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Review Article

Lifestyle Genetics-Based Reports in the Treatment of Obesity

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Abstract

Obesity becomes a chronic disease due to the increasing number of mortality and morbidity cases around the world. In most regions, chronic illnesses, such as obesity, are important sources of morbidity and mortality. Due to a lack of effective strategies for prevention and management, the adverse effects of obesity and related diseases on health continue to be a serious problem. Relevant information was searched from Google Scholar, Scopus, and PubMed using such different terms as “Obesity”, “Obesity Management”, “Obesity AND Physical activity”, “Obesity AND Genetics”, “Obesity AND Diet”, and “Obesity AND Nutrigenomics”. Obesity is characterized by a complex interaction of hereditary and lifestyle factors, which includes food. Diet is an environmental element that plays an important and considerable role in the management of health and reduces the risk of obesity and its comorbidities. Changes in lifestyle patterns not only help burn extra calories but also prevent the development of obesity via its modulating effect on genetic factors. Different people respond differently to an obesogenic environment. The notion of nutrigenetics emerged as a result of various genetic variations that may explain this heterogeneity. Nutritional genomics, also known as nutrigenetics, is the study that investigates and analyses gene variations linked to varied responses to certain foods; moreover, it links this variation to diseases, such as obesity. As a result, tailored nutrition advice based on a person's genetic profile may improve the outcomes of a specific dietary strategy and offer a novel dietary strategy to improve life quality and preventing obesity. This study concluded that physical activity and dietary interventions play an effective role in the management of obesity. Moreover, understanding of the function of the most prominent obesity-related genes, as well as the interaction between nutrition and gene expression, will help researchers design personalized treatment strategies for humans.

Keywords: Diet, Obesity, Physical activity, Nutrigenetics, Single Nucleotide Polymorphisms

1. Context

Previously, obesity was considered a disease only present in developed countries; however, from the start of the 21st century, it has been observed that the number of obese persons is also increasing dramatically day by day in low- and middle-income countries (1, 2). According to an estimate by the World Health Organization, approximately, 13% of the world population had been categorized as obese in 2016 (3). A person with a body mass index (BMI) of ≥30 kg/m^2 is considered obese. Obesity is also a major risk factor for several lifestyle-associated metabolic disorders, such as hypertension, cardiovascular diseases, type 2 diabetes mellitus, and cancer. Several lifestyle and environmental factors have been associated with the onset and subsequent progression of this global health epidemic. Epidemiological studies

have demonstrated that due to profound changes in society, there has been a substantial decrease in human physical activities and a higher intake of energy-dense foods along with reduced consumption of fiber-rich foods; accordingly, cases of obesity are increasing dramatically all over the world. A healthy lifestyle with a balanced diet and appropriate physical activities seems to be a possible remedy for obesity; however, the implementation of this has been proved to be quite difficult for the general population. Since every person has a different metabolism and genetic predisposition, s/he responds differently to an obesogenic environment. Therefore, even in the presence of strong obesogenic conditions, the etiology of the disease is controlled by genetic factors (3). Various investigations have been carried out within families, twins, and foster children during the last 20 years, and with the advancement of molecular techniques, various obesity-linked genes have been identified (4). Montague, Farooqi (5) discovered the leptin gene as the first gene linked to a non-syndromic Mendelian type of obesity in 1997. Since 2007, genome-wide studies have identified over 100 variants linked to the most prevalent (polygenic) type of obesity (6). Several previous studies have extensively described the association between lifestyle and hereditary factors for the onset and etiology of obesity (2, 7). These studies could help researchers understand how new and effective strategies for the control and management of obesity could be developed by changing the sedentary lifestyle to an active lifestyle depending on the genetic makeup of every obese person.

This review summarizes the latest studies relating to obesity management programs using dietary interventions and physical activity. Some important genetic variants of obesity have also been discussed in this study which could help researchers establish personalized treatment approaches for obesity management.

2. Evidence Acquisition

Obesity is a serious public health problem associated with many other metabolic disorders. Relevant information was searched from Google Scholar, Scopus, and PubMed using such different terms as "Obesity", "Obesity Management", "Obesity AND Physical activity", "Obesity AND Genetics", "Obesity AND Diet", "Obesity AND Nutrigenomics", and "Obesity AND Gene variant/SNPs". Only those articles were included and discussed in this study that described the management of obesity via lifestyle changes, as well as those studies representing how the modification of gene variants involved in obesity progression could help control obesity.

3. Results

3.1. Physical Activity and Obesity Management

In this era of modernization, a sedentary lifestyle has significantly reduced the physical activity of individuals living in urban areas. The onset of obesity depends on several factors, such as dietary, environmental, and genetic factors. Physical activity has been considered one of the most important environmental factors that can contribute to controlling the obesity epidemic. It not only helps burn extra calories but also prevents the development of obesity via its modulating effect on genetic factors. In an EpiDREAM cohort study comprising of 17,423 participants from six ethnic groups, Reddon, Gerstein (3) has observed that increased physical activity significantly reduced the BMI of the participants. Furthermore, it also reduced the influence of fat mass and obesity (FTO) gene variation on BMI. The results of this study showed that physical activity reduced the effect of FTO rs1421085 single nucleotide polymorphism (SNP) on adiposity by 36%-75% in the EpiDREAM cohort (6). In another study by Xiang, Wu (8), it has been found that individuals carrying homozygous FTO alleles showed more weight loss when putting on a dietary or lifestyle intervention program, compared to the control group. Scientists have identified several genetic loci that play a crucial role in the regulation of energy balance in the body that affect adiposity (or weight change) over time. However, our understanding of these loci and their influence on BMI or body weight is limited. A precise understanding of these loci will certainly play an

important role in offering personalized or precision medicines for individuals. This understanding will also pave the way for personalized nutrition. The approach of personalized nutrition will consider the genetic makeup of an individual before offering dietary recommendations. However, the available data on gene-based dietary recommendations is not sufficient, and further research is required to ascertain the effectiveness of this approach (9). Nevertheless, the idea of A personalized gene-based nutrition approach offers an innovative approach to solve this rising health issue.

3.2. Obesity and Genetic Association

Recent Genome-Wide Association Studies have demonstrated a close interplay between genes and higher susceptibility to developing obesity. Some of the genes which have been linked with obesity are *FTO*, Melanocortin-4 receptor (*MC4R*), leptin, and the genes associated with carbohydrate and fatty-acid metabolisms (10). Some studies have linked epigenetic changes to higher susceptibility to developing obesity. Epigenetic changes affect the functions of genes without causing any change in their DNA sequences. A family history of obesity and type 2 diabetes makes a person highly susceptible to the development of these disorders, and understanding genetic factors will certainly help determine the best approach for a successful intervention, including dietary or lifestyle (11).

In a study, Valsesia et al. (12) observed a regulatory role of the *HGTX* (NKX6.3 orthologue) gene in lipid metabolism. This gene has been linked with weight loss through its regulatory effects on lipid metabolism. One of the major body lipids is a triglyceride, and a higher level of triglycerides has been linked with increased adiposity. The study reported that knock-down of *NKX6.3/HGTX* in *Drosophila* led to a significant reduction in systemic triglyceride accumulation. A recent study observed in cohorts of Diabetes Prevention Program and Action for Health in Diabetes that a specific copy of the minor G allele of *MTIF3* was responsible for greater weight loss after an intervention program over four years in comparison with the control arm (13). The *MTIF3* gene is responsible for the formation of the initiation complex on the mitochondrial 55S ribosome. This complex plays a critical role in several essential biochemical processes, such as energy balance and ATP synthesis (13). These studies have tremendously helped identify the role of genetics in the onset of obesity and showed a clear interaction between the dietary factors and genes. These studies also explain why the weight-loss programs do not provide similar results in all individuals when they undergo an intervention. The data is promising and has found potential implications in designing future personalized diet and weight loss programs (14).

3.3. Diet and Obesity

Dietary factors are important in determining the chances of developing obesity. Therefore, controlling dietary patterns could be a suitable option to treat obesity. Weight loss could be achieved by a variety of dietary practices, including macronutrient and food-based. Reduced energy density is a major weight-loss approach that can be used with all dietary patterns. Clinical research has shown that decreasing energy density helps people lose weight and maintain weight. A range of effective methodologies and approaches can assist promote effective weight loss and maintenance by minimizing energy density, offering portion control, and increasing the quality of diet. Individuals can modify and personalize their diet plan to minimize energy intake for long-term weight loss because of the flexibility of energy density. A new area of research (nutrigenetics) has gained attention in the recent past. This approach involves selecting a dietary program based on the genetic profile of an individual. A study conducted by Arkadianos et al. (15) has shown that individuals on a nutrigenetic diet were more likely to lose weight, compared to the control group (73% vs. 32%, respectively). Moreover, a higher number of individuals on a nutrigenetic diet showed a reduction in

fasting plasma glucose levels than the control group (57% vs. 25%, respectively). The outcomes of the Food4Me randomized controlled study described that how the APO E genotype affected responsiveness to customized dietary patterns. The results of this trial demonstrated that a diet based on genotypic information resulted in a significantly greater reduction of saturated fatty acid consumption for gene-based personalized nutrition targeted to APO E in comparison to conventional diet recommendations (16) highlighting that this strategy could improve dietary behavior with prolonged health benefits (16). These studies bolster our opinion that dietary interventions are highly effective when the genetic makeup of an individual is also considered in deciding the intervention.

3.4. Obesity-Related SNPs

The metabolism and energy utilization in the body depends on several vital pathways that require specific signaling by orexigenic and anorexigenic hormones and their receptors. The definitive signaling helps in metabolism and energy generation in the body. Glucose and lipid metabolisms are highly dependent on several hormones, such as insulin, leptin, adiponectin, and ghrelin. Several SNPs have been reported that make a person either more prone to develop obesity/metabolic syndrome or prevent the onset of these disorders.

3.4.1. Leptin

Leptin, a product of the obesity gene, plays a central role in maintaining energy homeostasis in the body. It is predominantly synthesized and secreted by the adipocytes, and its concentration is generally proportional to the adipocytes' mass (17). It has been observed that leptin levels decrease during the fasting period, and its absence is linked with hyperphagia (17). Studies have shown that leptin administration to deficient humans or mice successfully reduced obesity and hyperphagia. However, obesity in most human subjects has been characterized by increased circulatory levels of leptin indicating a leptin-resistant state. This action of leptin was mediated through the anorexigenic pro-opiomelanocortin neurons (18).

3.4.1.1. LEP [A2548G]/rs7799039 SNP

A study conducted by Hinuy et al. (19) demonstrated in a cohort of Brazilian women (n=228) that LEP G-2548A was linked with an increased risk of obesity after adjusting other study parameters, such as age, smoking, physical activity, high blood pressure, and coronary artery disease. It was observed that LEP G-2548A polymorphism increased the risk of obesity four times. Moreover, the circulating levels of leptin were positively related to this polymorphism. This indicates that LEP G-2548A polymorphism and a higher risk of obesity are associated with increased leptin levels. The LEP G-2548A polymorphism was also positively related to BMI in Brazilian women (19). It was observed in patients with breast cancer that LEP [A2548G]/rs7799039 polymorphism was associated with a higher risk of developing obesity. Furthermore, the women with LEP [A2548G]/rs7799039 polymorphism showed higher levels of blood glucose levels and did not respond to cancer treatment. It was observed that samples with higher expression of estrogen receptors also showed higher levels of circulating leptin indicating a positive association between these two. The cancer progression was also advanced to clinical stage III in these women. All these data indicated that LEP [A2548G]/rs7799039 not only increased the risk of obesity but also provided a favourable environment for the progression of breast cancer (20). In addition, obesity has been associated with higher leptin levels but lower leptin-receptor in obese people from western Mexico, compared to normal persons. It was observed that LEP-2548A could be used as a genetic marker to understand the less fat accumulation observed in the subjects (21).

3.4.2. Leptin Receptor

Leptin receptor or obesity receptor is categorized under the class I cytokine receptor family and found in six different isoforms. The long-form of this receptor is present in the hypothalamus where it plays a key role in energy balance. The Janus Kinase/Signal Transducer and Activator of Transcription (JAK/STAT) pathway is the main signaling pathway through which the leptin signaling occurs (22).

3.4.2.1. LEPR [Q223R]/rs1137101 SNP

In Mexican women (n-177) with breast cancer, a positive correlation was observed between the risk of obesity/overweight and LEPR rs1137101. The polymorphism was also found associated with higher levels of estrogen receptor expression and circulating leptin levels in these obese subjects (20). In another study conducted among Saudi females with polycystic ovary syndrome (PCOS), it was observed that rs1137101 was not associated with the development of PCOS. Although the study reported that women with PCOS showed significantly higher levels of waist/hip (W/H) ratio, serum leptin concentration, and triglycerides levels, compared to the control group, the genotype frequencies of rs1137101 (23) was not statistically different in comparison with the control group (24). Yiannakouris, Yannakoulia (23) recognized an association between p.R223Q polymorphism and the risk of obesity. The study demonstrated that when the R223 allele is present in a homozygous form, one can predict both BMI and percentage fat mass after adjusting for other parameters, such as age and gender. In the South Indian population, the Q223R polymorphism was found associated with higher levels of serum leptin, insulin, and W/H ratio. The study concluded that individuals with Q223R are at risk for developing obesity. Moreover, they also develop diabetes in later years, and therefore, Q223R is an important marker for the development of diabetes (25).

3.4.3. 5HTR2A

The serotonin or 5-hydroxytryptamine is one of the most studied biological amines. It acts via its receptors and controls several biological functions, such as appetite, smooth muscle contraction, platelet aggregation, and pain (26).

3.4.3.1. 5HTR2A [G1439A]/rs6311 SNP

Halder, Muldoon (27) have found an association between -1438 GG genotype and metabolic syndrome. It was observed that -1438 GG had been associated with W/H ratio and central adiposity. However, this polymorphism was not associated with other important biochemical parameters, such as fasting glucose and circulating lipids, including triglycerides and high-density lipoprotein cholesterol (HDL) (27). 1438G>A polymorphism is not significantly associated with the risk of developing obesity. However, in the obese category, individuals with the AA allele demonstrated lower BMI, compared to the G allele indicating an association with the BMI (28). The serotonin system plays a very crucial role in food intake, and it has been observed that the binding of serotonin receptors in the cerebral cortex is positively associated with BMI (29).

3.4.4. Ghrelin

Ghrelin is a 28 amino acid-long peptide hormone that plays a central role in appetite regulation, glucose metabolism, energy expenditure, and nutrient sensing (30). An abnormal ghrelin signaling has been implicated in obesity, diabetes, and insulin resistance.

3.4.4.1. GHRL [L72M]/rs696217 SNP

Ghrelin polymorphism [L72M]/rs696217 has been associated with a higher activity of butyrylcholinesterase (BChE). The BChE enzyme has already been linked with BMI. Taken together, this SNP plays a role in altering metabolic processes, such as fat utilization in the body. GHRL SNP [L72M]/rs696217 plays a role in obesity due to its effect on BChE expression, fat metabolism, and ghrelin expression. In their study, Ukkola, Ravussin (31) showed that a mutation at codo*n 72 o*f the preproghrelin gene (*Leu72Met*) triggers an early onset of obesity (n=96). It was also demonstrated that tall and obese children with L72M SNP displayed a higher BMI, compared to the wild-type allele. Moreover, this SNP also modulated the overall glucose metabolism because it reduced the secretion of key glucose metabolizing hormone insulin. A longitudinal study of the Japanese population (n=2,238) showed that individuals with L72M polymorphism displayed higher waist circumference, W/H ratio, and more bodyweight at the age of 18, compared to the control group. However, the SNP was not linked with other important players in obesity, such as lipid profile, glucose, and

insulin concentration. The study concluded that the 72Met allele is a risk factor for weight gain in middle-aged men (32).

3.4.5. FTO

The *FTO* gene encodes for a protein called 2-oxoglutarate-dependent nucleic acid demethylase. This enzyme has been involved in DNA repair and energy homeostasis (29).

3.4.5.1. FTO [T23525A]/rs9939609 SNP

In a Romanian population (n=387), the carriers of the AA genotype showed 2.02 times higher risk for developing obesity, compared to AT+TT genotype carriers. These individuals displayed higher body weight and W/H ratio. They also showed increased serum levels of important metabolic parameters, such as fasting glucose, triglycerides, and total cholesterol. The levels of leptin and adiponectin were also higher (27). Harvest et al. reported that rs9939609 has been associated with increased BMI in diabetics (n=597) but not with diabetic nephropathy (28). Psoriasis is often found with obesity and other metabolic disorders. Malgorzata et al. showed that Psoriasis patients with rs9939609 polymorphism showed a higher risk of developing obesity and insulin resistance. They also demonstrated a higher concentration of inflammatory cytokine C-reactive proteins (33).

3.4.6. Adiponectin

It is secreted by adipocytes and plays a critical role in the overall energy balance in the body due to its modulating effects on glucose metabolism and fatty acid metabolism. Lower levels of adiponectin have been linked with a reduced risk for type 2 diabetes mellitus. Adiponectin levels are inversely proportional to the overall fat mass in the body (34).

3.4.6.1. ADIPOQ [G276T]/rs1501299 SNP

rs1501299 SNP (G276T) of the ADIPOQ gene has been associated with prediabetes condition. It was observed that rs1501299 SNP effects are possibly due to insulin resistance (35). A study in the Iranian population observed that SNP 45T/G rather than SNP 276G/T plays a role in the onset of type 2 diabetes in obese individuals (n=50) (36). Filippi et al. (37) demonstrated that obese individuals with +276G>T SNP showed higher BMI, insulin levels, and homeostatic model assessment for insulin resistance (HOMA-IR). The adiponectin polymorphism determined the risk of insulin resistance in individuals, especially lean individuals (37). It was observed in a Japanese study that 276G→T SNP was significantly linked with higher body weight, plasma glucose, insulin resistance, and W/H ratio. The homozygotes also displayed lower levels of serum adiponectin (38).

3.4.7. PEMT

Phosphatidylethanolamine N-methyltransferase (PEMT) is an important enzyme in the synthesis of choline. It plays a very important role in lipid synthesis, lipid transport, and helps maintain the integrity of the cell membrane. The PEMT has been implicated in non-alcoholic fatty liver disease (NAFLD).

3.4.7.1. PEMT [V175M]/rs7946 SNP

Garner, Conn (39) demonstrated that V175M mutation increased the risk for NAFLD. This mutation is a loss of function mutation and hinders the normal synthesis of very-low-density lipoproteins leading to the development of NAFLD. The PEMT [V175M]/rs7946 SNP was associated with increased BMI in the Hispanic population (39). A coding non-synonymous SNP (rs7946) has been linked with a marginal increase in W/H ratio (40). It was also reported that PEMT [V175M]/rs7946 SNP was linked with obesity and NAFLD due to its modulating effects on betaine metabolism in the Mexican population (41).

3.4.8. PLIN

Perilipin is a protein that is associated with adipocytes and surrounds the lipid droplets. Perilipin plays a central role in fat metabolism in the adipocytes by interacting with lipases. It has been implicated in obesity and insulin resistance due to its modulating effects on fat accumulation, lipolysis, and fat metabolism.

3.4.8.1. PLIN [G1482A]/rs894160 SNP

It was demonstrated that Chinese individuals with genotype AA (rs894160) showed a higher risk of obesity. These individuals were also prone to develop

diabetes due to a high waist circumference (42). PLIN locus has been linked with an increased risk of developing obesity in both Mallaya and Indian populations but not in the Chinese population. This indicated that the increased risk of obesity due to PLIN locus is dependent on ethnicity (43). Smith, Tucker (44) demonstrated a gene-diet link between the PLIN locus and the intake of dietary carbohydrates. It was observed that individuals with a higher intake of complex carbohydrates were protected against obesity if they possess a minor allele. However, the same allele increased the risk of obesity in individuals with lower carbohydrate intake. The study concluded that the findings can be efficiently used in suggesting the best dietary plans in obese people (44). Furthermore, PLIN 11482G-->A polymorphism increased the risk of developing obesity and insulin resistance among Asian women. The study reported a possible gene-diet interaction of PLIN polymorphism with the intake of saturated fatty acids and carbohydrates (45).

Table 1 shows some important genes, their variants, and risk alleles associated with obesity. The risk allele is actually "the allele that confers a risk of developing the obesity". The risk allele is statistically significantly associated with the risk of having a disease under study.

Table 1: Genes, genetic variations, and risk alleles associated with obesity. The risk allele is the allele conferring risk of developing obesity as determined by Genome-Wide Association Studies

Genes	Genetic variation	Risk allele	References
NDNF	rs10018902	A	(46)
ADGRV1	rs10074525	G	(46)
TRIB2	rs10198628	A, A, A	(47)
INADL	rs1056513	A	(46)
MTRNR2L5, PCDH15	rs10740609	T	(48)
BICD1	rs10844154	C	(49)
TMEM45B	rs10894147	A	(46)
SEC16B	rs10913469	C	(50)
FAM214B	rs10972341	G	(49)
CDK17, CFAP54	rs11108495	T	(48)
LPP	rs1152846	G	(49)
TRAV39, TRAV40	rs11845134	A	(46)
LIPC	rs11857380	C	(46)
LOC100419639, LOC105371365	rs11863065	A, A	(46)
ACTR3B	rs11981919	A	(46)

Genes	Genetic variation	Risk allele	References
MGAT1, OR2Y1	rs12517906	G	(49)
SQRDL	rs12594515	C	(48)
TPM1	rs12595433	A	(46)
LINC00083	rs12760731	A	(46)
MC4R	rs12970134	A	(50)
NBEAL1	rs16839626	A	(46)
ANAPC4	rs16877106	C	(48)
OR1K1, PDCL	rs16912238	G	(46)
LOC105378769	rs17124318	C	(48)
LOC105379082	rs17668565	G	(46)
ADAMTS14	rs1816002	G	(49)
ZNF521	rs1840440	G	(49)
LOC101928241, LOC105378866	rs1973993	C	(50)
WDR59	rs2042415	A	(46)
CEP120	rs2115172	A	(50)
FAM19A2	rs2198776	A	(46)
CYLC2, LINC00587	rs2210533	A	(46)
CHN2, CPVL	rs245914	A	(46)
TFPT	rs254262	A, A	(46)
LOC105378797	rs2568958	A	(50)
ANKRD20A19P	rs2765086	G	(49)
AIF1, NCR3	rs2844479	T	(50)
KCTD15	rs29941	C	(50)
PPIAP33	rs4400445	A	(46)
MIR378C	rs4750829	A	(46)

Genes	Genetic variation	Risk allele	References
COL4A1	rs494558	G	(46)
MOXD1	rs589756	A	(46)
BDNF	rs6265	G	(50)
GABRB1	rs6289	G	(46)
FTO	rs6499640	A	(50)
	rs6587515	A, A	(47)
TM4SF19-TCTEX1D2, UBXN7	rs6774852	G	(46)
BCDIN3D, RPL35AP28	rs7138803	A	(50)
CEP112	rs7209395	G	(49)
MTIF3, RNU6-63P	rs7336332	G	(50)
RBFOX1	rs7403856	A	(46)
BDNF-AS	rs7481311	T	(50)
SH2B1	rs7498665	G	(50)
LOC105373353	rs7561317	G	(50)
ETV5	rs7647305	C	(50)
ANLN, AOAH	rs7777593	A	(46)
DUPD1	rs7919006	G	(49)
FAS, MIR4679-2	rs7920888	A	(46)
PGBD3P1	rs7974425	G, G	(46)
FTO	rs8050136	A	(50)
LOC105375732, LOC105375733	rs907121	C	(48)
BDNF-AS	rs925946	T	(50)
RPL21P59, RPL7P20	rs9313296	C	(48)
CDKAL1	rs9460521	A	(46)

4. Conclusions

Diet, exercise, and behavioural strategies are used in weight management therapies to reduce calorie consumption and increase physical activity. The fact that existing approaches are focused on dietary guidelines for general populations and do not reflect the effect of hereditary factors and subsequent interaction with the environment may elucidate why there has been a modest improvement despite constant attempts. Personalized nutrition is an innovative intervention for the management and treatment of disease that considers genetic data, and some other factors, including age, ethnicity, physiological and pathological condition, and environmental factors, especially personal lifestyle. Genetic studies have contributed significantly to a better knowledge of how genetic polymorphisms and epigenetic changes are involved in the genesis of many clinical diseases, such as obesity, as well as how these genetic changes potentially modulate therapeutic responses.

Several genes related to obesity have been discussed in this study including Leptin, Ghrelin, FTO, PEMT, PLIN, and Adiponectin (7). These genes and their SNPs are identified as key contributors to promoting and developing obesity (9, 22). Personalized nutritional guidelines based on the genetic profile of each individual are an effective option for the management and cure of obesity (13). As a result of this information, researchers are looking for genetic and epigenetic biomarkers that might predict the likelihood of developing serious diseases. Dietary interventions based on specific nutrients and bioactive food components that can alter epigenetic marks and gene expression are other possible therapeutic targets. Such research discoveries are opening the way for the invention of novel methods for the control, management, and cure of obesity and other common serious diseases with a genetic origin in the realm of personalized nutritional approaches; however, caution should be considered in this regard.

Chronic metabolic diseases, such as obesity and associated disorders, are currently a major concern for researchers due to their increasing adverse effects on the general population and lack of effective treatment and management strategies. Obesity is rising at an unprecedented pace, and urgent attention is needed to curb this global health issue. Several epidemiological studies have established that environmental factors, such as physical activity and eating behavior along with genes and their variants play a very important role in deciding the susceptibility of an individual for the development of obesity. Recent approaches in the field of nutrigenetics have shown promising results. The completion of the Human Genome Project is a landmark achievement for the scientific community, and several genes associated with various disorders have been identified using the genome data obtained after the Human Genome Project. The time has come to utilize this vast data for “public health genomics” to find out effective solutions for chronic disorders, such as obesity. Current research studies bolster our opinion that dietary interventions are highly effective when the genetic makeup of an individual is also considered in deciding the intervention.

Authors' Contribution

Study concept and design: A. G.
Acquisition of data: P. K. M. and A. G.
Analysis and interpretation of data: S. N.
Drafting of the manuscript: A. G. and S. P.
Critical revision of the manuscript for important intellectual content: A. M.
Administrative, technical, and material support: P. K. M.

Conflict of Interest

The authors declare that they have no conflict of interest.

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