



International Research Journal of Public Health (ISSN:2573-380X)



Obesity and stress rather than the thrifty gene, major risk factors for type 2 diabetes mellitus

Zelalem Tafese

Hawassa University

ABSTRACT

Introduction: Type 2 Diabetes Mellitus (T2DM) is becoming one of the main problems identified for public health importance by the World Health Organization (WHO) particularly in developing countries. The exact cause for T2DM is unknown for sure but multiple predisposing factors are suggested including overweight, obesity, and sedentary lifestyle and stress. **Objectives:** To systematically review the major risk factors for T2DM giving more emphasis in the developing world. **Methodology:** The search for studies was performed through Google scholar, Medline, Pubmed, Cochrane Library, and Web of Science databases. The keywords that were used to search in the database included "T2DM" AND "predisposing factors" AND "determinants OR risk factor", relevant articles were included for review. **Results:** Of the fifty-eight study results that met the inclusion criteria; the majority reported stressful life as the risk factor for type 2 diabetes mellitus. Other articles noted obesity, out of which some reported obesity linked with the 'thrifty gene' as a risk factor. Furthermore, the remaining reports suggested genetic traits, aging, lack of exercise, and insulin resistance, and infections all contribute to the risk of diabetes. **Conclusion:** Although not everyone with T2DM is overweight, the presence of obesity, lack of physical activity, and stressful life increase the chances of acquiring T2DM. But it is doubtful to accept the thrifty gene hypothesis to play a role in the higher proportion of diabetes. This review highlights the need for a further study focused on finding a new approach to manage and prevent diabetes is recommended.

Keywords: Determinants; Obesity; Prevention; Stress, Thrifty gene; Type 2 diabetes mellitus

*Correspondence to Author:

Zelalem Tafese

Hawassa University

How to cite this article:

Zelalem Tafese. Obesity and stress rather than the thrifty gene, major risk factors for type 2 diabetes mellitus. International Research Journal of Public Health, 2022; 6:64.



eSciPub LLC, Houston, TX USA.

Website: <https://escipub.com/>

Introduction

Globally an estimated 425million people are diabetic and, this figure is expected to grow to over 600 million by 2045 ^[1]. Diabetes mellitus is one of the main health challenges, with an estimated 95% of the global diabetic population having type 2 diabetes^[2]. Studies reported the burden of diabetes mellitus to increase year to year^[3,4] and noted the prevalence of diabetes for all age groups worldwide was estimated to be 2.8% in 2000 and 4.4% in 2030. The rise in the number of diabetes is expected to be high particularly among the urban population in developing countries ^[5].

The etiology of diabetes is mainly the interaction of environmental and genetic factors ^[6]. Previous studies noted although multiple risk factors are suggested for the occurrence of T2DM ^[7]most of these factors are mainly determined by the role genetic and metabolic factors including, ethnicity, family history of diabetes, and previous gestational diabetes, older age, overweight and obesity, unhealthy diet, physical inactivity excess alcoholic drink, and smoking to increase risk ^[8,9]. On the other hand, scholars propose the thrifty phenotype hypothesis pointing to the epidemiological associations between poor fetal and infant growth and the subsequent development of type 2 diabetes, which produces permanent changes in glucose-insulin metabolism ^[10]. They also suggested the association of exposure to famine resulting in low birth weight due to nutritional deprivation in utero resulted in the dysfunction of beta-cell expressed as T2DM when those with these genotypes were exposed to an environment with abundant food ^[11,12].

But the relative contribution of the genetic versus the environmental factors to cause T2DM remained controversial. With this regard, other scholars debated rather than the mere thrifty gene hypothesis, dietary quality, lack of physical exercise, and stressful life play a significant role in increasing the risk of T2DM ^[13,14]. Recent studies also indicated that there is the coexistence of nutritional deficiencies and over-

nutrition associated with physical inactivity, due to urbanization and industrialization ^[15]. Now a day's developing countries face a rapid change in the nutrition transition and increases in non-communicable diseases ^[16]. Mass media access and a change in technological advancement facilitate physical inactivity in turn this promotes obesity and the risk of chronic non-communicable diseases ^[17]. All these conditions increase the prevalence of diabetes in low and middle-income countries ^[18]. On the other hand, research evidence points out a key role of infectious agents as a possible predictor for diabetes mellitus^[1] Identifying the most important risk factors may be important to formulate strategies towards decreasing the prevalence of T2DM, a disease with no definitive cause ^[19]. Hence, this review article examines the modifiable risk factor for T2DM by reviewing high-quality works of literature/articles. The findings fill an evidence gap to inform policy-makers on the main factors associated with T2DM and to formulate a preventive strategy to halt the incidence.

METHODS

The published results from high-quality human observational and experimental studies which analyzed the factors associated with T2DM were all included in this literature-based analysis. An electronic search of Medline, Pub Med, Google Scholar, Medline, Cochrane Library, and Web of Science databases published up to the end of 2019 was conducted. The search was done in keywords: ["factors associated with diabetes" OR "diabetes mellitus" AND ["Impacts" OR "factors associated"] AND [Observational studies OR experimental studies]. A function extracting related articles as well as reference lists from research, reviews, and editorials was used during the search process. The full version of the English-language analyzed articles and abstracts of most found papers were available during the selection process. All pieces of literature, including observational studies, systematic review, and Meta-analysis, published in the English language, were included. There

were no limits on the place of the study, and gender. In the primary search, 285 records were found. After the studies/reviews which did not examine the major factors associated with T2DM, duplicated, and the majority of the outdated publications that were published before 2000 were excluded and 190 articles were selected. During the second selection, 58 articles were evaluated as potentially relevant considering factors associated with T2DM. The flow diagram of the article screening and selection process is shown in Fig. 1.

Result and discussion

Overall from the final articles include for the present review and analysis, fifteen of them noted stressful life as a cause of diabetes, twelve of them were related to the effect of diet and obesity and metabolic syndrome as a factor that increases the risk of T2DM. Likewise, eleven articles propose the thrifty phenotype hypothesis as a risk factor for T2DM of course some articles opposed this hypothesis, five articles suggested the combined effect of genetic traits, aging, lack

PRISMA 2009 flow diagram

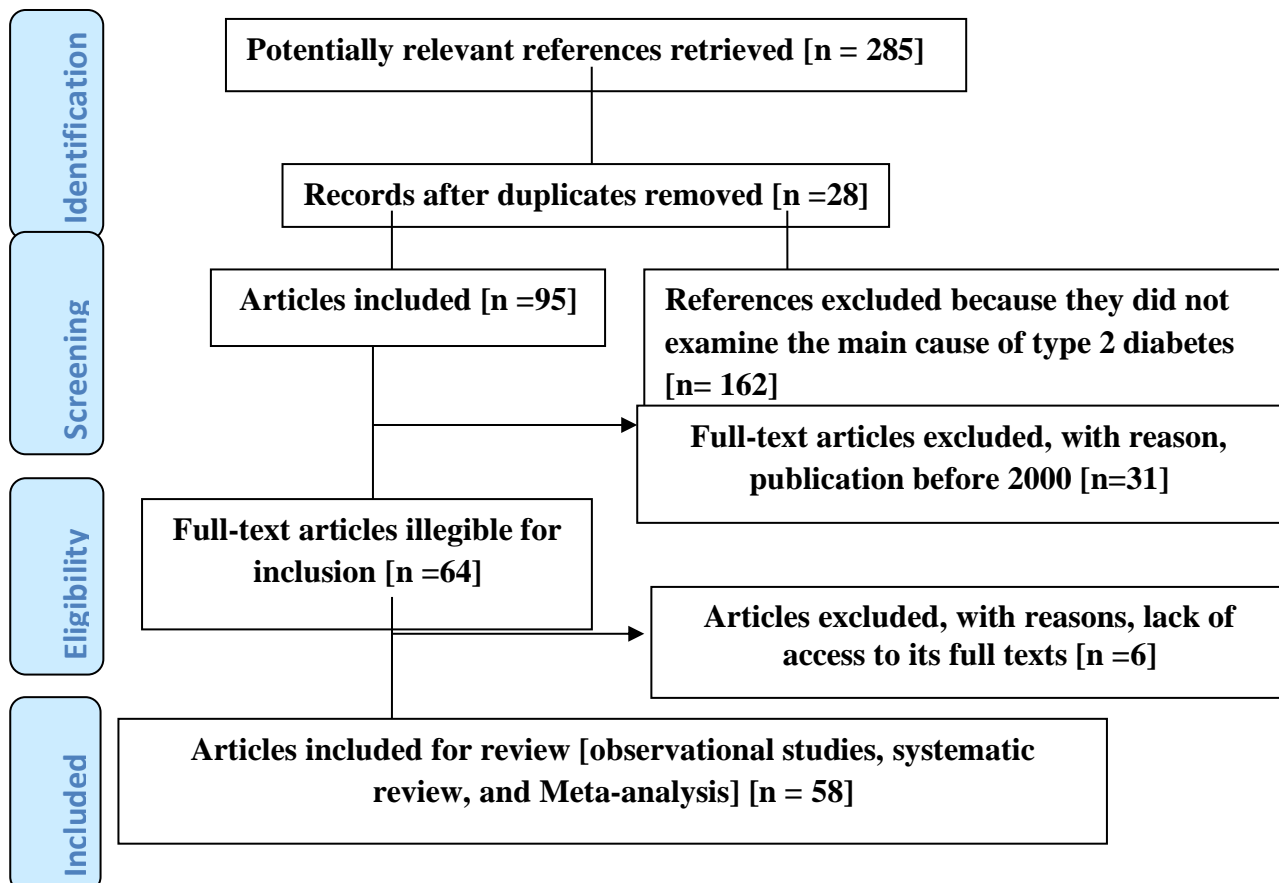


Figure.1 The PRISMA chart used to select the studies for the present review

of exercise, obesity, and insulin resistance all contribute to the risk of diabetes, three articles inadequate fruit in a diet as a predisposing factor for T2DM, five of their alcohol consumption and the remaining eight articles reviewed were about the effects of infections to increase the risk

Although multiple risk factors are suggested, none of these factors is the definitive cause for T2DM. But the results of the final analysis of most articles reviewed, emphasized the interaction between unhealthy diet, obesity, and stressful life as a risk factor for its occurrence. Epidemiological studies suggested that obesity

is among the major risk factor of diabetes and having normal body weight is crucial in the prevention of T2DM, regardless of genetic predisposition [20,21].

Previous review articles noted that genetic traits, obesity, and insulin resistance increase the risk of diabetes in the African American community [22]. On a similar venue, the parallel increment rates of obesity and higher prevalence of diabetes among the African American populations were also reported [23]. Few studies argued that although the role of genetics, aging, and ethnicity are reported in developing insulin sensitivity, the contribution of excess body weight, large waist circumference, lack of physical exercise, and smoking played a crucial role behind insulin resistance [24-27]. For this reason, maintaining optimal waist circumference and body weight should be emphasized as an important strategy to reduce insulin resistance and subsequent metabolic diseases [28].

Although a strong relationship of increase in the body mass index with diabetes and insulin resistance was reported; contradicting data was also reported by another study in which healthy metabolic profile and the absence of diabetes risk factors is not protective young adults from the incident of diabetes associated with overweight and obesity [29]. This indicates other hidden factors to be taken into consideration.

Although there have been several well-documented hypotheses for insulin resistance [30]; the suggested factors of insulin resistance among obese individuals include an increase in the amount of nonesterified fatty acids, glycerol, hormones, cytokines, pro-inflammatory markers, and other substances [31]. But the development of diabetes becomes more likely if the dysfunction β -cells of the islet of the pancreas are accompanied by insulin resistance. Based on the available evidence maintaining normal body weight, doing regular physical exercise, and preventing abdominal obesity are among important strategies to prevent T2DM [32].

Researchers are still studying many factors that could make diabetes more common among people of certain ethnicities [33]. Another study evidenced that the risk of T2DM among ethnic minority groups living in Europe compared to Europeans varies by geographical origin [34]. The same study emphasized the need for future research and policy initiatives on T2DM among ethnic minority taking ethnic differences into account.

The occurrence of metabolic syndrome is also one of the leading factors for the occurrence of T2DM [35]. Although the exact cause for the metabolic syndrome is not known for sure, a previous study reported it to have resulted from a complex interaction of genetic, metabolic, socio-economic, and environmental factors [36]. Similar results were also shown in previous studies that lower education level, past smoking, the absence of fruit/vegetables in the diet [37-39]. Additionally, carbonated soft drinks and the consumption of significant amounts of alcohol were risk factors for the occurrence of metabolic syndrome, and this may increase the risk of T2DM [40].

The interrelationship between alcoholism and diabetes is reported by a review article explaining the link between the effects of a disruption of glucose homeostasis and insulin resistance [41], which are affected by the altered appetite that regulates the peptides and neurotrophic factors [42]. On the other hand, a moderate amount of alcohol is reported to play a role in preventing the incidence of diabetes and lowering the mortality risk among diabetic individuals. But these benefits may be varied according to age, gender, body mass index, ethnics and type of alcohol drink [43]. Similarly, scholars evidenced that compared with beer or other distilled liquor, wine was reported to be associated with a significant decrease risk of T2DM [44]. These findings support the recommendation of the functional benefit of wine in the Mediterranean diet [45].

Previous studies noted that metabolic syndrome is an important risk factor for T2DM and it might

be useful as a practical tool to predict these major metabolic disorders. The risk of T2DM can be high among individuals with multiple cardiovascular disease risk factors [46-48]. On the other hand, it is reported that metabolic syndrome predicts diabetes independently of other factors^[49].

Whether it's related to work, relationships, or some other aspect of your life, stress is another suggested factor for T2DM. Many of the final articles reviewed; reported emotional stress to play a role in the etiology of T2DM ^[50-52]. Inline, a review article by Lloyd and colleagues ^[53] documented depression as a risk factor for the development of T2DM emphasizing stressful experiences might affect diabetes, in terms of both its onset and its complication. Furthermore, other reviewed articles suggested that not only depression but also general emotional stress, anxiety, sleeping problems, anger, and hostility have an association with an increased risk for the development of T2DM ^[50]. There are different causes for stress, among the various causes; immigration is one of the reported factors for stress in one's life [54]. Immigrants are expected to experience considerable stress, which may arise from a variety of sources including, ethnicity, gender and socioeconomic^[55], language difficulties^[56] separation from family and social networks^[57], and greater exposure to racism and discrimination not previously experienced in one's country of birth ^[58]. In due course, continued stress exposure was reported to play a vital role in increasing the risk of disease risk ^[59, 24]. A study among African children emphasized frequent exposure to stressful life events in childhood to increase the risk of diabetes in their adult life ^[60]. Inline, a population-based, study in Finland evidenced those individuals who are exposed to extremely stressful life events and high workload had an increased risk of having a metabolic syndrome and insulin resistance as well ^[52]. The same studies noted life events perceived as stressful, particularly those related to finance and work, maybe a signal for poor metabolic health. In

another review article, it is evidenced that stressful life events are related to poor metabolic control among diabetic older adolescents who are diabetic ^[61]. A recent longitudinal study using causal modeling documented that majority of the effect of the estimate of stress on diabetes risk is not mediated by the traditional risk factors of hypertension, physical activity, smoking, diet quality, and body mass index ^[62]. To date existing literature-based evidence showed although the impact of lifestyle changes including dietary modification on disease risk has been extensively studied, associations of psychosocial stress and disease risk do not get adequate attention ^[63,51]. This implicates the need for further trials on diabetic prevention and clinical care.

The other suggested factor to play a role in the incident of T2DM is the thrifty phenotype hypothesis which states early growth restriction as a result of adaptation to environmental deprivation ^[10, 11]. However, scholars claimed that the thrifty phenotype hypothesis fails to explain why environmentally induced changes are lost so early in development ^[15]. This is because the pre-conditions for the evolution of thrifty fetal programming are restricted if the correlation between in utero and lifetime conditions is poor. Such a correlation is not observed in natural courses of famine. Baig and colleagues ^[12] claimed that if there is fetal programming for thriftiness, it could have evolved in anticipation of social factors affecting nutrition that can result in a positive correlation.

On the other hand, the thrifty gene hypothesis is opposed by claiming this hypothesis is deeply flawed ^[64, 12]. Some of these scholars suggested alternatives, based on the central notion that genetic drift rather than positive selection was a dominant factor, may be called the 'drifty gene' hypothesis ^[11]. This alternative gives a possible explanation that the change in the frequency of an existing gene variant in a population is due to chance. In the context of the identified pieces of evidence mentioned above, it is doubtful to accept the thrifty gene hypothesis played a role

in the higher proportion of diabetes among black Americans^[65].

Similarly, scholars noted that may be individuals with a thrifty phenotype, having small bodies and specialized metabolisms adapted to cope with poor-quality diets, get into T2DM if instead, they find themselves growing up in a wealthy society to which they are poorly adapted. But this speculation is debated as it might be regarded as hateful ^[38]. It could be seen as encouraging the rich to look self-satisfied with their impoverished fellow human beings.

The findings of other reviewed articles suggested ethnicity as the other factor in the increased risk of T2DM ^[66, 67, 22, 33]. Inline, a cohort that compared black and white participants noted an increased risk of incident T2DM among black women and men. However, after adjusting for modifiable risk factors during young adulthood, no longer statistically significant ^[68]. On a similar venue, a review by Golden and colleagues documented that genetic susceptibility has not been uniform among the world's racial/ethnic groups^[69]. The same study provided the evidence in the USA; in which ethnicity is associated with many other risk factors for type 2 diabetes, including being obese, diet, and socioeconomic status, although studies in this area are inadequate.

Besides the role of the genetic factors in the genesis of diabetes, other external environmental factors are also known to be the cause. Few studies revealed the association of viral infection with diabetes ^[70, 71]. Though the exact mechanisms involved are not known for sure, it is clear that several viruses can directly affect β cells of the pancreas to decrease or inhibit insulin synthesis ^[72]. Additionally, it is emphasized the role of infections to contribute to insulin resistance should be considered as a risk factor for T2DM ^[73, 2]. These studies noted that among biological agents possibly linked to diabetes mellitus, the gut microbiome [a variety of microbes that are both helpful and potentially harmful], and the hepatitis C virus, are mainly discussed. Similarly, a shred of supporting

evidence reported by recent studies pointing out the bidirectional relationship between Covid-19 and diabetes, showing both to increase the risk of incidence of diabetes among COVID 19 survivors and increased risk of severe disease outcome from Covid-19 ^[74,75]. The pathophysiologies on the occurrence of diabetes on COVID survivors is due to the coronavirus entering islets of the pancreas using angiotensin-converting enzyme 2 [ACE2] as its receptor and damages islets causing acute diabetes^[76,77]. Based on this fact it is predictive that the toll of diabetes may be increased during post COVID period.

Therefore, it is clear to see that although the predictor for T2DM is not conclusive, any or all of the suggested risk factors are believed to increase its incidence. This emphasizes the need for further trials to fill the literature gap and improve the prevention and clinical practice.

The major strength of this review was the gathering of the latest research on the major predictors of T2DM. Included studies provided existing literature evidence on the suggested causes and hypotheses for T2DM. Moreover, it provides useful information on the literature gap needing further study and evidence for prevention approaches for T2DM. However, this systematic review still had limitations to be acknowledged, it failed to run a meta-analysis due to the study quality and high heterogeneity of included studies. High heterogeneity hindered this review to provide more information for further analysis.

Conclusion

From the present review, it was possible to learn that almost all available studies conducted in various parts of the world revealed no definitive cause for T2DM reported, and multiple risk factors are suggested for the occurrence of T2DM. It can be concluded that the proposed thrifty phenotype hypothesis lacks soundness to show association with T2DM. This paper identified several risk factors for T2DM out of these having normal body weight, and a healthy lifestyle pattern including decreased stress

levels could lead to decreased risk for T2DM. Our findings indicated the need for specific strategies for public health interventions to reduce the future incidence of T2DM. Interventions for the promotion of physical activity and a healthy lifestyle and dietary pattern combined with interventions against the increased incidence of obesity could halt the increase in the toll of T2DM incidence in near future. Future implementation research should focus on identifying efficient strategies to modify lifestyles and predisposing factors including dietary patterns that lead to overweight, obesity, and stress alleviation approaches are recommended. Further studies to identify a better approach to managing and preventing diabetes are recommended.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

ZT developed the review parameters and secured support. He also undertook partial literature search, data extraction, and analysis. SA made a detailed and extensive literature search and involved a synthesis of the findings. Both authors read and approved the final manuscript.

References

- [1]. Toniolo A, Cassani G, Puggioni A, Rossi A, Clombo A, Onodera T, et al. The diabetes pandemic and associated infections: Suggestions for clinical microbiology. *Rev Med Microbiol.* 2019;30[1]:1–17.
- [2]. Chakraborty S, Bhattacharyya R, Banerjee D. Infections: A Possible Risk Factor for Type 2 Diabetes. In: *Advances in Clinical Chemistry.* Academic Press Inc.; 2017. p. 227–51.
- [3]. Animaw W, Seyoum Y. Increasing prevalence of diabetes mellitus in a developing country and its related factors. Schooling CM, editor. *PLoS One* [Internet]. 2017 Nov 7;12[11]:e0187670. Available from: <https://doi.org/10.1371/journal.pone.0187670>
- [4]. Saeedi P, Petersohn I, Salpea P, Malanda B, Karuranga S, Unwin N, et al. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: Results from the International Diabetes Federation Diabetes Atlas, 9th edition. *Diabetes Res Clin Pract.* 2019 Nov;157:107843.
- [5]. Wild, Roglic, Green, Sicree & K. Estimates for the year 2000 and projections for 2030. *World Health.* 2004;27[5].
- [6]. Ershow AG. Environmental Influences on Development of Type 2 Diabetes and Obesity: Challenges in Personalizing Prevention and Management Introduction: Environmental Influences on Diabetes and Obesity. Vol. 3, *J Diabetes Sci Technol.* 2009. Available from: www.journalofdst.org
- [7]. Wu Y, Ding Y, Tanaka Y, Zhang W. Risk Factors Contributing to Type 2 Diabetes and Recent Advances in the Treatment and Prevention. *Int J Med Sci.* 2014 [cited 2021 Aug 25];11[11]:1185–200. Available from: <http://www.medsci.org1185>
- [8]. Schulze MB, Hu FB. Primary prevention of diabetes: What Can Be Done and How Much Can Be Prevented? *Annu Rev Public Health.* 2005 Apr 21;26[1]:445–67.
- [9]. WHO Global Report on Diabetes. *Global Report on Diabetes.* Vol. 978, Isbn. 2016.
- [10]. Hales N, Barker DJP. The thrifty phenotype hypothesis. Vol. 60, *British Medical Bulletin.* 2001. Available from: <https://academic.oup.com/bmb/article/60/1/5/322752>
- [11]. Speakman JR. Thrifty genes for obesity, an attractive but flawed idea, and an alternative perspective: the 'drifty gene' hypothesis. *Int J Obes.* 2008 Nov 14;32[11]:1611–7.
- [12]. Baig U, Belsare P, Watve M, Jog M. Can Thrifty Gene[s] or Predictive Fetal Programming for Thriftiness Lead to Obesity? *J Obes.* 2011;2011:1–11.
- [13]. de Koning L, Chiuve SE, Fung TT, Willett WC, Rimm EB, Hu FB. Diet-Quality Scores and the Risk of Type 2 Diabetes in Men. *Diabetes Care.* 2011 May 1;34[5]:1150–6.
- [14]. Harris ML, Oldmeadow C, Hure A, Luu J, Loxton D, Attia J. Stress increases the risk of type 2 diabetes onset in women: A 12-year longitudinal study using causal modelling. Samocha-Bonet D, editor. *PLoS One* . 2017 Feb 21;12[2]:e0172126.
- [15]. Wells JCK. The thrifty phenotype hypothesis: Thrifty offspring or thrifty mother? *J Theor Biol.* 2003 Mar 7;221[1]:143–61.
- [16]. Amuna P, Zotor FB. Epidemiological and nutrition transition in developing countries: impact on human health and development. *Proc Nutr Soc.* 2008 Feb;67[1]:82–90.

- [17]. Kljakovic M. A comparison of the respiratory care given to asthmatic and nonasthmatic children in a general practice. *N Z Med J* . 1994 Jun 22;107[980]:240–2.
- [18]. Popkin BM. Nutrition Transition and the Global Diabetes Epidemic. Vol. 15, *Current Diabetes Reports*. 2015.
- [19]. Beaglehole R, Bonita R, Horton R, Adams C, Alleyne G, Asaria P, et al. Priority actions for the non-communicable disease crisis. *Lancet* . 2011 Apr 23 [cited 2021 Sep 22];377[9775]:1438–47.
- [20]. Schnurr TM, Jakupović H, Carrasquilla GD, Ångquist L, Grarup N, Sørensen TIA, et al. Obesity, unfavourable lifestyle and genetic risk of type 2 diabetes: a case-cohort study. *Diabetologia*. 2020 Jul 15;63[7]:1324–32.
- [21]. Joanne Z. Rogers, D. C. Obesity and Type 2 Diabetes. In: Nyamdorj R, editor. *Epidemiology of Type 2 Diabetes*. BENTHAM SCIENCE PUBLISHERS; 2012. p. 39–64.
- [22]. Marshall MC. Diabetes in African Americans. *Postgrad Med J*. 2005;81[962]:734–40.
- [23]. Barnes AS. The epidemic of obesity and diabetes: Trends and treatments. *Texas Hear Inst J* . 2011;38[2]:142–4.
- [24]. Kacker S, Saboo N, Jitender S, Head. Prediabetes: Pathogenesis and Adverse Outcomes Prediabetes: Pathogenesis and Adverse Outcomes. *Int J Med Res Prof*. 2018;[November]:2–8.
- [25]. Kumar Khemka V, Banerjee A. Metabolic risk factors in obesity and diabetes mellitus: implications in the pathogenesis and therapy. *Integr Obes Diabetes*. 2017;3[3]:1–4. Available from: <http://www.oatext.com/metabolic-risk-factors-in-obesity-and-diabetes-mellitus-implications-in-the-pathogenesis-and-therapy.php>
- [26]. Gupta S, Bansal S. Does a rise in BMI cause an increased risk of diabetes?: Evidence from India. Böckerman P, editor. *PLoS One*. 2020 Apr 1;15[4]:e0229716. Available from: <https://doi.org/10.1371/journal.pone.0229716>
- [27]. Chung JO, Cho DH, Chung DJ, Chung MY. Associations among Body Mass Index, Insulin Resistance, and Pancreatic β -Cell Function in Korean Patients with New-Onset Type 2 Diabetes. *Korean J Intern Med*. 2012;27[1]:66.
- [28]. Jiang J, Cai X, Pan Y, Du X, Zhu H, Yang X, et al. Relationship of obesity to adipose tissue insulin resistance. *BMJ Open Diabetes Res Care*. 2020 Apr 2;8[1]:e000741.
- [29]. Twig G, Afek A, Derazne E, Tzur D, Cukierman-Yaffe T, Gerstein HC, et al. Diabetes risk among overweight and obese metabolically healthy young adults. *Diabetes Care*. 2014 Nov;37[11]:2989–95.
- [30]. Ye J. Mechanisms of insulin resistance in obesity. *Front Med [Internet]*. 2013 Mar 9;7[1]:14–24. Available from: <http://link.springer.com/10.1007/s11684-013-0262-6>
- [31]. Algoblan A, Alalfi M, Khan M. Mechanism linking diabetes mellitus and obesity. *Diabetes, Metab Syndr Obes Targets Ther*. 2014 Dec;587.
- [32]. Steyn N, Mann J, Bennett P, Temple N, Zimmet P, Tuomilehto J, et al. Diet, nutrition and the prevention of type 2 diabetes. *Public Health Nutr*. 2004;7[1a]:147–65.
- [33]. Oldroyd J, Banerjee M, Heald A, Cruickshank K. Diabetes and ethnic minorities. *Postgrad Med J*. 2005;81[958]:486–90.
- [34]. Meeks K, Freitas-Da-Silva D, Adeyemo A, Beune E, Modesti P, Stronks K, et al. Disparities in type 2 diabetes among ethnic minority groups resident in Europe - a meta-analysis. *Eur J Public Health*. 2015 Oct;25[suppl_3]. Available from: https://academic.oup.com/eurpub/article/25/suppl_3/ckv174.014/2484261
- [35]. Moreira GC, Cipullo JP, Ciorlia LAS, Cesarino CB, Vilela-Martin JF. Prevalence of metabolic syndrome: Association with risk factors and cardiovascular complications in an urban population. *PLoS One*. 2014;9[9].
- [36]. Henry GN, Paul MR. The Obesity Metabolic Syndrome and Type 2 Diabetes Mellitus Pandemic. 2010;4[2]:113–9.
- [37]. Ayana DA, Dessie Y, Teji K, Ayele D. Type 2 diabetes mellitus among government employees in Harar, Eastern Ethiopia: a cross-sectional study. *Res Reports Endocr Disord [Internet]*. 2015 Jul;5:71–5.
- [38]. Bateson P. Fetal experience and good adult designa. *Int J Epidemiol [Internet]*. 2001 Oct;30[5]:928–34.
- [39]. Du H, Li L, Bennett D, Guo Y, Turnbull I, Yang L, et al. Fresh fruit consumption in relation to incident diabetes and diabetic vascular complications: findings from the China Kadoorie Biobank Study. *Lancet Diabetes Endocrinol [Internet]*. 2016 Nov;4:S12.
- [40]. Cozma A, Sitar-Taut A, Urian L, Fodor A, Suharoschi R, Muresan C, et al. Unhealthy lifestyle and the risk of metabolic syndrome - the Romanian experience. *J Mind Med Sci*. 2018;5[2]:218–29.
- [41]. Wannamethee SG, Shaper AG, Perry IJ, Alberti KGMM. Alcohol consumption and the incidence of type II diabetes. In: *Journal of*

- Epidemiology and Community Health. 2002. p. 542–8.
- [42]. Kim S-J, Kim D-J. Alcoholism and Diabetes Mellitus. *Diabetes Metab J*. 2012;36[2]:108.
- [43]. Volaco A. Alcohol Consumption and its Relationship to Diabetes Mellitus: Friend or Foe? *Endocrinol Int J* . 2018 Jan 24;6[1].
- [44]. Huang J, Wang X, Zhang Y. Specific types of alcoholic beverage consumption and risk of type 2 diabetes: A systematic review and meta-analysis. *J Diabetes Investig [Internet]*. 2017 Jan;8[1]:56–68.
- [45]. Ortega RM. Importance of functional foods in the Mediterranean diet. *Public Health Nutr*. 2006;9[8A]:1136–40.
- [46]. Song SH, Hardisty CA. Diagnosing metabolic syndrome in type 2 diabetes: does it matter? *QJM* . 2008 Jan 21;101[6]:487–91.
- [47]. Zerga AA, Bezabih AM. Metabolic syndrome and lifestyle factors among type 2 diabetes mellitus patients in Dessie Referral Hospital, Amhara region, Ethiopia. *PLoS One*. 2020;15[11 November]:50–9.
- [48]. Agyemang-Yeboah F, Eghan BAJ, Annani-Akollor ME, Togbe E, Donkor S, Oppong Afranie B. Evaluation of Metabolic Syndrome and Its Associated Risk Factors in Type 2 Diabetes: A Descriptive Cross-Sectional Study at the Komfo Anokye Teaching Hospital, Kumasi, Ghana. *Biomed Res Int*. 2019 May 2;2019:1–8.
- [49]. Lorenzo C, Okoloise M, Williams K, Stern MP, Haffner SM. The Metabolic Syndrome as Predictor of Type 2 Diabetes: The San Antonio Heart Study. *Diabetes Care*. 2003;26[11]:3153–9.
- [50]. Kelly SJ, Ismail M. Stress and Type 2 Diabetes: A Review of How Stress Contributes to the Development of Type 2 Diabetes. *Annu Rev Public Health* . 2015 Mar 18;36[1]:441–62.
- [51]. Fang CY, Boden G, Siu PT, Tseng M. Stressful life events are associated with insulin resistance among Chinese immigrant women in the United States. *Prev Med Reports [Internet]*. 2015;2:563–7.
- [52]. Pyykkönen AJ, Räikkönen K, Tuomi T, Eriksson JG, Groop L, Isomaa B. Stressful life events and the metabolic syndrome: The prevalence, prediction and prevention of diabetes [PPP]-botnia study. *Diabetes Care [Internet]*. 2010;33[2]:378–84.
- [53]. Diabetes: A Review of the Links. *Diabetes Spectr* . 2005 Apr 1;18[2]:121–7.
- [54]. Urzúa A, Leiva-Gutiérrez J, Caqueo-Úrizar A, Vera-Villarreal P. Rooting mediates the effect of stress by acculturation on the psychological well-being of immigrants living in Chile. Capraro V, editor. *PLoS One*. 2019 Aug 13;14[8]:e0219485.
- [55]. Straiton ML, Ledesma HML, Donnelly TT. A qualitative study of Filipina immigrants' stress, distress and coping: the impact of their multiple, transnational roles as women. *BMC Womens Health [Internet]*. 2017 Dec 5;17[1]:72.
- [56]. Sangalang CC, Becerra D, Mitchell FM, Lechuga-Peña S, Lopez K, Kim I. Trauma, Post-Migration Stress, and Mental Health: A Comparative Analysis of Refugees and Immigrants in the United States. *J Immigr Minor Heal [Internet]*. 2019 Oct 22;21[5]:909–19.
- [57]. Pickett KE, Wilkinson RG. People like us: Ethnic group density effects on health. Vol. 13, *Ethnicity and Health*. 2008. p. 321–34.
- [58]. Gee GC, Ro A, Shariff-Marco S, Chae D. Racial Discrimination and Health Among Asian Americans: Evidence, Assessment, and Directions for Future Research. *Epidemiol Rev [Internet]*. 2009 Nov 1;31[1]:130–51.
- [59]. Acevedo-Garcia D, Sanchez-Vaznaugh E V., Viruell-Fuentes EA, Almeida J. Integrating social epidemiology into immigrant health research: A cross-national framework. *Soc Sci Med*. 2012 Dec 1;75[12]:2060–8.
- [60]. Djarova T, Dube S, Tivchev G, Chivengo A. Frequency of stressful life events as risk indicating factors for the onset of type 1 diabetes in African children. *S Afr J Sci*. 2007;103[7–8]:286–8.
- [61]. Helgeson VS, Escobar O, Siminerio L, Becker D. Relation of Stressful Life Events to Metabolic Control Among Adolescents With Diabetes: 5-Year Longitudinal Study. *Heal Psychol*. 2010;29[2]:153–9.
- [62]. Harris ML, Oldmeadow C, Hure A, Luu J, Loxton D, Attia J. Stress increases the risk of type 2 diabetes onset in women: A 12-year longitudinal study using causal modelling. *PLoS One*. 2017;12[2]:1–13.
- [63]. Macleod J, Davey Smith G. Psychosocial factors and public health: A suitable case for treatment? . Vol. 57, *Journal of Epidemiology and Community Health*. 2003. p. 565–70.
- [64]. Baig U, Belsare P, Watve M, Jog M. Can Thrifty Gene[s] or Predictive Fetal Programming for Thriftiness Lead to Obesity? *J Obes*. 2011;2011:1–11.
- [65]. Marshall MC. Diabetes in African Americans. *Postgrad Med J* . 2005 Dec 1;81[962]:734–40.
- [66]. Zhu Y, Sidell MA, Arterburn D, Daley MF, Desai J, Fitzpatrick SL, et al. Racial/ethnic

disparities in the prevalence of diabetes and prediabetes by BMI: Patient Outcomes Research to Advance learning [Portal] multisite cohort of adults in the U.S. Diabetes Care. 2019;42[12]:2211–9.

- [67]. Banerjee M, Vyas A, Cruickshank JK. Ethnicity and its implications in diabetes management. *Pract Diabetes Int.* 2004;21[4]:135–7.
- [68]. Bancks MP, Kershaw K, Carson AP, Gordon-Larsen P, Schreiner PJ, Carnethon MR. Association of modifiable risk factors in young adulthood with racial disparity in incident type 2 diabetes during middle adulthood. *JAMA - J Am Med Assoc.* 2017;318[24]:2457–65.
- [69]. Golden SH, Yajnik C, Phatak S, Hanson RL, Knowler WC. Racial/ethnic differences in the burden of type 2 diabetes over the life course: a focus on the USA and India. *Diabetologia* [Internet]. 2019 Oct 27;62[10]:1751–60.
- [70]. Manderwad GP. Role of Coxsackie virus B in Type 1 Diabetes- Brief Review Role of Enterovirus in Diabetes. *iMedPub Journals.* 2017;1[1–3]:1–2.
- [71]. Turk Wensveen T, Gašparini D, Rahelić D, Wensveen FM. Type 2 diabetes and viral infection; cause and effect of disease. *Diabetes Res Clin Pract.* 2021 Feb 1 [cited 2021 Jul 31];172:108637.
- [72]. Petzold A, Solimena M, Knoch KP. Mechanisms of Beta Cell Dysfunction Associated With Viral Infection. *Curr Diab Rep.* 2015;15[10].
- [73]. Turk Wensveen T, Gašparini D, Rahelić D, Wensveen FM. Type 2 diabetes and viral infection; cause and effect of disease. *Diabetes Res Clin Pract.* 2021 Feb;172:108637.
- [74]. Singh AK, Gupta R, Ghosh A, Misra A. Diabetes in COVID-19: Prevalence, pathophysiology, prognosis and practical considerations. *Diabetes Metab Syndr Clin Res Rev.* 2020 Jul;14[4]:303–10.
- [75]. Rubino F, Amiel SA, Zimmet P, Alberti G, Bornstein S, Eckel RH, et al. New-Onset Diabetes in Covid-19. *N Engl J Med* [Internet]. 2020 Aug 20;383[8]:789–90.
- [76]. Yang J-K, Lin S-S, Ji X-J, Guo L-M. Binding of SARS coronavirus to its receptor damages islets and causes acute diabetes. *Acta Diabetol.* 2010 Sep;47[3]:193–9.
- [77]. Lim S, Bae JH, Kwon H-S, Nauck MA. COVID-19 and diabetes mellitus: from pathophysiology to clinical management. *Nat Rev Endocrinol.* 2021 Jan 13;17[1]:11–30.



-
- Title: International Research Journal of Public Health
 - ISSN: 2573-380X
 - DOI: 10.28933/IRJPH
 - IF: 1.36 (citefactor)
 - Email: IRJPH@escipub.com
 - TEL: +1-281-656-1158
-



About the journal

The journal is hosted by eSciPub LLC. Our aim is to provide a platform that encourages publication of the most recent research and reviews for authors from all countries.

About the publisher

eSciPub LLC is a publisher to support Open Access initiative located in Houston, Texas, USA. It is a member of the largest community of professional publishers in the United States: the Independent Book Publishers Association. It hosts more than 100 Open Access journals in Medicine, Business & Economics, Agriculture, Biological Sciences, Chemistry, Education, Physical Sciences, Sociology, and Engineering and Technology.

Rapid Response Team

Please feel free to contact our rapid response team if you have any questions. Our customer representative will answer your questions shortly.

CC BY 4.0

This work and its PDF file(s) are licensed under under a Creative Commons Attribution 4.0 International License.

Terms of Use/Privacy Policy/ Disclaimer/ Other Policies:

You agree that by using our site, you have read, understood, and agreed to be bound by all of our terms of use/privacy policy/ disclaimer/ other policies (click here for details). This site cannot and does not contain professional advice. The information on this site is provided for general informational and educational purposes only and is not a substitute for professional advice. Accordingly, before taking any actions based upon such information, we encourage you to consult with the appropriate professionals. We do not provide any kind of professional advice. The use or reliance of any information contained on this site or our mobile application is solely at your own risk. Under no circumstance shall we have any liability to you for any loss or damage of any kind incurred as a result of the use of the site or our mobile application or reliance on any information provided on the site and our mobile application. We may publish articles without peer-review. Published articles of authors are open access. Authors hold the copyright and retain publishing rights without restrictions. Authors are solely responsible for their articles published in our journals. Publication of any information in authors' articles does not constitute an endorsement by us. We make no representation or warranty of any kind, express or implied, regarding the accuracy, adequacy, validity, reliability, availability or completeness of any information that authors provided. more.....