ROS) and a decrease in antioxidant defenses, leading to cellular damage and senescence [85]. Furthermore, obesity is closely linked to insulin resistance, causing metabolic dysfunction that elevates blood glucose levels and insulin production, ultimately leading to type 2 diabetes and its associated complications, which hasten aging [86]. Hormonal imbalances, such as leptin resistance, disrupt energy homeostasis and exacerbate weight gain, while also affecting reproductive health and accelerating aging [87]. Mitochondrial dysfunction, a hallmark of aging, is exacerbated by obesity, leading to decreased cellular energy production and increased ROS, further accelerating cellular aging [88]. Additionally, obesity accelerates telomere shortening, which limits cellular replication and longevity, contributing to premature aging and higher disease risk. Obesity also impairs immune

Table 3. Some studies on the effects of fast food on the obesity and weight gain

Key findings	Sample size	Human/ Animal models	Reference
Consuming fast food is a major factor in the development of weight gain and obesity	16 studies	Human	[74]
Regularly consuming fast food is linked to weight gain and heightened insulin resistance	3,031	Human	[31]
Fast food consumption in teenagers is linked to higher BMI and body fat percentage	Not specified	Human	[75]
Consuming fast food leads to a diet that is rich in energy but lacks important micronutrients	At least 1,000	Human	[72]
Fast food consumption in children leads to higher caloric intake and poorer diet quality.	6,212	Human	[76]
Heightened consumption of fast food and the habit of missing breakfast are indicative of weight increase from adolescent to adulthood.	9,919	Human	[77]
Aging reduces appetite and energy intake, impacting weight management	3,574 (older adults)	Human	[78]
Regular fast food consumption is linked to weight gain and adverse metabolic outcomes	3,643	Human	[79]
Fast food consumption in children is linked to increased caloric intake and poorer diet quality	National survey	Human	[80]
Periodic and intermittent fasting are helpful to health, since they combat age-related illnesses and obesity	Not specified	Animal (rats and mice)	[81]
The consumption of probiotic yogurt alongside a Western diet prevents age-related weight gain and alters the profiles of pro-inflammatory immune cells	Not specified	Animal (mice)	[82]
Obese mice showed increased body fat and weight gain compared to lean mice, indicating metabolic differences	Not specified	Animal (mice)	[83]

function, leading to immunosenescence, a diminished immune response that increases vulnerability to infections and reduces tissue repair capacity [89]. Cardiovascular strain from excess weight causes hypertension and atherosclerosis, further aging the cardiovascular system [90]. Lastly, the metabolic and inflammatory stresses induced by obesity can lead to neurodegeneration, increasing the risk of diseases such as Alzheimer's disease and impairing bone and joint health, leading to conditions such as osteoarthritis and reduced mobility, which are critical aspects of aging [91]. Figure 3 shows the mechanism of action on aging.

Cardiovascular health

Regular consumption of fast food extends its effects on heart health. This section elaborates on how fast foods always have unhealthy fats, which therefore enhance the development of most heart diseases. To begin with, fast foods contain unhealthy fats such as trans fat and saturated fat. Saturated fats raise the level of low-density lipoprotein (LDL) in the blood, the so-called "bad" cholesterol. This is critical for the atherosclerosis process. Trans fats, which have a long shelf life in fast food items, are still more harmful. Not only do they raise serum LDL cholesterol, but they

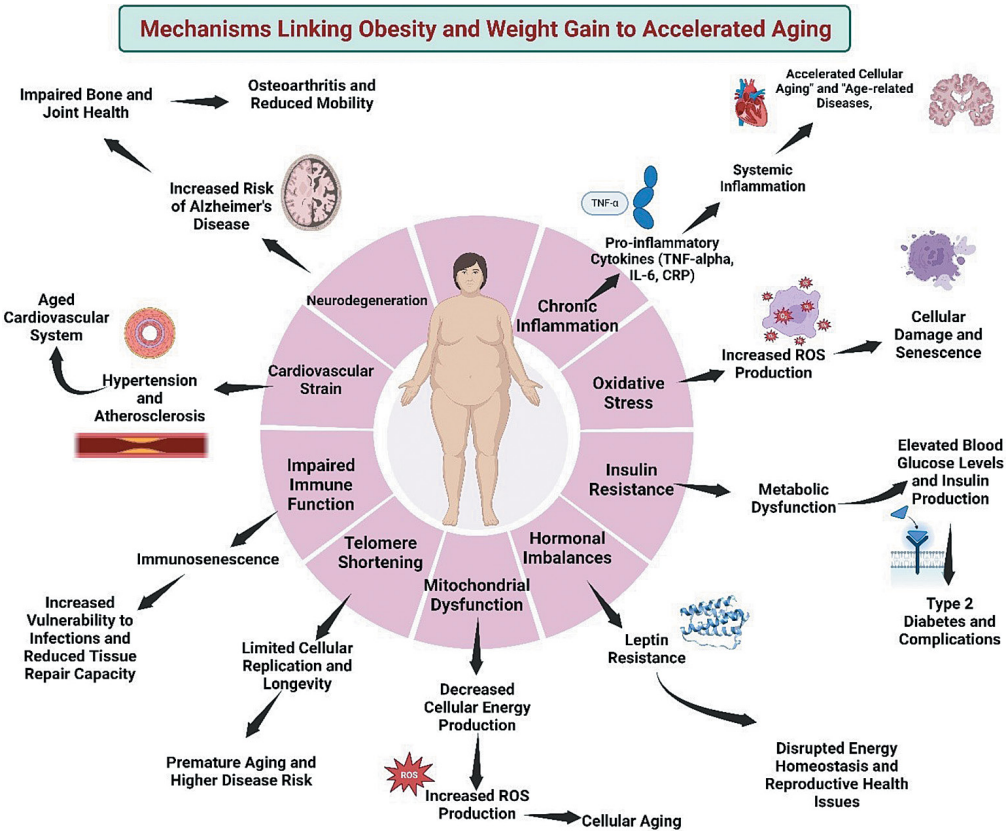

Figure 3. Mechanisms linking obesity and weight gain with aging

also reduce the HDL cholesterol that removes LDL cholesterol from the blood [9]. Another extreme health concern is that the rate, at which the consumption of fast food increases the level of sodium in the body, is alarming. High sodium intake has been recorded as a substantial contributing factor to the increase in high blood pressure, which is the bedrock of most chronic illnesses, especially heart attacks and strokes [92]. In a simple meal, most fast foods exceed the limit on daily sodium consumption. For instance, fast food with a hamburger and fries can easily contain over 1,500 milligrams of sodium — close to the 2,300 milligrams per day recommendation from the American Heart Association [93,94]. Also, most fast food is high in sugar and refined carbohydrates, particularly beverages and desserts. Overconsumption of sugar leads to weight gain and obesity prevalence, both of which are associated with numerous cardiovascular disorders. Carbohydrates also cause the rapid elevation of blood glucose levels, leading to insulin resistance and type 2 diabetes — conditions closely related to cardiovascular health issues [26]. Furthermore, there are many studies showing how unhealthy fats and sugars in fast food raise levels of oxidative stress and inflammation, which are found in most cardiovascular diseases. Essentially, oxidative stress is a state that renders an individual susceptible to an imbalance between free radicals and antioxidant defense that leads to cellular and tissue damage. Inflammation is meant to be the body's defense against harmful stimuli, but it damages the arteries and, in that regard makes atherosclerosis worse once it becomes a chronic process [1]. It also leads to chronic blood lipid abnormalities among regular fast food consumers. High LDL cholesterol and triglycerides, along with a fall in HDL cholesterol, are expected consequences of fast-food consumption. These lipid abnormalities are significant contributors to the development of coronary artery disease and other cardiovascular diseases [1]. Several studies have found links between fast food consumption and adverse cardiovascular outcomes. For instance, revealed a significantly elevated risk for coronary heart disease in individuals who included fast foods in their daily meal plan. This study also found that the frequency of fast food consumption increased the risk [95]. High levels of CRP in the system indicate a high risk for cardiovascular events such as heart attack and stroke, independently of other risk factors [96,97]. In addition, a study found that regular fast food consumption led to significantly increased BMI, blood sugar levels, and lipid profiles, indicating higher cardiovascular disease risk [98]. A study conducted in Bangladesh showed that young individuals with hypertension who excessively consume fast food face an elevated susceptibility to obesity and cardiovascular complications. Implementing awareness programs is crucial to mitigate the consumption of fast food [53]. Ramadani and Jannah revealed a significant prevalence of fast food consumption among students, resulting in elevated obesity rates and possible cardiovascular hazards [58]. Vercammen et al. discovered that there is a substantial increase in the likelihood of developing cardiovascular disorders when individuals experience food hardship. Individuals classified as adults with severe food insecurity had a greater likelihood of having a 10-year cardiovascular disease risk of at least 20%. This emphasizes the necessity for initiatives aimed at enhancing food security and diminishing reliance on fast food [99]. Bahadoran et al. [1] conducted a comprehensive analysis of the effects of fast food on cardiometabolic disorders, such as obesity, insulin resistance, and cardiovascular diseases. The study emphasizes that regularly consuming fast food is linked to higher calorie intake, lower diet quality, and an elevated risk of metabolic syndrome. Nadeem et al. [100] carried out a study to examine the correlation between the consumption of fast food and the occurrence of coronary heart disease in males residing in Peshawar. The results demonstrated that regular intake of fast food dramatically elevates the likelihood of acquiring coronary heart disease [100]. Duffey et al. [79] investigated the effects of consuming fast food on the quality of one's diet and metabolic outcomes. The results showed that a higher intake of fast food is linked to weight gain, insulin resistance, and dyslipidemia in young adults. Bowman et al. [76] studied the dietary habits of children and found that fast food consumption leads to higher caloric intake, increased fat and sugar consumption, and reduced dietary quality, contributing to obesity risk. Whitton et al. [101] revealed that a healthy dietary pattern is inversely associated with cardiovascular risk factors such as BMI, LDL cholesterol, and fasting triglycerides, highlighting the benefits of reducing fast food intake.

Fraser et al. [75] performed a spatial study that demonstrated a correlation between fast food intake and elevated BMI and body fat percentage in UK adolescents. This suggests a significant connection between the availability of fast food and obesity. Ferrara et al. [102] compared the impact of fast food versus slow food on hypertension control, showing that fast food significantly worsens blood pressure and metabolic profiles, while slow food, particularly Mediterranean diets, offers protective benefits. Basu et al. [103] highlighted that frequent fast food consumption is associated with lower nutrient adequacy, particularly in meeting Dietary Reference Intakes (DRIs) for essential nutrients, while increasing sodium and sugar intake. Bahadoran et al. [104] found that increased fast food consumption among Iranian adults is linked to higher intakes of unhealthy nutrients and poor cardiovascular health metrics, such as increased BMI and serum triglycerides. Odegaard et al. [105] demonstrated that frequent intake of Western-style fast food significantly increases the risk of type 2 diabetes and coronary heart disease mortality among Chinese Singaporeans. Sohouli et al. [106] revealed that fast food consumption is linked to adverse lipid profiles and increased obesity rates among patients with diabetic nephropathy, exacerbating cardiovascular risk factors. Finally, Schmidt et al. [107] showed that fast food consump-

tion among black and white adolescent girls is associated with higher intake of calories, fat, and sodium, leading to poorer diet quality and increased cardiovascular risk. In conclusion, the effects of eating fast foods on the cardiovascular system are pretty pronounced and shocking. High amounts of fat clog the arteries. Moreover, fast food contains massive sodium and sugar levels and has relatively very low nutritional value. Public health interventions or individual lifestyle changes can assume dietary patterns to reduce the harm fast food may do to cardiovascular health.

Mechanisms linking cardiovascular health with aging

Cardiovascular health plays a crucial role in the aging process, influencing the onset and progression of age-related diseases. The key aspect is the occurrence of inflammageing, in which there is a low-level systemic inflammatory state that speeds up vascular aging and contributes to cardiovascular diseases (CVD), such as atherosclerosis and heart failure [108]. Such an inflammatory process is mostly the result of oxidative stress and mitochondrial disarray, which is associated with the loss of cellular function and the appearance of senescence in cardiovascular cells. Telomere loss, a more common symptom of aging, will prevent cardiovascular tissues from being repaired and will thus worsen age-related conditions [109]. Furthermore, these also include hormonal changes and metabolic dysregulation, such as insulin resistance and dyslipidemia, that result in cardiovascular aging via the promotion of endothelial dysfunction and arterial stiffness, respectively [110]. It is also important to examine the role microRNA is playing in the regulation of autophagy and other cellular protection. There are various control mechanisms and microRNA are among them being crucial for a cell to survive. The accurate regulation of this process is imperative when a person goes through the COVID-19 pandemic, otherwise, excessive cellular degradation and cardiovascular impairment can occur [111]. Furthermore, hereditary and epigenetic influences are the main factors that impact cardiovascular health in a significant manner, leading to the need to study how dietary interventions (for example, intermittent fasting) can affect the aging cardiovascular system [112]. Exercise and diet must be an integral part of person's efforts to keep a healthy heart and avoid heart diseases as they become older. Regular exercise improves endothelial function, reduces oxidative stress, and decreases inflammation, thereby mitigating age-related cardiovascular decline [113]. Similarly, certain dietary patterns and nutraceuticals can enhance cardiovascular health by targeting the fundamental mechanisms of aging, such as enhancing nitric oxide bioavailability and reducing systemic inflammation [114]. Figure 4 shows the mechanism of CVD and aging.

Metabolic disorders

A vast array of metabolic disorders, including clusters of conditions that increase the risk of heart disease, stroke, and type 2 diabetes, have closely linked fast food

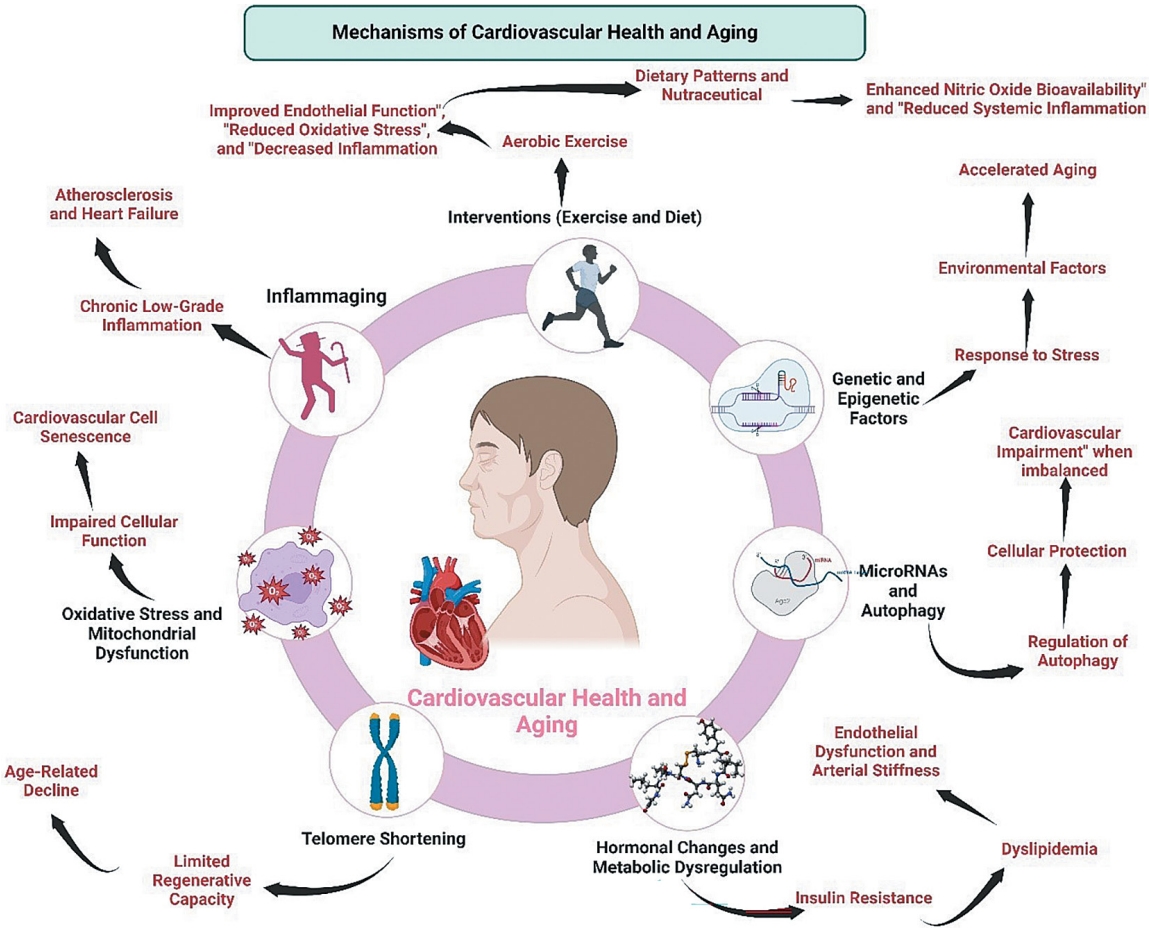

Figure 4. Mechanisms of cardiovascular health and aging

consumption to their development [1]. This section focuses on how the average nutritional profile of fast food contributes to metabolic disorders, while also addressing broader health concerns. First, fast foods are high in refined carbohydrates and sugars, which bear a high glycemic load, i. e., they are foods with a high glycemic load. As a result, they can cause sharp, consecutive peaks in blood sugar, which then necessitate increased insulin production. This leads to cells becoming resistant to insulin, the hormone responsible for controlling blood sugar levels. This phenomenon is a classic characteristic of metabolic syndrome and type 2 diabetes [27,115]. For instance, studies have directly linked the development of insulin resistance to high consumption of sugar-sweetened beverages, a standard component of fast food meals. In addition to high caloric content and poor nutritional quality, fast food intake directly leads to weight gain and, subsequently, obesity — all crucial predictors of metabolic disorders. Obesity, especially abdominal obesity, assumes a central role in metabolic syndrome. This syndrome is characterized by a cluster of symptoms, which include elevated blood pressure, high blood sugar, excessive abdominal fat, and abnormal levels of cholesterol or triglycerides [116,117]. These conditions tend to occur together, thus raising the risk of heart disease, stroke, and diabetes. Fast foods, once again, contain high levels of unhealthy fats, such as saturated and trans fats, which negatively impact lipid profiles. Dyslipidemia is one of the most common outcomes associated with regular fast food consumption. It is characterized by high levels of LDL cholesterol and triglycerides, accompanied by significantly low HDL cholesterol. Such lipid abnormalities certainly contribute to the development of atherosclerosis, a condition that dramatically increases cardiovascular disease risk and is one of the metabolic syndrome components [9,118]. In addition, fast food causes chronic inflammation and oxidative stress, which contribute to metabolic disorders. Fast foods are usually rich in unhealthy fats and refined sugars, which can lead to obesity. A frequent consequence of obesity is metabolic syndrome associated with the pro-inflammatory states [119]. The synthesis of pro-inflammatory cytokines and reactive species of oxygen in the body is stimulated, thus causing cellular damage and developing chronic diseases. Chronic inflammation is a central player in insulin resistance and metabolic syndrome development in several cases. Furthermore, recent scientific studies have focused on diet and gut microbiota, a population of microorganisms living in the intestinal tract [120]. Fast food consumption can cause adverse change in gut microbiota, favoring the development of harmful bacteria while inhibiting good bacteria. This dysbiosis may, as a consequence of that, become an inflammatory, insulin-resistant and metabolic disorder [121]. It is important to mention that many studies have been focused on this close association of fast food consumption and metabolic disorders. Pereira et al. observed the presence of insulin resistance and type 2 diabetes for those who eat fast foods at least thrice or even four times a week, which was the worst diet [13]. Fast food has been a major contributing factor to the development of inflammatory and oxidative stress markers, that eventually can lead to the development of metabolic syndrome [122]. The effect of fast food on metabolic diseases is not the only one but also the very sophisticated one. People must be serious in dealing with dietary problems that junk foods might cause. In particular, the intake of fast foods that are high in calories, and have unhealthy fats such as trans fats, refined carbohydrates, and sugars, constitute the major part of these problems. The fast food industry presents us with fast food as the primary cause of obesity, insulin resistance, dyslipidemia, inflammation, and gut microbiota imbalance, as well as a bad way of eating. Therefore, all these would consequently make metabolic syndrome and type 2 diabetes complicated ways. Even in the case of severe health complications, there should always be a rule on the public level of no junk food consumption, followed by alterations in dietary habits, to avoid these risks for good health. Furthermore, dietary patterns that incorporate high consumption of processed and fast foods have been linked to significant increases in abdominal adiposity and metabolic-associated fatty liver disease (MAFLD). Longitudinal studies have shown that higher average fast food intake over decades correlates with increased visceral adipose tissue and liver fat levels, indicating the long-term metabolic risks associated with fast food diets [123]. The relationship between fast food consumption and metabolic disorders is further complicated by its role in exacerbating inflammation and oxidative stress, key drivers of metabolic dysfunction. Fast food diets, rich in fats and sugars, contribute to chronic low-grade inflammation and oxidative stress, promoting insulin resistance and metabolic dysregulation [124]. One critical mechanism by which fast food exacerbates metabolic disorders is through its impact on gut microbiota and bile acid metabolism. The investigation has demonstrated that a single fast-food binge can change gut microbiota composition, which may lead to an increase in bile acids and lead to liver function and inflammation alterations [125]. This modification in the gut-liver axis is an original metabolic process keeping in touch fast food consumption with the birth of metabolic disorders.

Mechanisms linking metabolic disorders with aging

The aging process is significantly influenced by metabolic disorders through different pathways. One of the principal ways is the chronic inflammation called "inflammageing", which is the case where the metabolic disorder is persistent low-grade inflammation. Chronic inflammation accelerates cellular aging and along the way, it is also a contributor to age-related diseases, such as cardiovascular disease and type 2 diabetes [126,127]. Besides this, mitochondrial disorder the problem with mitochondria, which are the powerhouses of the cells, is one of the prominent signs of old age and metabolism-related diseases. Throughout an individual's lifespan, the efficiency of mitochondria drops,

which in turn, along with the higher production of reactive oxygen species (ROS), poses the risk of occurrence of oxidative stress later on, which in its sense can be extremely harmful to cell structures and lead to aging. Insulin resistance, a common feature of metabolic disorders, disrupts glucose homeostasis and exacerbates aging by impairing cellular metabolism and increasing the risk of developing age-related diseases [128]. Furthermore, metabolic slowdown, which involves a gradual reduction in metabolic rate and efficiency, contributes to the hallmark features of aging such as weight gain, basal inflammation, and insulin resistance [129]. Autophagy, the cellular process that removes damaged organelles and proteins, declines with age and in conditions of overnutrition, leading to the accumulation of cellular damage and further metabolic derangements [130]. Moreover, metabolic disorders, such as obesity and type 2 diabetes, induce changes in cellular energy metabolism, including decreased insulin sensitivity and altered mitochondrial function, which are significant contributors to aging [131]. The hypothalamus, which plays a central role in regulating metabolic physiology, also undergoes functional decline with age, further exacerbating metabolic dysregulation and accelerating the aging process [132]. The interplay between metabolic disorders and aging is also evident in the regulation of proteostasis, where metabolic imbalance affects protein homeostasis, leading to the accumulation of misfolded proteins and cellular stress [133]. Additionally, metabolic disorders exacerbate neurodegenerative diseases, with mechanisms such as insulin resistance and neuroinflammation playing critical roles [134].

Cancer risks

The starved generation has been mentioned for a long time. If one is consuming a diet of unhealthy food for a long time and is not active, he/she is likely to get metabolic disease specifically diabetes, stroke, kidney disease, and heart disease. The greater the obesity, the higher the likelihood of developing the irremediable problem. This section discusses how the nutritional elements of fast food result in cancer and delves deeper into broader health concerns. First, fast foods are often highly processed and include meats such as bacon, sausage, and hot dogs, which fall into the category of Group 1 carcinogens, according to the International Agency for Research on Cancer [135]. Smoking, curing, salting, or adding chemical preservatives preserves these meats, but introduces carcinogenic compounds like nitrates and nitrites. These compounds, upon ingestion into the body, form N-nitroso compounds (NOCs), which are carcinogenic and known to cause DNA damage and cancer, such as colorectal cancer [136]. Many fast food items, especially fried ones, contain acrylamide, a chemical formed during the high-temperature cooking of starchy foods. The IARC has classified this chemical as a probable human carcinogen. Frequent consumption of deep-fried food items, like French fries and deep-fried chicken, raises the intake of acrylamide [137], linked to a higher risk of cancers such as ovarian, endometrial, and renal cell cancer. Additionally, most fast food-related meals serve high-sugar drinks, which significantly contribute to the daily caloric intake and further promote obesity. Obesity is known to be an essential risk factor for many cancers, including those of the colorectum, endometrium, postmenopausal breast, kidney, and pancreas. Excess body fat capacitively increases levels of insulin and insulin-like growth factors, leading to increased cell proliferation and decreased apoptosis; it also produces and circulates estrogen. Furthermore, adipose tissue is linked to hormone-related cancers. Moreover, the high fat content in most fast foods, which is incredibly saturated with trans fats, is a worrying factor. Researchers have linked these types of fats to an increased risk of contracting cancer. For example, a high intake of saturated fats has been associated with breast cancer. Risk factors include maintaining a state of chronic inflammation, insulin resistance, and changes in cell membrane structures that can potentiate cancer cell differentiation and metastasis [117,138]. Fast foods are also generally low in essential nutrients and antioxidants that protect cells from oxidative damage. Antioxidants, primarily present in fruits, vegetables, and whole grains, neutralize free radicals that often cause DNA damage and increase the risk of cancer. A diet high in fast food and low in nutrient-dense foods might predispose one to cancer due to the insufficient levels of compounds with protective effects [118,139]. Therefore, the impact of fast food consumption on cancer risk is vast and multi-fold. Fast food consumption significantly increases the risk of various cancers due to its high content of processed meats, the presence of acrylamide in fried foods, the consumption of sugary beverages that lead to being overweight, and the high-fat content with low nutrient density. Thus, the public health intervention that would change these dietary habits and thus provide a balanced diet with high amounts of fruits such as vegetables and whole grains instead of fast food would be the best solution to lower the cancer risk. It certainly would. A study by Huybrechts et al. [140] found that ultra-processed foods (UPFs), including fast foods, were associated with an increased risk of breast cancer in young women in Latin America. Papadimitriou et al. [141] reviewed diet and cancer risk, finding strong evidence linking alcohol and red meat consumption to increased cancer risks, whereas fast food consumption was implicitly connected to these risk factors. Similarly, Jafari et al. [142] demonstrated a positive association between UPF consumption, including fast foods, and colorectal cancer risk in Tehran, Iran. Farvid et al. [143] identified processed meat, often found in fast foods, as a significant risk factor for various cancers, including colorectal, lung, and breast cancers. Wang et al. [144] found that high consumption of ultra-processed foods was associated with increased colorectal cancer risk, with significant gender differences in risk profiles. Bevel et al. [145] highlighted the association

between living in food swamps, characterized by high fast food availability, and elevated obesity-related cancer mortality rates in the US. Zhong et al. [146] found that deep-fried foods, common in fast food diets, were linked to a reduced risk of pancreatic cancer, though further research is needed to confirm these findings. Arya [147] studied the link between fast food consumption and anthropometric risk factors among college students, indirectly highlighting cancer risks through obesity-related mechanisms. Wang et al. [144] quantified the obesity-related cancer burden associated with UPF consumption, indicating significant contributions to new cancer cases. Khong et al. [148] found a correlation between high fasting blood glucose levels and increased cancer risk, implicating dietary habits including fast food consumption. Wu et al. [149] discussed the rising fast food consumption in Asia and its implications for obesity and cancer risk, calling for regulatory measures. Li et al. [150] compared fast food consumption across 54 low- and middle-income countries, highlighting the prevalence and associated health risks, including cancer. Bohlouli et al. [151] reviewed the impact of fast food consumption on COVID-19 severity and long-term complications, including increased cancer risks through chronic inflammation. Papier et al. [152] examined the association between meat consumption, common in fast foods, and various health conditions, highlighting increased cancer risks. Additionally, Kim et al. [153] investigated the link between fasting blood glucose levels and pancreatic cancer, underscoring the role of dietary habits in cancer risk. Brandhorst [154] discussed how dietary interventions, including fasting, can augment cancer treatment, indirectly implicating the role of fast food in cancer progression. Furthermore, Aveta et al. [155] reviewed the impact of meat intake on bladder cancer, emphasizing the carcinogenic potential of red and processed meats found in fast foods. Givens [156] summarized evidence linking dairy consumption to cancer risk, indirectly relating to fast food dietary patterns. Finally, Wijaya et al. [57] explored the impact of junk food, including fast foods, on body weight and associated cancer risks, emphasizing the role of dietary habits in health outcomes.

Mechanisms linking cancer risk with aging

Cancer risk increases significantly with aging due to various biological mechanisms that interlink aging and cancer development. One major mechanism is chronic inflammation, also known as "inflammageing," where persistent low-grade inflammation contributes to genomic instability and carcinogenesis. Aging-related changes in the immune system, such as immunosenescence, reduce the body's ability to detect and eliminate cancer cells, thereby increasing cancer risk [157]. Additionally, the accumulation of DNA damage over time, coupled with decreased DNA repair efficiency, leads to mutations that drive cancer progression [158]. Mitochondrial dysfunction, common in both aging and cancer, results in increased production of reactive oxygen species (ROS), which further damages DNA and cellular components, promoting oncogenesis [159]. Epigenetic alterations, such as DNA methylation and histone modification, also play a crucial role in linking aging to cancer by modifying gene expression in a way that favors tumorigenesis [160]. Furthermore, cellular senescence, a state where cells stop dividing but do not die, is a double-edged sword in aging and cancer. While it initially acts as a barrier to cancer by halting the proliferation of damaged cells, the accumulation of senescent cells contributes to the pro-inflammatory environment and tissue dysfunction, creating a conducive environment for cancer development [161]. Metabolic reprogramming in aging, characterized by altered nutrient sensing and energy production, supports cancer cell survival and growth [162]. The declining efficiency of proteostasis, the process by which cells maintain protein balance, leads to the accumulation of misfolded proteins and cellular stress, which are implicated in both aging and cancer [163]. Besides this, cell damage caused by telomere shortening, a part of the natural aging process, hinders the cell's ability to divide and preserve tissue integrity, and it also causes the genomic instability that has the starting power for the development of cancer [164]. To summarize, the interaction between aging and cancer is considerably complicated and is rooted in chronic inflammation, the malfunctioning immune system, DNA damage, mitochondrial dysfunction, epigenetic changes, cellular senescence, metabolic reproduction, impaired proteostasis, and telomere shortening. These pathways collectively result in the augmentation of cancer susceptibility linked to old age.

Liver disease

A lot of discussions have been going over recent months on the bad impact of fast food on people's health. The increasing prevalence of fast food meals has been associated with one of the primary concerns called non-alcoholic fatty liver disease (NAFLD). Studies have indicated that lower fat and lower calorie intake cause a significant decrease in liver disease. Moreover, the research indicates that the causes of the disease are the high caloric and unhealthy fat content typical of fast foods. The given collection of studies aims to supply a comprehensive overview of the vital link between fast food consumption and the development and progression of NAFLD. The relationship between fast food and liver disease, particularly non-alcoholic fatty liver disease (NAFLD), has been investigated extensively. Tavakoli et al. [165] discovered that NAFLD risk significantly grows in men who consume fast foods incessantly. Moreover, Mohammadi et al. [166] specified that the consumption of different kinds of fast foods, especially pizza, was the factor that made the risk of NAFLD higher, and pizza had the highest risk association. The study by Mohammadi et al. [166] eventually led to the conclusion that the intake of fast food in the patients with NAFLD was by far most pronounced among the reasons for the diseases as compared to the normal healthy individuals, hence, drawing

the researcher's pointed call for both the medical community and public to look into the future driver of the turn in liver health due to fast food intake. This conclusion is similar to that made by Marchesini et al. [167] who talked about the likely damage to the liver by fast food, namely the metabolic syndrome and liver dysfunction in the case of over-consumption of fast food. Furthermore, Mager et al. [168] discovered that consuming a meal rich in saturated fat resulted in an extended period of elevated levels of fat in the blood after eating, increased insulin levels, and changed expression of lipoproteins in obese children. These findings establish a connection between these factors and non-alcoholic fatty liver disease (NAFLD). Figge et al [125] showed that even a single fast food binge can induce significant metabolic changes and liver injury markers, suggesting a robust gut-liver axis response. Odegaard et al. [104] found a significant correlation between regular intake of fast food over a period of 25 years and the occurrence of NAFLD. This correlation remained significant even after accounting for other characteristics related to diet and lifestyle. In addition, Odegaard et al. [123] investigated the correlation between the consumption of fast food and the accumulation of fat in the liver. They found that higher levels of fat in the abdominal area and an increased likelihood of developing metabolic-related fatty liver disease (MAFLD) were associated with a larger intake of fast food. Khatatbeh et al. [169] observed that fast food consumption was associated with significant weight gain and altered liver enzyme levels among university students, suggesting potential long-term liver health implications. Takahashi et al. [170] highlighted a gender difference in the association between eating speed and NAFLD in type 2 diabetes patients, with fast eating significantly increasing NAFLD risk in men. Tamargo et al. [171] demonstrated that food insecurity, often leading to higher fast food intake, exacerbated the risk of NAFLD and liver fibrosis, particularly among low-income individuals. Charlton et al. [172] developed a mouse model demonstrating that a fast food-based diet induced progressive fibrosis and steatohepatitis, mirroring human NAFLD. Moreover, Bayol et al. [173] showed that a maternal junk food diet during pregnancy and lactation predisposed offspring to NAFLD, highlighting the long-term effects of early dietary exposure. Kalafati et al. [174] found that a fast food-type dietary pattern significantly increased the odds of NAFLD in a Greek population, with associated higher levels of C-reactive protein and uric acid. In the same vein, Uchiyama et al. [175] conducted a pilot study showing that a traditional Japanese dietary pattern inversely related to liver fat indices, suggesting that diet quality impacts liver health.; Delzenne et al. [176] showed that fasting followed by a high carbohydrate-fat-free diet in rats led to significant liver lipid accumulation and steatosis, offering insights into dietary impacts on liver health. Ouyang et al. [177] found that patients with NAFLD had significantly higher fructose consumption, with fructose metabolism contributing to liver fat accumulation. Finally, Henney et al. [178] demonstrated a dose-response relationship between ultra-processed food intake and NAFLD, suggesting that public health measures to reduce such consumption are crucial.

Mechanisms linking liver disease with aging

Liver disease significantly impacts the aging process through a variety of complex mechanisms that exacerbate age-related physiological decline. Chronic inflammation, known as "inflammaging" plays a central role in aging and liver disease, where persistent low-grade inflammation accelerates liver dysfunction and fibrosis. This chronic inflammation is often driven by age-related changes in the immune system, particularly the accumulation of pro-inflammatory hepatic macrophages, which secrete cytokines such as TNF-α and IL-6, leading to tissue damage and impaired liver function [179]. Fuel molecules, such as glucose or fatty acids are burnt during the process of respiration in the cells to produce ATP. There are other important pathways to aging including the above mentioned ones, but the oxidative stress abbreviation comprising liver disease emerges on top. Age-related ROS rise is the primary cause of mitochondrial damage in the liver, which results in the imbalance of energy that eventually becomes NASH and NAFLD. Thus, the aforementioned process of living takes its toll and then we feel old, wondering how on earth it has happened [180]. In addition to this, liver mitochondria malfunction stops the liver from breaking down fats, something that already exists. Besides this, in elderly people or patients with NAFLD, the condition is significantly progressive, and the prognosis is even worse than in younger people [181]. Liver aging gets more help from telomere shortening and cellular senescence rather than anything else. Telomere shortening and senescence are the two main mechanisms that function in the aging of hepatocytes. As the hepatocytes get older, their telomeres are diminished, which causes a series of events resulting in genomic instability and cellular senescence that prevents liver regeneration and causes chronic liver diseases such as cirrhosis and hepatocellular carcinoma (HCC) [182]. Moreover, the sequestration of the cell implies that the inflammatory and fibrogenic substances are increasing that is making liver fibrosis and hepatic dysfunction worse [183]. What's more, aging -induced liver injury goes on a separate but important course and that has to do with deteriorating endothelial function of the liver. Changes over time in the liver sinusoidal endothelial cells (LSECs) such as a decrease of fenestration and an increase of oxidative stress are the main factors that affect hepatic microcirculation and, thus, cause liver fibrosis and inflammation [184]. Additionally, dysregulated nutrient sensing and altered metabolic pathways in the aging liver lead to a higher risk of metabolic liver diseases, further complicating the aging process [185]. Epigenetic changes and impaired proteostasis also play vital roles in liver aging. Age-related epigenetic modifications, such as DNA methylation and histone

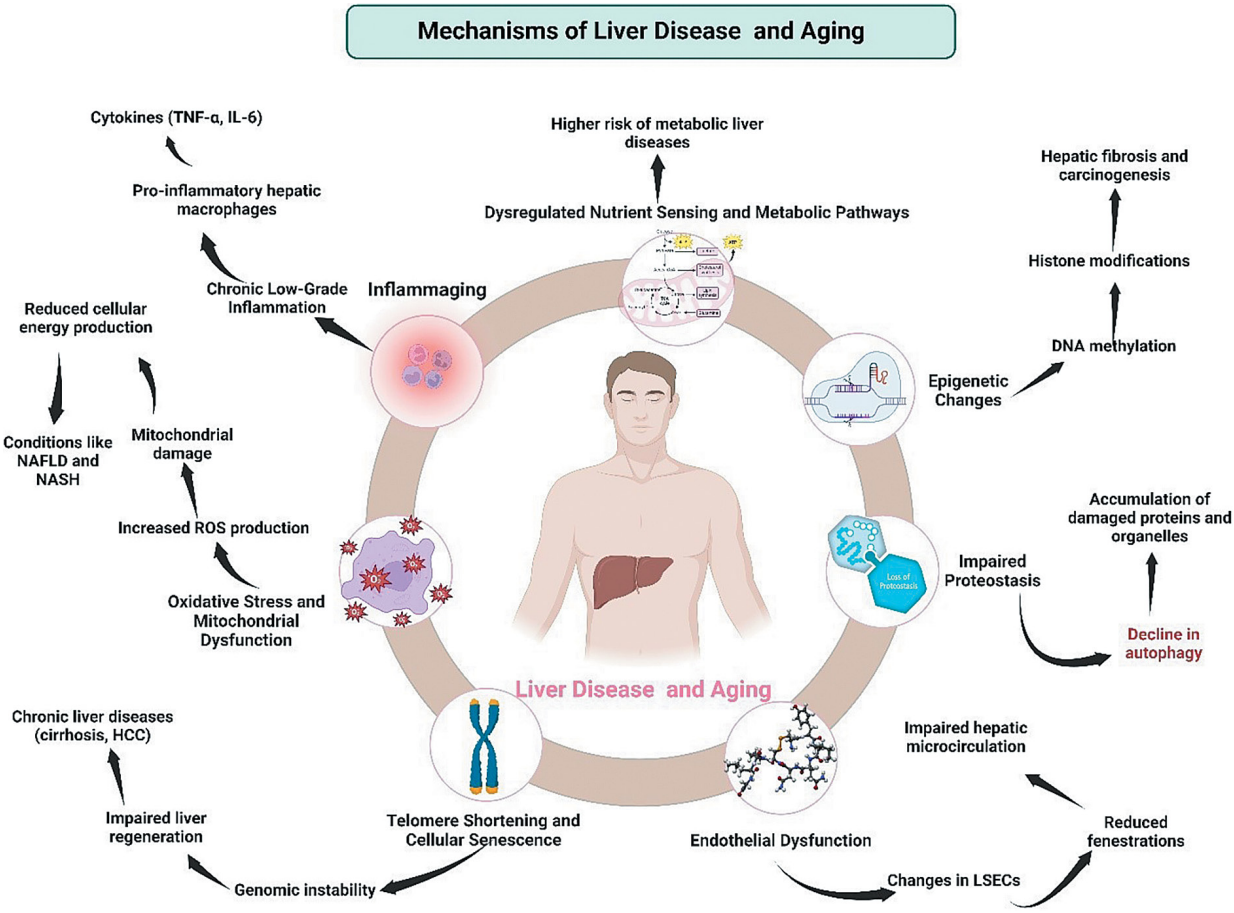

Figure 5. Mechanisms linking liver disease and aging

modifications, alter gene expression patterns, promoting hepatic fibrosis and carcinogenesis [186]. Additionally, the decline in autophagy and proteostasis with age leads to the accumulation of damaged proteins and organelles in liver cells, exacerbating liver disease progression and impairing liver function [187]. Figure 5 shows mechanisms linking liver disease and aging.

Conclusions

In conclusion, fast food consumption has profound effects on the aging process through multiple biological mechanisms. Chronic inflammation, oxidative stress, and mitochondrial dysfunction are central to the accelerated aging associated with cardiovascular diseases, metabolic disorders, cancer risks, liver disease and obesity. Public health strategies focused on reducing fast food consumption and promoting healthier dietary choices are essential to mitigate these adverse effects and enhance the quality of life and longevity in aging populations. Comprehensive nutrition education and policy changes are imperative to shift dietary patterns and reduce the global burden of fast food-related health issues.

REFERENCES

1. Bahadoran, Z., Mirmiran, P., Azizi, F. (2015). Fast food pattern and cardiometabolic disorders: A review of current studies. *Health Promotion Perspectives*, 5(4), 231–240. http://doi.org/10.15171/hpp.2015.028
2. Jaworowska, A., Blackham, T., Davies, I. G., Stevenson, L. (2013). Nutritional challenges and health implications of takeaway and fast food. *Nutrition Reviews*, 71(5), 310–318. https://doi.org/10.1111/nure.12031
3. Bowman, S.A., Vinyard, B.T. (2004). Fast food consumers VS non-fast food consumers: A comparison of their energy intakes, diet quality, and overweight status. *Journal of American College of Nutrition*, 23(2), 163–168.
4. Salvestrini, V., Sell, C., Lorenzini, A. (2019). Obesity may accelerate the aging process. *Frontiers in Endocrinology*, 10, Article 266. http://doi.org/10.3389/fendo.2019.00266
5. Garcia, G., Sunil, T. S., Hinojosa, P. (2012). The fast food and obesity link: Consumption patterns and severity of obesity. *Obesity Surgery*, 22(5), 810–818. http://doi.org/10.1007/s11695-012-0601-8
6. Zhang, Y., Fischer, K. E., Soto, V., Liu, Y., Sosnowska, D., Richardson, A. et al. (2015). Obesity-induced oxidative stress, accelerated functional decline with age and increased mortality in mice. *Archives of Biochemistry and Biophysics*, 576, 39–48. http://doi.org/10.1016/j.abb.2014.12.018
7. Fuhrman, J. (2018). The hidden dangers of fast and processed food. *American Journal of Lifestyle Medicine*, 12(5), 375–381. http://doi.org/10.1177/1559827618766483
8. Siddiqui, A., Anusha P, N. (2012). Deleterious effects of food habits in present era. *Journal of Allergy and Therapy*, 03(01), Article 1000114. http://doi.org/10.4172/2155-6121.1000114
9. Mozaffarian, D., Katan, M. B., Ascherio, A., Stampfer, M. J., Willett, W. C. (2006). Trans fatty acids and cardiovascular disease. *New England Journal of Medicine*, 354(15), 1601–1613. https://doi.org/10.1056/nejmra054035

10. Ganguly, R., Pierce, G. N. (2015). The toxicity of dietary trans fats. *Food and Chemical Toxicology*, 78, 170–176. https://doi.org/10.1016/j.fct.2015.02.004
11. Asghari, G., Yuzbashian, E., Mirmiran, P., Mahmoodi, B., Azizi, F. (2015). Fast food intake increases the incidence of metabolic syndrome in children and adolescents: Tehran lipid and glucose study. *PLoS ONE*, 10(10), Article e0139641. https://doi.org/10.1371/journal.pone.0139641
12. Isganaitis, E., Lustig, R. H. (2005). Fast food, central nervous system insulin resistance, and obesity. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 25(12), 2451–2462. https://doi.org/10.1161/01.atv.0000186208.06964.91
13. Pereira, M. A., Kartashov, A. I., Ebbeling, C. B., Van Horn, L., Slattery, M. L., Jacobs, D. R. et al. (2005). Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. *The Lancet*, 365(9453), 36–42. https://doi.org/10.1016/s0140-6736(04)17663-0
14. Lechleitner, M. (2011). Diabetes mellitus typ 2 und niere: Die rolle der insulinresistenz. *Journal für Hypertonie — Austrian Journal of Hypertension*, 15(1), 14–18.
15. Uranga, R. M., Keller, J. N. (2019). The complex interactions between obesity, metabolism and the brain. *Frontiers in Neuroscience*, 13, Article 513. http://doi.org/10.3389/fnins.2019.00513
16. Miranda-Díaz, A. G., García-Sánchez, A., Cardona-Muñoz, E. G. (2020). Foods with potential prooxidant and antioxidant effects involved in Parkinson's disease. *Oxidative Medicine and Cellular Longevity*, 1, Article 281454. https://doi.org/10.1155/2020/6281454
17. Gautam, S. (2023). Hazardous effect of fast food on the health status of children: A review. *The Pharma Innovation Journal*, 12(6), 4236–4240.
18. Francis, H., Stevenson, R. (2013). The longer-term impacts of Western diet on human cognition and the brain. *Appetite*, 63, 119–128. https://doi.org/10.1016/j.appet.2012.12.018
19. Alsabieh, M., Alqahtani, M., Altamimi, A., Albasha, A., Alsulaiman, A., Alkhamshi, A. et al. (2019). Fast food consumption and its associations with heart rate, blood pressure, cognitive function and quality of life. Pilot study. *Heliyon*, 5(5), Article e01566. https://doi.org/10.1016/j.heliyon.2019.e01566
20. Farooqui, Alwi, M., S. K. K. (2019). Fast food trend analysis by evaluating factors leading to customer satisfaction. *Journal of Marketing and Consumer Research*, 55, 54–70. https://doi.org/10.7176/jmcr
21. Stender, S., Dyerberg, J., Astrup, A. (2006). High levels of industrially produced trans fat in popular fast foods. *New England Journal of Medicine*, 354(15), 1650–1652. https://doi.org/10.1056/nejmc052959
22. Mohd Hatta, N., Ali, A., Yusof, A., Wan Zainal Shukri, W. H., Kamarudin, K. S. (2022). Socio-demographic determinants of fast-food consumption in malaysian young adults. *Malaysian Applied Biology*, 51(6), 65–72. https://doi.org/10.55230/mabjournal.v51i6.2392
23. Sun, Y., Liu, B., Snetselaar, L.G., Robinson, J.G., Wallace, R.B., Peterson, L.L. et al. (2019). Association of fried food consumption with all cause, cardiovascular, and cancer mortality: Prospective cohort study. *BMJ,* 364, Article k5420. https://doi.org/10.1136/bmj.k5420
24. Powell, L.M., Nguyen, B.T., Dietz, W.H. (2015). Energy and nutrient intake from pizza in the United States. *Pediatrics*, 135(2), 322–330. https://doi.org/10.1542/peds.2014-1844
25. Hur, I., Marquart, L.F., Reicks, M. (2014). Nutrient intakes among children and adolescents eating usual pizza products in school lunch compared with pizza meeting HealthierUS School Challenge criteria. *Journal of the Academy of Nutrition and Dietetics*, 114(5), 768–773. https://doi.org/10.1016/j.jand.2013.07.034
26. Malik, V. S., Schulze, M. B., Hu, F. B. (2006). Intake of sugar-sweetened beverages and weight gain: A systematic review1–3. *The American Journal of Clinical Nutrition*, 84(2), 274–288. https://doi.org/10.1093/ajcn/84.1.274
27. Ludwig, D. S. (2002). The glycemic index: Physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. *Jama*, 287(18), 2414–2423. https://doi.org/10.1001/jama.287.18.2414
28. Mahajan, S. A., Gothankar, J. S. (2020). Fast food consumption pattern amongst undergraduates of various disciplines of private colleges in Pune. *International Journal of Community Medicine and Public Health*, 7(2), 505. https://doi.org/10.18203/2394-6040.ijcmph20196069
29. Dunn, C. G., Gao, K. J., Soto, M. J., Bleich, S. N. (2021). Disparities in adult fast-food consumption in the U.S. by race and ethnicity, national health and nutrition examination survey 2017–2018. *American Journal of Preventive Medicine*, 61(4), e197–e201. https://doi.org/10.1016/j.amepre.2021.01.043
30. Powell, L. M., Nguyen, B. T. (2013). Fast-food and full-service restaurant consumption among children and adolescents. *JAMA Pediatrics*, 167(1), 14–20. https://doi.org/10.1001/jamapediatrics.2013.417
31. Pereira, M. A., Kartashov, A. I., Ebbeling, C. B., Van Horn, L., Slattery, M. L., Jacobs, D. R. et al., (2005). Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. *The Lancet*, 365(9453), 36–42. https://doi.org/10.1016/s0140-6736(04)17663-0
32. Anderson, B., Rafferty, A.P., Lyon-Callo, S., Fussman, C., Imes, G. (2011). Fast-food consumption and obesity among Michigan adults. *Preventing Chronic Disease*, 8(4), Article A71.
33. Wilcox, S., Sharpe, P. A., Turner-McGrievy, G., Granner, M., Baruth, M. (2013). Frequency of consumption at fast-food restaurants is associated with dietary intake in overweight and obese women recruited from financially disadvantaged neighborhoods. *Nutrition Research*, 33(8), 636–646. https://doi.org/10.1016/j.nutres.2013.05.007
34. Doak, C. M., Adair, L. S., Bentley, M., Monteiro, C., Popkin, B. M. (2004). The dual burden household and the nutrition transition paradox. *International Journal of Obesity*, 29(1), 129–136. https://doi.org/10.1038/sj.ijo.0802824
35. Beal, T., Morris, S. S., Tumilowicz, A. (2019). Global patterns of adolescent fruit, vegetable, carbonated soft drink, and fast-food consumption: A meta-analysis of global school-based student health surveys. *Food and Nutrition Bulletin*, 40(4), 444–459. https://doi.org/10.1177/0379572119848287
36. Yoon, S. R., Fogleman, S. K., Kim, H., Lee, K. E., Kim, O. Y. (2020). Breakfast intake effect on the association between fast-food consumption and the risk of obesity and dyslipidemia in Korean adults aged 20–39 years based on the Korea National Health and Nutrition Examination Survey IV 2013–2014. *Clinical Nutrition Research*, 9(2), 107–121. http://doi.org/10.7762/cnr.2020.9.2.107
37. Mohr, B., Dolgopolova, I., Roosen, J. (2019). The influence of sex and self-control on the efficacy of nudges in lowering the energy content of food during a fast food order. *Appetite*, 141, Article 104314. https://doi.org/10.1016/j.appet.2019.06.006
38. Matejowsky, T. (2010). Gender, fast food, and nutritional perspectives in contemporary Philippines. *Asia-Pacific Social Science Review*, 10(1), 1–20. https://doi.org/10.3860/apssr.v10i1.1578
39. Kevinli, Gultom, P. (2020). The effect of brand equity, price and location on consumer purchasing decisions on quality fried chicken Jalan Ismaliyah Medan *Jurnal Manajemen Bisnis Eka Prasetya,* 6(1), 44–53. https://doi.org/10.47663/jmbep.v6i1.36
40. Habib, F.Q., Dardak, R.A., Zakaria, S. (2011). Consumers' preference and consumption towards fast food: Evidences

from Malaysia. *Business and Management Quarterly Review*, 2(1), 14–27.
41. Saunders, P., Middleton, J. (2023). Exposure to multiple sources of accessible, cheap, energy dense fast foods in a deprived community. *The European Journal of Public Health*, 33(Supplement_2) https://doi.org/10.1093/eurpub/ckad160.1063
42. Kök Şan, C., Gökçay, G. F. (2023). Nutritional aspects of commercial infant and toddler food products sold in Turkey. *Nutrition and Health*, 2023, Article 02601060231194652. http://doi.org/10.1177/02601060231194652
43. Jindarattanaporn, N., Suya, I., Lorenzetti, L., Kantachuvesiri, S., Thamarangsi, T. (2023). Nutritional content of popular menu items from online food delivery applications in Bangkok, Thailand: Are they healthy? *International Journal of Environmental Research and Public Health*, 20(5), Article 3992. https://doi.org/10.3390/ijerph20053992
44. Bernstein, J. T., Christoforou, A. K., Flexner, N., L'Abbe, M. R. (2023). Comparing the nutritional composition of foods and beverages in the Canadian Nutrient File to a large representative database of Canadian prepackaged foods and beverages. *PLOS ONE*, 18(3), Article e0280028. https://doi.org/10.1371/journal.pone.0280028
45. Marshellina, C. J., Tejoyuwono, A. A. T., Irsan, A. (2023). Factors affecting patterns of fast food consumption in students of medical study program, Faculty of Medicine, Tanjungpura University. *Journal of Agromedicine and Medical Sciences*, 9(3), Article 146. https://doi.org/10.19184/ams.v9i3.41505
46. Rodríguez-Martín, N. M., Córdoba, P., Sarriá, B., Verardo, V., Pedroche, J., Alcalá-Santiago, Á. et al. (2023). Characterizing meat- and milk/dairy-like vegetarian foods and their counterparts based on nutrient profiling and food labels. *Foods*, 12(6), Article 1151. https://doi.org/10.3390/foods12061151
47. Mackay, S., Gontijo de Castro, T., Young, L., Shaw, G., Ni Mhurchu, C., Eyles, H. (2021). Energy, sodium, sugar, and saturated fat content of New Zealand fast-food products and meal combos in 2020. *Nutrients*, 13(11), Article 4010. https://doi.org/10.3390/nu13114010
48. Dunn, C. G., Soto, M. J., Hua, S. V., Keenan, E. A., Jaacks, L. M., Wolfson, J. A. et al. (2021). Availability and nutrient composition of vegetarian items at US fast-food restaurants. *Journal of the Academy of Nutrition and Dietetics*, 121(7), 1306–1311.e8. https://doi.org/10.1016/j.jand.2021.01.010
49. Maldonado-Pereira, L., Barnaba, C., de Los Campos, G., Medina-Meza, I. G. (2022). Evaluation of the nutritional quality of ultra-processed foods (ready to eat + fast food): Fatty acid composition. *Journal of Food Science*, 87(8), 3659–3676. https://doi.org/10.1111/1750-3841.16235
50. Martin, C., Hoy, M. K., Murayi, T., Moshfegh, A. (2020). Nutrient intake and dietary quality among children and adolescents by fast food consumption status: What we eat in America, NHANES2013–2016. *Current Developments in Nutrition*, 4, Article nzaa043_086. https://doi.org/10.1093/cdn/nzaa043_086
51. Úbeda, N., González, M. P., Achón, M., García-González, Á., Ballestero-Fernández, C., Fajardo, V. et al. (2023). Nutritional composition of breakfast in children and adolescents with and without celiac disease in Spain — Role of gluten-free commercial products. *Nutrients*, 15(10), Article 2368. https://doi.org/10.3390/nu15102368
52. Gul, F.H. (2023). Nutrient profile of commercially packaged food products in Türkiye. *The Eurasia Proceedings of Science Technology Engineering and Mathematics*, 25, 163–167.
53. Taniim, T., Rahlaa, Dr. F., Mohibbullah, Sultana, S., Faruqui, M. K., Md Rizwan, A. A. et al. (2023). Level of knowledge on the effect of fast foods on health among young hypertensive patients in Bangladesh. *International Journal of Multidisciplinary Research and Growth Evaluation*, 4(2), 521–524. https://doi.org/10.54660/.ijmrge.2023.4.2.521-524
54. Nyangoya, D., Attoni, R. (2023). Fast food and its effects among teenagers in the Municipal of Cachoeiro De Itapemirim-Espirito Santo, Brazil. *International Journal of Research and Innovation in Social Science*, VII(IX), 308–326. https://doi.org/10.47772/ijriss.2023.70926
55. Parvin, S., Kabir, R., Parsa, A. D., Sivasubramanian, M. (2023). An investigation into the fast-food consumption habits of public health and nursing students at the University of Sunderland in London, UK. *Libyan International Medical University Journal*, 08(02), 063–069. https://doi.org/10.1055/s-0043-1776398
56. Baskati, A., Pareek, N. (2023). Healthy eating: Challenges of the present era. *International Journal of Scientific Research in Engineering and Management*, 07(12), 1–11. https://doi.org/10.55041/ijsrem27670
57. Wijaya, N. V., Dahliah, D., Pancawati, E. (2023). The impact of junk food eating habits on body weight. *OPSearch: American Journal of Open Research*, 2(7), 567–573. https://doi.org/10.58811/opsearch.v2i6.62
58. Ramadani, F., Jannah, F. (2023). The relationship between fast food consumption and the incidence of obesity in students of SMA Negeri 3 Subang and its review according to Islamic views. *Junior Medical Journal*, 1(7), 923–931.
59. El-hasry, R.T., Sadek Abd-El Hameed, H., Mohamed Sobhy ElSayed, D. (2023). Perception of mothers regarding effect of fast food on preschool children's health status. *Journal of Nursing Science Benha University*, 4(2), 23–36. https://doi.org/10.21608/jnsbu.2023.306047
60. Pratheepkumar, S., Laith Hamdan, Isa Khashiev, Jayadevan Sreedharan. (2023). Practice of fast food consumption among university students and variables associated with the practice. *Journal of Medical and Health Studies*, 4(5), 06–13. https://doi.org/10.32996/jmhs.2023.4.5.2
61. AlTamimi, J. Z., AlFaris, N. A., Alshwaiyat, N. M., Alkhalidy, H., AlKehayez, N. M., Alsemari, M. A. et al. (2023). Prevalence of fast-food intake among a multi-ethnic population of middle-aged men and connection with sociodemographic factors and obesity. *Medicine*, 102(15), Article e33555. https://doi.org/10.1097/md.0000000000033555
62. Fitrianti, D., Mardhiati, R., Novianus, C. (2023). Determinants of fast food consumption behavior in adolescents in the city of *Jakarta. Jurnal Riset Kesehatan*, 12(1), 1–7. https://doi.org/10.31983/jrk.v12i1.9190
63. Abrahamsson, S., Bütikofer, A., Karbownik, K. (2023). Swallow this: Childhood and adolescent exposure to fast food restaurants, BMI, and cognitive ability. Discussion paper series. IZA Institute of Labor Economics. IZA DP No. 16109. Retrieved from https://docs.iza.org/dp16109.pdf Accessed Oktober 11, 2024
64. Alanazi, A. M. L., Alanazi, A. M. L., Alanazi, S. M. L., Alanazi, S. S. M. (2023). Impact of social media on fast food consumption and increased bad nutritional habits: Systematic review. *Saudi Journal of Medical and Pharmaceutical Sciences*, 9(12), 839–844. https://doi.org/10.36348/sjmps.2023.v09i12.010
65. Pushkar, K., Kaushik, S. K., Nagarjuna, P., Mukherjee, G., Teli, P., Yadav, A. K. (2022). Fast-food culture — Prevalence, pattern, and preference trends and its association with body mass index of medical students. *Journal of Marine Medical Society*, 25(1), 37–42. https://doi.org/10.4103/jmms.jmms_36_22
66. Kasmarini, F., Andriani, E., Sabrina, S. (2023). The relationship between fast food consumption, pocket money, and quality of sleep with over-nutrition in adolescents. *Jurnal Gizi Prima (Prime Nutrition Journal)*, 8(2), Article 116. https://doi.org/10.32807/jgp.v8i2.400

67. Lestari, H., Handayani, L., Nurfadilah H, S. (2023). Relationship between knowledge and fast food with obesity in adolescents. *Community Research of Epidemiology (CORE)*, 93–100. https://doi.org/10.24252/corejournal.vi.38353
68. Mendonca, V. (2023). Modern food habits and its impact on human health. *International Journal of Innovative Research in Engineering and Management*, 10(4), 182–185. https://doi.org/10.55524/ijirem.2023.10.4.24
69. Kagathara, N., Patel, M., Satapara, N., Kagathara, J., Padaliya, D., Gandhi, R. (2023). A study on association between dietary eating habits and mental health among medical college students of western city of Gujarat. *International Journal of Scientific Research*, 12(05), 59–61. https://doi.org/10.36106/ijsr/0908002
70. Saragih, W.N., Angkat, A.H., Gizi, J. (2023). The effect of nutrition counseling with animation media about obesity on knowledge and habits of fast food consumption in class VIII obese adolescents at SMP Negeri 1 Lubuk Pakam. *Indonesian Journal of Interdisciplinary Research in Science and Technology*, 1(9), 791–804. https://doi.org/10.55927/marcopolo.v1i9.6577
71. Fraser, L. K., Clarke, G. P., Cade, J. E., Edwards, K. L. (2012). Fast food and obesity: A spatial analysis in a large United Kingdom population of children aged 13–15. *American Journal of Preventive Medicine*, 42(5), e77–e85. https://doi.org/10.1016/j.amepre.2012.02.007
72. Bowman, S. A., Vinyard, B. T. (2004). Fast food consumption of U.S. adults: Impact on energy and nutrient intakes and overweight status. *Journal of the American College of Nutrition*, 23(2), 163–168. https://doi.org/10.1080/07315724.2004.10719357
73. Rouhani, M.H., Mirseifinezhad, M., Omrani, N., Esmaillzadeh, A., Azadbakht, L. (2012). Fast food consumption, quality of diet, and obesity among Isfahanian adolescent girls. *Journal of Obesity*, 2012, Article 597924. https://doi.org/10.1155/2012/597924
74. Rosenheck, R.E. (2008). Fast food consumption and increased caloric intake: A systematic review of a trajectory towards weight gain and obesity risk. *Obesity Reviews*, 9(6), 535–547. https://doi.org/10.1111/j.1467-789X.2008.00477.x
75. Fraser, L. K., Clarke, G. P., Cade, J. E., Edwards, K. L. (2012). Fast food and obesity: A spatial analysis in a large United Kingdom population of children aged 13–15. *American Journal of Preventive Medicine,* 42(5), e77-e85. https://doi.org/10.1016/j.amepre.2012.02.007
76. Bowman, S. A., Gortmaker, S. L., Ebbeling, C. B., Pereira, M. A., Ludwig, D. S. (2004). Effects of fast-food consumption on energy intake and diet quality among children in a national household surve. *Pediatrics*, 113(1), 112–118. https://doi.org/10.1542/peds.113.1.112
77. Niemeier, H. M., Raynor, H. A., Lloyd-Richardson, E. E., Rogers, M. L., Wing, R. R. (2006). Fast food consumption and breakfast skipping: Predictors of weight gain from adolescence to adulthood in a nationally representative sample. *Journal of Adolescent Health*, 39(6), 842–849. https://doi.org/10.1016/j.jadohealth.2006.07.001
78. Giezenaar, C., Chapman, I., Luscombe-Marsh, N., Feinle-Bisset, C., Horowitz, M., Soenen, S. (2016). Ageing is associated with decreases in appetite and energy intake — A meta-analysis in healthy adults. *Nutrients*, 8(1), Article 28. https://doi.org/10.3390/nu8010028
79. Duffey, K. J., Gordon-Larsen, P., Steffen, L. M., Jacobs, D. R., Popkin, B. M. (2009). Regular consumption from fast food establishments relative to other restaurants is differentially associated with metabolic outcomes in young adults. *The Journal of Nutrition*, 139(11), 2113–2118. https://doi.org/10.3945/jn.109.109520
80. Vikraman, S., Fryar, C. D., Ogden, C. L. (2015). Caloric intake from fast food among children and adolescents in the United States, 2011–2012. *NCHS Data Brief*, 213, 1–8.
81. Mattson, M. P., Longo, V. D., Harvie, M. (2017). Impact of intermittent fasting on health and disease processes. *Ageing Research Reviews*, 39, 46–58. https://doi.org/10.1016/j.arr.2016.10.005
82. Poutahidis, T., Kleinewietfeld, M., Smillie, C., Levkovich, T., Perrotta, A., Bhela, S. et al. (2013). Microbial reprogramming inhibits Western diet-associated obesity. *PLoS ONE*, 8(7), Article e68596. https://doi.org/10.1371/journal.pone.0068596
83. Lin, P.-Y., Romsos, D. R., Leveille, G. A. (1977). Food intake, body weight gain, and body composition of the young obese (ob/ob) mouse. *The Journal of Nutrition*, 107(9), 1715–1723. https://doi.org/10.1093/jn/107.9.1715
84. Zatterale, F., Longo, M., Naderi, J., Raciti, G.A., Desiderio, A., Miele, C. et al. (2020). Chronic adipose tissue inflammation linking obesity to insulin resistance and type 2 diabetes. *Frontiers in Physiology*, 10, Article 1607. https://doi.org/10.3389/fphys.2019.01607
85. Furukawa, S., Fujita, T., Shimabukuro, M., Iwaki, M., Yamada, Y., Nakajima, Y. et al. (2004). Increased oxidative stress in obesity and its impact on metabolic syndrome. *The Journal of Clinical Investigation*, 114(12), 1752–1761. https://doi.org/10.1172/JCI21625
86. Gupta, D., Krueger, C.B., Lastra, G. (2012). Over-nutrition, obesity and insulin resistance in the development of β-cell dysfunction. *Current Diabetes Reviews*, 8(2), 76–83. https://doi.org/10.2174/157339912799424564
87. Carter, S., Caron, A., Richard, D., Picard, F. (2013). Role of leptin resistance in the development of obesity in older patients. *Clinical Interventions in Aging*, 8, 829–844. https://doi.org/10.2147/CIA.S36367
88. Maldonado, E., Morales-Pison, S., Urbina, F., Solari, A. (2023). Aging hallmarks and the role of oxidative stress. *Antioxidants*, 12(3), Article 651. https://doi.org/10.3390/antiox12030651
89. Villareal, D.T. (2023). Obesity and accelerated aging. *The Journal of Nutrition, Health and Aging*, 27(5), 312–313. https://doi.org/10.1007/s12603-023-1922-0
90. Hamczyk, M.R., del Campo, L., Andrés, V. (2018). Aging in the cardiovascular system: Lessons from Hutchinson-Gilford progeria syndrome. *Annual Review of Physiology*, 80(1), 27–48. https://doi.org/10.1146/annurev-physiol-021317–121454
91. Frasca, D., Blomberg, B.B., Paganelli, R. (2017). Aging, obesity, and inflammatory age-related diseases. *Frontiers in Immunology*, 8, Article 1745. https://doi.org/10.3389/fimmu.2017.01745
92. Jacobson, M. F., Havas, S., McCarter, R. (2013). Changes in sodium levels in processed and restaurant foods, 2005 to 2011. *JAMA Internal Medicine*, 173(14), 1285–1291. https://doi.org/10.1001/jamainternmed.2013.6154
93. Moshfegh, A.J., Holden, J.M., Cogswell, M.E., Kuklina, E.V., Patel, S.M., Gunn, J.P. et al. (2012). Vital signs: Food categories contributing the most to sodium consumption — United States, 2007–2008. Morbidity and Mortality Weekly Report, 61(5), 92–98.
94. Ahuja, J. K. C., Wasswa-Kintu, S., Haytowitz, D. B., Daniel, M., Thomas, R., Showell, B. et al. (2015). Sodium content of popular commercially processed and restaurant foods in the United States. *Preventive Medicine Reports*, 2, 962–967. https://doi.org/10.1016/j.pmedr.2015.11.003
95. Puckerin, K, (2016). The link between the consumption of fast food and their cardiovascular risks in university students. Doctoral dissertation. The University of the West Indies, 2016. Retrieved from https://uwispace.sta.uwi.edu/items/0972978a-2a65-4484-9b19-88639f0f6985 Accessed Oktober 16, 2024

96. Lane, M. M., Lotfaliany, M., Forbes, M., Loughman, A., Rocks, T., O'Neil, A. et al. (2022). Higher ultra-processed food consumption is associated with greater high-sensitivity C-reactive protein concentration in adults: Cross-sectional results from the Melbourne Collaborative Cohort Study. *Nutrients*, 14(16), Article 3309. https://doi.org/10.3390/nu14163309
97. Yeo, R., Yoon, S. R., Kim, O. Y. (2017). The association between food group consumption patterns and early metabolic syndrome risk in non-diabetic healthy people. *Clinical Nutrition Research*, 6(3), Article 172. https://doi.org/10.7762/cnr.2017.6.3.172
98. Afshan, A., Salgar, V., Sugoor, M., Deshpande, A. (2014). The impact of fast food consumption on the lipid profile, BMI and blood sugar levels. *International Journal of Bioassays*, 3, 3221–3223.
99. Vercammen, K. A., Moran, A. J., McClain, A. C., Thorndike, A. N., Fulay, A. P., Rimm, E. B. (2019). Food security and 10-year cardiovascular disease risk among U.S. adults. *American Journal of Preventive Medicine*, 56(5), 689–697. https://doi.org/10.1016/j.amepre.2018.11.016
100. Nadeem, M., Rehman, H. U., Mustafa, M. O. H. (2022). Association between fast-food consumption and the risk of developing coronary heart disease among the male population in Peshawar. *International Journal of Health Sciences*, 6(S9), 5017–5024. https://doi.org/10.53730/ijhs.v6ns9.14584
101. Whitton, C., Rebello, S. A., Lee, J., Tai, E. S., van Dam, R. M. (2018). A healthy asian a posteriori dietary pattern correlates with a priori dietary patterns and is associated with cardiovascular disease risk factors in a multiethnic asian population. *The Journal of Nutrition*, 148(4), 616–623. https://doi.org/10.1093/jn/nxy016
102. Ferrara, L., Pacioni, D., Vitolo, G., Staiano, L., Riccio, E., Gaetano, G. (2008). Fast food versus slow food and hypertension control. *Current Hypertension Reviews*, 4(1), 30–35. https://doi.org/10.2174/157340208783497219
103. Basu, A., Shay, C. M., Colangelo, L., Jacobs, D. R., Van Horn, L. (2014). Abstract P177: Higher frequency of fast food consumption is associated with lower likelihood of meeting dietary reference intakes (DRIs): Findings from the coronary artery risk development in young adults (CARDIA) Study. *Circulation*, 129(Suppl_1). https://doi.org/10.1161/circ.129.suppl_1.p177
104. Bahadoran, Z., Mirmiran, P., Golzarand, M., Hosseini-Esfahani, F., Azizi, F. (2012). Fast food consumption in Iranian adults; dietary intake and cardiovascular risk factors: Tehran lipid and glucose study. *Archives of Iranian Medicine*, 15(6), 346–351.
105. Odegaard, A. O., Koh, W. P., Yuan, J.-M., Gross, M. D., Pereira, M. A. (2012). Western-style fast food intake and cardiometabolic risk in an Eastern country. *Circulation*, 126(2), 182–188, 2012. https://doi.org/10.1161/circulationaha.111.084004
106. Sohouli, M.H., Lari, A. (2020). The association between fast food consumption with cardiovascular diseases risk factors in patients with diabetic nephropathy. *Qom University of Medical Sciences Journal*, 4(2), 57–67. (In Persian)
107. Schmidt, J. M., Epel, E. S., Jacobs, L. M., Mason, A. E., Parrett, B., Pickett, A. M. et al. (2023). Controlled trial of a workplace sales ban on sugar-sweetened beverages. *Public Health Nutrition*, 26(10), 2130–2138. https://doi.org/10.1017/s1368980023001386
108. Barcena, M. L., Aslam, M., Pozdniakova, S., Norman, K., Ladilov, Y. (2022). Cardiovascular inflammageing: Mechanisms and translational aspects. *Cells*, 11(6), Article 1010. https://doi.org/10.3390/cells11061010
109. Fossel, M., Bean, J., Khera, N., Kolonin, M. G. (2022). A unified model of age-related cardiovascular disease. *Biology*, 11(12), Article 1768. https://doi.org/10.3390/biology11121768
110. Pietri, P., Stefanadis, C. (2021). Cardiovascular aging and longevity: JACC state-of-the-art review. *Journal of the American College of Cardiology*, 77(2), 189–204. https://doi.org/10.1016/j.jacc.2020.11.023
111. Sermersheim, M.A., Park, K.H., Gumpper, K., Adesanya, T.M.A., Song, K., Tan, T. et al. (2017). MicroRNA regulation of autophagy in cardiovascular disease. Frontiers in bioscience (Landmark edition), 22(1), 48–65. https://doi.org/10.2741/4471
112. Liberale, L., Kraler, S., Camici, G. G., Lüscher, T. F. (2020). Ageing and longevity genes in cardiovascular diseases. *Basic and Clinical Pharmacology and Toxicology*, 127(2), 120–131. https://doi.org/10.1111/bcpt.13426
113. Li, M., Feng, M. Y., Feng, Z. H., Li, J., Zhang, X., Gao, F. (2023). Exercise promotes healthy cardiovascular aging. *Sheng Li Xue Bao: Acta Physiologica Sinica*, 75(6), 887–902.
114. Clayton, Z. S., Craighead, D. H., Darvish, S., Coppock, M., Ludwig, K. R., Brunt, V. E. et al. (2022). Promoting healthy cardiovascular aging: Emerging topics. *The Journal of Cardiovascular Aging*, 2(4), Article 43. https://doi.org/10.20517/jca.2022.27
115. Greenwood, D. C., Threapleton, D. E., Evans, C. E. L., Cleghorn, C. L., Nykjaer, C., Woodhead, C. et al. (2013). Glycemic index, glycemic load, carbohydrates, and type 2 diabetes: Systematic review and dose-response meta-analysis of prospective studies. *Diabetes Care*, 36(12), 4166–4171. https://doi.org/10.2337/dc13-0325
116. Eckel, R. H., Grundy, S. M., Zimmet, P. Z. (2005). The metabolic syndrome. *The Lancet*, 365(9468), 1415–1428. https://doi.org/10.1016/s0140-6736(05)66378-7
117. Malik, V. S., Hu, F. B. (2019). Sugar-sweetened beverages and cardiometabolic health: An update of the evidence. *Nutrients*, 11(8), Article 1840. https://doi.org/10.3390/nu11081840
118. Jaworowska, A., Blackham, T., Davies, I. G., Stevenson, L. (2013). Nutritional challenges and health implications of takeaway and fast food. *Nutrition Reviews*, 71(5), 310–318. https://doi.org/10.1111/nure.12031
119. Yadav, U. C., Rani, V., Deep, G., Singh, R. K., Palle, K. (2016). Oxidative stress in metabolic disorders: Pathogenesis, prevention, and therapeutics. *Oxidative Medicine and Cellular Longevity*, 2016, Article 9137629. https://doi.org/10.1155/2016/9137629
120. Saad, M.J.A., Santos, A., Prada, P.O. (2016). Linking gut microbiota and inflammation to obesity and insulin resistance. *Physiology*, 31(4), 283–293. https://doi.org/10.1152/physiol.00041.2015
121. Ridaura, V. K., Faith, J. J., Rey, F. E., Cheng, J., Duncan, A. E., Kau, A. L. et al. (2013). Gut microbiota from twins discordant for obesity modulate metabolism in mice. *Science*, 341(6150), Article 1241214. https://doi.org/10.1126/science.1241214
122. Hariri, N., Thibault, L. (2010). High-fat diet-induced obesity in animal models. *Nutrition Research Reviews*, 23(2), 270–299. https://doi.org/10.1017/s0954422410000168
123. Odegaard, A. O., Jacobs, Jr, D. R., Van Wagner, L. B., Pereira, M. A. (2022). Levels of abdominal adipose tissue and metabolic-associated fatty liver disease (MAFLD) in middle age according to average fast-food intake over the preceding 25 years: The CARDIA Study. *The American Journal of Clinical Nutrition*, 116(1), 255–262. https://doi.org/10.1093/ajcn/nqac079
124. Vellapandian, C., Singh, A. (2023). High risk of metabolic complications due to high consumption of processed foods. *Current Nutrition and Food Science*, 19(3), 198–208. https://doi.org/10.2174/1573401318666220622162038
125. Figge, A., Sydor, S., Wenning, C., Manka, P., Assmuth, S., Vilchez-Vargas, R. et al. (2021). Gender and gut microbiota

composition determine hepatic bile acid, metabolic and inflammatory response to a single fast-food meal in healthy adults. *Clinical Nutrition*, 40(5), 2609–2619. https://doi.org/10.1016/j.clnu.2021.04.008
126. Spinelli, R., Parrillo, L., Longo, M., Florese, P., Desiderio, A., Zatterale, F. et al. (2020). Molecular basis of ageing in chronic metabolic diseases. *Journal of Endocrinological Investigation*, 43(10), 1373–1389. https://doi.org/10.1007/s40618-020-01255-z
127. Boccardi, V., Marano, L. (2024). Aging, cancer, and inflammation: The telomerase connection. *International Journal of Molecular Science*, 25(15), Article 8542. https://doi.org/10.3390/ijms25158542
128. Frasca, D., Pallikkuth, S., Pahwa, S. (2022). Effects of aging on metabolic characteristics of human B cells. *JAIDS Journal of Acquired Immune Deficiency Syndromes*, 89(S1), S23–S28. https://doi.org/10.1097/qai.0000000000002860
129. Wordsworth, J., Yde Nielsen, P., Fielder, E., Chandrasegaran, S., Shanley, D. (2023). Metabolic slowdown as the proximal cause of ageing and death. *bioRxiv,* 08(01), Article 551537 https://doi.org/10.1101/2023.08.01.551537
130. Kitada, M., Koya, D. (2021). Autophagy in metabolic disease and ageing. *Nature Reviews Endocrinology*, 17, 647–661.
131. Hamrick, M. W., Stranahan, A. M. (2020). Metabolic regulation of aging and age-related disease. *Ageing Research Reviews*, 64, Article 101175. https://doi.org/10.1016/j.arr.2020.101175
132. Liu, T., Xu, Y., Yi, C.-X., Tong, Q., Cai, D. (2021). The hypothalamus for whole-body physiology: From metabolism to aging. *Protein and Cell*, 13(6), 394–421. https://doi.org/10.1007/s13238-021-00834-x
133. Ottens, F., Franz, A., Hoppe, T. (2021). Build-UPS and breakdowns: Metabolism impacts on proteostasis and aging. *Cell Death and Differentiation*, 28(2), 505–521. https://doi.org/10.1038/s41418-020-00682-y
134. Tyagi, A., Mirita, C., Taher, N., Shah, I., Moeller, E., Tyagi, A. et al. (2020). Metabolic syndrome exacerbates amyloid pathology in a comorbid Alzheimer's mouse model. *Biochimica et Biophysica Acta (BBA) — Molecular Basis of Disease*, 1866(10), Article 165849. https://doi.org/10.1016/j.bbadis.2020.165849
135. Monge, A., Lajous, M. (2018). Ultra-processed foods and cancer. *BMJ,* 360, Article k599. https://doi.org/10.1136/bmj.k599
136. Bouvard, V., Loomis, D., Guyton, K. Z., Grosse, Y., Ghissassi, F. E., Benbrahim-Tallaa, L. et al. (2015). Carcinogenicity of consumption of red and processed meat. *The Lancet Oncology*, 16(16), 1599–1600. https://doi.org/10.1016/s1470-2045(15)00444-1
137. Loh, Y. H., Jakszyn, P., Luben, R. N., Mulligan, A. A., Mitrou, P. N., Khaw, K.-T. (2011). N-nitroso compounds and cancer incidence: The European Prospective Investigation into Cancer and Nutrition (EPIC) — Norfolk study. *The American Journal of Clinical Nutrition*, 93(5), 1053–1061. https://doi.org/10.3945/ajcn.111.012377
138. Boyd, N., Martin, L., Noffel, M., Lockwood, G., Trichler, D. (1993). A meta-analysis of studies of dietary fat and breast cancer risk. *British Journal of Cancer*, 68(3), 627–636. https://doi.org/10.1038/bjc.1993.398
139. Rock, C. L., Thomson, C., Gansler, T., Gapstur, S. M., McCullough, M. L., Patel, A. V. et al. (2020). American Cancer Society guideline for diet and physical activity for cancer prevention. *CA: A Cancer Journal for Clinicians*, 70(4), 245–271. https://doi.org/10.3322/caac.21591
140. Huybrechts, I., Romieu, I., Kandpur, N., Katsikari, K., Torres-Mejia, G., Sanchez, G. I. et al. (2020). Ultra-processed food consumption and breast cancer risk. *Proceedings of the Nutrition Society*, 79(OCE2), E182. https://doi.org/10.1017/s0029665120001305
141. Papadimitriou, N., Markozannes, G., Kanellopoulou, A., Critselis, E., Alhardan, S., Karafousia, V. et al. (2021). An umbrella review of the evidence associating diet and cancer risk at 11 anatomical sites. *Nature Communications*, 12(1), Article 4579. https://doi.org/10.1038/s41467-021-24861-8
142. Jafari, F., Yarmand, S., Nouri, M., Nejad, E. T., Ramezani, A., Sohrabi, Z. et al. (2022). Ultra-processed food intake and risk of colorectal cancer: A matched case-control study. *Nutrition and Cancer*, 75(2), 532–541. https://doi.org/10.1080/01635581.2022.2125990
143. Farvid, M. S., Sidahmed, E., Spence, N. D., Mante Angua, K., Rosner, B. A., Barnett, J. B. (2021). Consumption of red meat and processed meat and cancer incidence: A systematic review and meta-analysis of prospective studies. *European Journal of Epidemiology*, 36(9), 937–951. https://doi.org/10.1007/s10654-021-00741-9
144. Wang, L., Du, M., Wang, K., Khandpur, N., Rossato, S. L., Drouin-Chartier, J.-P. et al. (2022). Association of ultra-processed food consumption with colorectal cancer risk among men and women: Results from three prospective US cohort studies. *BMJ*, 378, Article e068921. https://doi.org/10.1136/bmj-2021-068921
145. Bevel, M., Tsai, M.-H., Parham, A., Andrzejak, S., Jones, S. R., Moore, J. X. (2023). Abstract 6483: The association between food deserts, food swamps, and obesity-related cancer mortality in the United States: The new epidemic. *Cancer Research*, 83(7_Suppl), 6483–6483. https://doi.org/10.1158/1538-7445.am2023–6483
146. Zhong, G.-C., Zhu, Q., Gong, J.-P., Cai, D., Hu, J.-J., Dai, X. et al. (2022). Fried food consumption and the risk of pancreatic cancer: A large prospective multicenter study. *Frontiers in Nutrition*, 9, Article 889303. https://doi.org/10.3389/fnut.2022.889303
147. Arya, S. (2020). Fast food consumption and anthropometric risk factors among college girl students. *Indian Journal of Pure and Applied Biosciences*, 8(1), 351–359. https://doi.org/10.18782/2582-2845.7772
148. Khong, T. M. T., Bui, T. T., Kang, H.-Y., Lee, J., Park, E., Oh, J.-K. (2024). Cancer risk according to fasting blood glucose trajectories: A population-based cohort study. *BMJ Open Diabetes Research and Care*, 12(1), Article e003696. https://doi.org/10.1136/bmjdrc-2023-003696
149. Wu, Y., Wang, L., Zhu, J., Gao, L., Wang, Y. (2021). Growing fast food consumption and obesity in Asia: Challenges and implications. *Social Science and Medicine*, 269, Article 113601. https://doi.org/10.1016/j.socscimed.2020.113601
150. Li, L., Sun, N., Zhang, L., Xu, G., Liu, J., Hu, J. et al. (2020). Fast food consumption among young adolescents aged 12–15 years in 54 low-and middle-income countries. *Global Health Action*, 13(1), Article 1795438. https://doi.org/10.1080/16549716.2020.1795438
151. Bohlouli, J., Moravejolahkami, A. R., Ganjali Dashti, M., Balouch Zehi, Z., Hojjati Kermani, M. A., Borzoo-Isfahani, M. et al. (2021). COVID-19 and fast foods consumption: A review. *International Journal of Food Properties*, 24(1), 203–209. https://doi.org/10.1080/10942912.2021.1873364
152. Papier, K., Fensom, G. K., Knuppel, A., Appleby, P. N., Tong, T. Y. N., Schmidt, J. A. et al (2021). Meat consumption and risk of 25 common conditions: Outcome-wide analyses in 475,000 men and women in the UK Biobank study. *BMC Medicine*, 19(1), Article 53. https://doi.org/10.1186/s12916-021-01922-9
153. Kim, Y. J., Oh, C.-M., Park, S. K., Jung, J. Y., Kim, M.-H., Ha, E. et al. (2022). Fasting blood glucose and risk of incident pancreatic cancer. *PLOS ONE*, 17(10), Article e0274195. https://doi.org/10.1371/journal.pone.0274195
154. Brandhorst, S. (2021). Fasting and fasting-mimicking diets for chemotherapy augmentation. GeroScience, 43(3), 1201–1216. https://doi.org/10.1007/s11357-020-00317-7

155. Aveta, A., Cacciapuoti, C., Barone, B., Di Zazzo, E., Del Giudice, F., Maggi, M. et al. (2022). The impact of meat intake on bladder cancer incidence: Is it really a relevant risk? *Cancers*, 14(19), Article 4775. https://doi.org/10.3390/cancers14194775
156. Givens, D. I. (2020). Dairy foods and the risk of cancer. Chapter in a book: Milk and Dairy Foods. Academic Press, 2020. https://doi.org/10.1016/B978-0-12-815603-2.00015-2
157. Haj-Mirzaeian, A., Afshari, K., Abdolghaffari, A.H. (2021). Aging and Cancer Prognosis. Chapter in a book: Cancer Immunology Bench to Bedside Immunotherapy of Cancers. Springer Nature Switzerland, 2021. https://doi.org/10.1007/978-3-030-50287-4_24
158. Patel, J., Baptiste, B. A., Kim, E., Hussain, M., Croteau, D. L., Bohr, V. A. (2020). DNA damage and mitochondria in cancer and aging. *Carcinogenesis*, 41(12), 1625–1634. https://doi.org/10.1093/carcin/bgaa114
159. Smith, A. L. M., Whitehall, J. C., Greaves, L. C. (2022). Mitochondrial DNA mutations in ageing and cancer. *Molecular Oncology*, 16(18), 3276–3294. https://doi.org/10.1002/1878-0261.13291
160. Carlberg, C., Velleuer, E. (2021). Aging and cancer. Chapter in a book: Cancer Biology: How Science Works. Springer, Cham. 2021.
161. Berben, L., Floris, G., Wildiers, H., Hatse, S. (2021). Cancer and aging: Two tightly interconnected biological processes. *Cancers*, 13(6), Article 1400. https://doi.org/10.3390/cancers13061400
162. Drapela, S., Ilter, D., Gomes, A. P. (2022). Metabolic reprogramming: A bridge between aging and tumorigenesis. *Molecular Oncology*, 16(18), 3295–3318. https://doi.org/10.1002/1878-0261.13261
163. Chen, X.-Q., Shen, T., Fang, S.-J., Sun, X.-M., Li, G.-Y., Li, Y.-F. (2023). Protein homeostasis in aging and cancer. *Frontiers in Cell and Developmental Biology*, 11, Article 1143532. https://doi.org/10.3389/fcell.2023.1143532
164. Havas, A., Yin, S., Adams, P. D. (2022). The role of aging in cancer. *Molecular Oncology*, 16(18), 3213–3219. https://doi.org/10.1002/1878-0261.13302
165. Tavakoli, H.R., Rahmati-Najarkolaei, F., Malkami, A., Dizavi, A.R. (2018). The relation between fast food consumption and non-alcoholic fatty liver: A case-control study. *Iranian Journal of Endocrinology and Metabolism*, 20(1), 22–30. (In Persian)
166. Mohammadi, F.D., Vazirinejad, R., Rezaeian, M., Vazirinejad, E., Bastam, D., Ahmadinia, H. et al. (2019). Fast food consumption and the risk of non-alcoholic fatty liver in adults: A community-based case-control study. *Journal of Occupational Health and Epidemiology*, 8(4), 176–184. https://doi.org/10.29252/johe.8.4.176
167. Marchesini, G., Ridolfi, V., Nepoti, V. (2008). Hepatotoxicity of fast food? *Gut*, 57(5), 568–570. https://doi.org/10.1136/gut.2007.143958
168. Mager, D. R., Mazurak, V., Rodriguez-Dimitrescu, C., Vine, D., Jetha, M., Ball, G. et al. (2012). A meal high in saturated fat evokes postprandial dyslipemia, hyperinsulinemia, and altered lipoprotein expression in obese children with and without nonalcoholic fatty liver disease. *JPEN Journal of Parenteral and Enteral Nutrition*, 37(4), 517–528. https://doi.org/10.1177/0148607112467820
169. Khatatbeh, M., Momani, W., Altaani, Z., Al Saad, R., Al Bourah, A. (2021). Fast food consumption, liver functions, and change in body weight among university students: A cross-sectional study. *International Journal of Preventive Medicine*, 12(1), Article 109. https://doi.org/10.4103/ijpvm.ijpvm_194_19
170. Takahashi, F., Hashimoto, Y., Kawano, R., Kaji, A., Sakai, R., Kawate, Y. et al. (2020). Eating fast is associated with non-alcoholic fatty liver disease in men but not in women with type 2 diabetes: A cross-sectional study. *Nutrients*, 12(8), Article 2174. https://doi.org/10.3390/nu12082174
171. Tamargo, J. A., Sherman, K. E., Campa, A., Martinez, S. S., Li, T., Hernandez, J. et al. (2021). Food insecurity is associated with magnetic resonance-determined nonalcoholic fatty liver and liver fibrosis in low-income, middle-aged adults with and without HIV. *The American Journal of Clinical Nutrition*, 113(3), 593–601. https://doi.org/10.1093/ajcn/nqaa362
172. Charlton, M., Krishnan, A., Viker, K., Sanderson, S., Cazanave, S., McConico, A. et al. (2011). Fast food diet mouse: Novel small animal model of NASH with ballooning, progressive fibrosis, and high physiological fidelity to the human condition. *American Journal of Physiology Gastrointestinal and Liver Physiology*, 301(5), G825–G834. https://doi.org/10.1152/ajpgi.00145.2011
173. Bayol, S. A., Simbi, B. H., Fowkes, R. C., Stickland, N. C. (2010). A maternal "junk food" diet in pregnancy and lactation promotes nonalcoholic fatty liver disease in rat offspring. *Endocrinology*, 151(4), 1451–1461. https://doi.org/10.1210/en.2009-1192
174. Kalafati, I.-P., Borsa, D., Dimitriou, M., Revenas, K., Kokkinos, A., Dedoussis, G. V. (2019). Dietary patterns and non-alcoholic fatty liver disease in a Greek case-control study. *Nutrition*, 61, 105–110. https://doi.org/10.1016/j.nut.2018.10.032
175. Uchiyama, M., Maruyama, C., Umezawa, A., Kameyama, N., Sato, A., Kamoshita, K. et al. (2022). A cross-sectional pilot study on food intake patterns identified from very short FFQ and metabolic factors including liver function in healthy Japanese adults. *Nutrients*, 14(12), Article 2442. https://doi.org/10.3390/nu1412244
176. Delzenne, N. M., Hernaux, N. A., Taper, H. S. (1997). A new model of acute liver steatosis induced in rats by fasting followed by refeeding a high carbohydrate-fat-free diet: biochemical and morphological analysis. *Journal of Hepatology*, 26(4), 880–885. https://doi.org/10.1016/s0168-8278(97)80256-5
177. Ouyang, X., Cirillo, P., Sautin, Y., McCall, S., Bruchette, J. L., Diehl, A. M. et al. (2008). Fructose consumption as a risk factor for non-alcoholic fatty liver disease. *Journal of Hepatology*, 48(6), 993–999. https://doi.org/10.1016/j.jhep.2008.02.011
178. Henney, A. E., Gillespie, C. S., Alam, U., Hydes, T. J., Cuthbertson, D. J. (2023). Ultra-processed food intake is associated with non-alcoholic fatty liver disease in adults: A systematic review and meta-analysis. *Nutrients*, 15(10), Article 2266. https://doi.org/10.3390/nu15102266
179. Bloomer, S. A., Moyer, E. D. (2021). Hepatic macrophage accumulation with aging: Cause for concern? *American Journal of Physiology — Gastrointestinal and Liver Physiology*, 320(4), G496–G505. https://doi.org/10.1152/ajpgi.00286.2020
180. Lozada-Delgado, J. G., Torres-Ramos, C. A., Ayala-Peña, S. (2020). Aging, oxidative stress, mitochondrial dysfunction, and the liver. Chapter in a book: Aging (Second Edition). Academic Press, 2020. https://doi.org/10.1016/b978-0-12-818698-5.00004-3
181. Li, Y., Adeniji, N. T., Fan, W., Kunimoto, K., Török, N. J. (2022). Non-alcoholic fatty liver disease and liver fibrosis during aging. *Aging and Disease*, 13(4), 1239–1251. https://doi.org/10.14336/ad.2022.0318
182. Mason, C. E., Sierra, M. A., Feng, H. J., Bailey, S. M. (2024). Telomeres and aging: On and off the planet! *Biogerontology*, 25(2), 313–327. https://doi.org/10.1007/s10522-024-10098-7
183. Radonjić, T., Dukić, M., Jovanović, I., Zdravković, M., Mandić, O., Popadić, V. et al. (2022). Aging of liver in its different diseases. *International Journal of Molecular Sciences*, 23(21), Article 13085. https://doi.org/10.3390/ijms232113085
184. Wan, Y., Li, X., Slevin, E., Harrison, K., Li, T., Zhang, Y. et al. (2021). Endothelial dysfunction in pathological processes of

chronic liver disease during aging. *The FASEB Journal*, 36(1), Article e22125. https://doi.org/10.1096/fj.202101426R
185. Maeso-Díaz, R., Gracia-Sancho, J. (2020). Aging and chronic liver disease. *Seminars in Liver Disease*, 40(04), 373–384. https://doi.org/10.1055/s-0040-1715446
186. Yang, S., Liu, C., Jiang, M., Liu, X., Geng, L., Zhang, Y. et al. (2023). A single-nucleus transcriptomic atlas of primate liver aging uncovers the pro-senescence role of SREBP2 in hepatocytes. *Protein and Cell*, 15(2), 98–120. https://doi.org/10.1093/procel/pwad039
187. Azman, K. F., Safdar, A., Zakaria, R. (2021). D-galactose-induced liver aging model: Its underlying mechanisms and potential therapeutic interventions. *Experimental Gerontology*, 150, Article 111372. https://doi.org/10.1016/j.exger.2021.111372

AUTHORS' INFORMATION:

Farhang H. Awlqadr, Assistant Lecturer, Food Science and Quality Control, Halabja Technical College, Sulaimani Polytechnic University, Sulaymaniyah, Iraq. Tel.: +964–770–142–09–62, E-mail: farhang.hamid.a@spu.edu.iq
ORCID: https://orcid.org/0000-0002-1737-5920

Ammar B. Altemimi, Associate Professor, Department of Food Science, College of Agriculture, University of Basrah. Basrah, 61004, Iraq. Tel.: +964–773–564–00–90, E-mail: ammar.ramddan@uobasrah.edu.iq
ORCID: https://orcid.org/0000-0001-7750-5988
* corresponding author

Syamand Ahmed Qadir, Lecturer, Medical Laboratory Techniques Department, Halabja Technical Institute, Research center, Sulaimani Polytechnic University, Sulaymaniyah, Iraq. Tel.: +964–773–017–52–51, E-mail: syamand.qadir@spu.edu.iq
ORCID: https://orcid.org/0000-0002-6498-2643

Zina T. Alkanan, Associate Professor, Department of Food Science, College of Agriculture, University of Basrah. Basrah, 61004, Iraq. Tel.: +964–783–327–55–53, E-mail: zina.alkanan@uobasrah.edu.iq
ORCID: https://orcid.org/0000-0002-0607-1358

Aryan Mahmood Faraj, Lecturer, Medical Laboratory Science Department/Halabja Technical College of Applied Sciences/ Sulaimani Polytechnic University Sulaymaniyah, Iraq. Tel.: +964–773–264–80–94, E-mail: aryan.faraj@spu.edu.iq
ORCID: https://orcid.org/0000-0002-7229-3717

Qausar Hamed ALKaisy, Associate Professor, Department of Dairy Science and Technology, College of Food Sciences, University of AL-Qasim Green, Al Qasim, Iraq. Tel.: +964–781–373–90–00, E-mail: qayssarhamad@fosci.uoqasim.edu.iq
ORCID: https://orcid.org/0000-0003-2039-6551

Tarek G. Abedelmaksoud, Associate Professor, Department of Food Science, Faculty of Agriculture, Cairo University 1 Gamaa Street, 12613, Giza, Egypt. Tel.: +2–0110–144–12–80, E-mail: tareekgamal_88@agr.cu.edu.eg
ORCID: https://orcid.org/0000-0002-7012-6667
* corresponding author

All authors bear responsibility for the work and presented data.

All authors made an equal contribution to the work.

The authors were equally involved in writing the manuscript and bear the equal responsibility for plagiarism.

The authors declare no conflict of interest.