Discoveries in gene-environment interactions that influence CVD, lipid traits, obesity, diabetes, and hypertension appear to be able to influence gene therapy.

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Abstract

In a relatively short amount of time, significant progress has been made in discovering gene-environment interactions that influence CVD, lipid traits, obesity, diabetes, and hypertension. These correlations appear to change genetic vulnerability, which may help researchers better understand the genetic processes that influence CVD development in the future. In order to advance the field, further research is required to confirm initial comparisons, identify the biological processes by which environmental influences modify genetic risk, and investigate strategies that use this knowledge to influence clinical genetic therapy outcomes.

The leading cause of morbidity and death in the world is cardiovascular disease (CVD). 1 The majority of CVD cases are caused by modifiable risk factors including diabetes, asthma, obesity, dyslipidemia, smoking, lifestyle factors, psychosocial stressors, alcohol intake levels, and physical inactivity, which account for more than 90% of MI cases and more than 80% of stroke cases worldwide. 2 and 3 Despite the fact that CVD prevalence is decreasing in high-income countries, it is rising in many low-and middle-income countries as a result of rapid lifestyle changes, growth, and urbanization. Such noncommunicable cardiometabolic disorders, such as obesity, hypertension, and diabetes, have also increased as a result of these shifts. 4 and 5 Several ethnic groups in Canada, including South Asians, Aboriginals, Chinese, and Afro-Caribbeans, are at risk of contracting cardiometabolic diseases. 6 To reduce global CVD pressure, individual and population-level interventions aimed at improving modifiable cardiovascular risk factors are needed.

Our awareness of CVD is becoming increasingly genetically informed. Many genetic variants linked to increased CVD risk have recently been discovered in genome-wide interaction studies (GWAS), showing that CVD is a nuanced disorder with polygenic and environmental effects. 7, 8 Single nuclear polymorphisms (SNPs) have been shown to affect the progression of coronary artery disease (CAD), with all of them inducing atherosclerosis by mechanisms unrelated to typical risk factors. 9 other polymorphisms have been linked to lipid characteristics, diabetes, obesity, and hypertension. We are just beginning to comprehend the impact of genetics on cardiovascular disease, including how genetic and modifiable risk factors combine to affect cardiovascular risk.

When behavioural factors (such as smoking, physical exercise, or nutritional factors) alter the association between genetic polymorphisms and phenotypic characteristics, this is known as a gene-environment interaction. Some intriguing findings indicate that environmental conditions may alter the genetic risks associated with a variety of cardiometabolic diseases. Gene-environment interactions could lead to the discovery of new pathways in the production of CVD, as well as a better understanding of how genetic factors influence cardiovascular risk. Though the most exciting opportunity for this area of study is the use of gene-environment associations to guide possible "personalized" approaches to treatment, this application has yet to be proved. Several analytical hurdles must be solved in order to properly assess the therapeutic efficacy of these experiences.

Furthermore, although certain associations have been reliably found in different cultures, the majority of them have not been adequately replicated. This review look at the current state of knowledge about gene-environment interactions in CAD, obesity, diabetes, hypertension, and lipid metabolism, as well as the conceptual hurdles that must be addressed in order to advance cardiovascular gene-environment science.

The majority of GWAS-identified polymorphisms increase MI risk in small to moderate ways, accounting for around 10% of the inherited variance in disease growth.7, 9 As a result, the majority of heritable CAD risk remains unaccounted for, which may be clarified by alternate genetic mechanisms such as gene-environment interactions, gene-gene interactions, and unusual polymorphisms with significant effects or genetic variations with limited effects that are difficult to diagnose with current techniques.

Polymorphisms in the 9p21 chromosomal region have been linked to a variety of CVD symptoms. 18 The 9p21 mutation is widespread (up to 50% of people in certain ethnic groups carry a risk allele) and one of the most powerful genetic predictors of CAD, raising CAD risk by 1.20 to 1.29 times per risk allele. 7 and 8

About its strong link to vascular risk, the mechanisms by which 9p21 acts remain largely unclear. Polymorphisms in a noncoding region of DNA are thought to influence CAD production by controlling the expression of neighboring genes. Polymorphisms in 9p21 have been found to occupy enhancer regions that influence the protein binding of the signal transducer and activator of transcription 1 (STAT1), which regulates the expression of the neighboring CDKN2B gene and CDKN2BAS (also known as ANRIL) within the 9p21 area. 19

The most powerful behavioral risk factor linked to the growth of CAD is smoking. 2 Interactions between genes that control inflammatory pathways and smoking have been discovered in many studies assessing genetic CVD risk. Xie et al. found a significant interaction between the rs20417 SNP in PTGS2, which encodes the cyclooxygenase-2 enzyme, and CAD risk (P for interaction = 0.009) in a case-control study of 900 participants. 21 However, interpretation of this interaction with smoking should be done with caution because the gene has not been independently associated with CVD risk; this would be expected given the high pr. The association between APOE polymorphisms and CVD risk tends to be altered by smoking. Major isoforms of apolipoprotein E.22 are encoded by APOE polymorphisms (2, 3, 4).

Humphries et al. found a substantial rise in CAD risk associated with the 4 allele (OR, 2.79; 95 percent CI, 1.59-4.91) in smokers relative to nonsmokers in a study of 2258 males, while 2 carriers and 3/3 carriers did not (P value for association = 0.007). 11 Gustavsson et al. also looked at the relationship between APOE polymorphisms and smoking status in 6269 Swedish people, finding that CAD risk due to smoking was slightly smaller in female 2 carriers (OR, 1.33; 95 percent CI, 0.70-2.52) compared to female carriers of the 3/3 (OR, 1.72; 95 percent CI, 1.29-2.31) or 4 (OR, 3.62; 95 percent CI, 2.32–5.63) alleles (While the interaction was not substantial (P = 0.09), smoking-related CAD risk was trending lower in men carrying the 2 polymorphism compared to the other genotypes. 12 While interesting, further research is needed to establish if the observed relationship is generalizable.

Metabolism of Lipids

Over the last few decades, improvements in diet and lifestyle have resulted in a substantial decrease in serum cholesterol levels in North America and Western Europe, which has been counterbalanced by a rise in emerging regions such as Southeast Asia and the Pacific. 23 GWAS has identified 95 loci that contribute to high-density lipoprotein (HDL), low-density lipoprotein (LDL), or triglyceride metabolism, among other lipid traits. 24 The interaction of genetic and environmental influences on lipid metabolism is still being studied, with the majority of results coming from single research.

Since these dietary components tend to change metabolic and inflammatory pathways, as well as potentially modify CVD risk, there has been a lot of interest in studying gene-diet interactions associated with n-3 and n-6 polyunsaturated fatty acids (PUFAs) (also known as-3 and-6 fatty acids). 25 About 90% of PUFA intake is made up of the n-6 PUFA linolenic acid (LA) and the n-3 PUFA-LA (ALA). 26 Linoleic acid is contained in edible oils and margarines and is metabolized to arachidonic acid; ALA is found in green vegetables, nuts, and certain oils and is metabolized to eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) (DHA). EPA and DHA can also be used in marine foods. Eicosanoids and docosanoids, which have strong anti-inflammatory effects, are produced by EPA and DHA. 27, 28

PUFAs seem to alter the effects of many genes involved in lipid metabolism, including the FADS gene cluster, which encodes desaturases that metabolize LA and ALA, according to several reports. 24

In a sample of 3575 Europeans, PUFA consumption substantially altered the influence of the FADS1 polymorphism (rs174546), with higher HDL concentrations in rs174546-C carriers who ingested large levels of n-6 PUFAs and no effect on those who consumed low amounts of n-6 PUFAs (P for interaction = 0.02). 28 ABCG1, which encodes the adenosine triphosphate binding cassette transporter G1, which mediates cholesterol transfer from cells to HDL lipoproteins, is another gene that regulates lipid metabolism and is affected by PUFA intake. 29 In a study of 2435 Spanish participants, Abellan et al. looked at the impact of dietary PUFA intake on the interaction between a polymorphism in the ABCG1 gene (rs4148102) and different lipid phenotypes. High PUFA consumption was linked to higher mean total cholesterol (246.8 32.9 vs 198.0 37.5) and LDL concentration (159.0 32.6 vs 114.3 33.1) in homozygous carriers of the rs4148102-A allele, but no major variations were seen with low PUFA consumption (P for correlation = 0.006 and 0.003 for total cholesterol and LDL, respectively). Despite the fact that ABCG1 is predominantly involved in HDL metabolism, there was no clear relationship between ABCG1 polymorphisms and HDL in relation to PUFA intake. 30

Additional behavioral influences have been shown to influence gene-lipid metabolism interactions. In 1123 European participants, Smith et al. looked at the impact of sedentary behavior, as measured by screen viewing time, on the relationship between LIPG polymorphisms (rs2000813 and rs6507931) and HDL concentration. HDL was slightly lower in female homozygous carriers of the rs6507931-T allele compared to C allele carriers in participants reporting high screen viewing times (2.6 h/d; P = 0.005), although there was no major genetic impact in participants reporting low screen viewing times (P for association = 0.003). 31 Higher HDL concentrations have also been found in Chinese individuals that are homozygous for the rs2000813-T allele and drink alcohol, relative to nondrinkers. 32

The effects of genes that control lipid metabolism tend to be altered by smoking. The ABCG5 and ABCG8 genes are responsible for transporting cholesterol from enterocytes to the interstitial lumen. 33, 34 Polymorphisms in the ABCG5/8 loci have been found to interfere with smoking in Latin Americans. ABCG5 polymorphisms impacted HDL and total cholesterol levels in smoking, while ABCG8 polymorphisms controlled triglyceride and very low density lipoprotein levels. Nonsmokers' lipid levels were unaffected by these polymorphisms. Smoking is thought to reduce HDL by downregulating ABCG5/8 and inhibiting reverse cholesterol transport.

However, further research is required to validate the mechanisms behind this interaction. 34 Several gene-environment associations that seem to influence lipid metabolism have been identified, but they have mostly been published in single studies. Replication trials are also needed to provide further definitive proof for these results.

Diabetes and Metabolic Syndrome are two terms that are used interchangeably.

Over the last three decades, the global burden of diabetes has risen sharply. 5 Since diabetes is linked to both genetic and environmental causes, correlations between the two are likely to play a role in its rising prevalence. However, little research has been conducted to examine how environmental factors influence the impact of genetic polymorphisms linked to diabetes, metabolic syndrome, and insulin resistance.

The Diabetes Prevention Program Research Group found a 58 percent decrease in diabetes risk with a lifestyle-based intervention (consisting of an instructional program promoting food, exercise, and behavior modification) relative to placebo in a randomized controlled study with people at higher risk for developing diabetes. 35 Florez et al. examined whether polymorphisms (rs12255372 and rs7903146) in the TCF7L2 gene impaired diabetes risk and whether this risk could be changed by treatments in 3548 participants with genetic evidence. In the placebo population, homozygous carriers of the rs7903146-T allele were at an elevated genetic risk of contracting diabetes (hazard ratio, 1.81; 95 percent CI, 1.21-2.70; P = 0.004), but there was no such risk in the lifestyle intervention community (hazard ratio, 1.15; 95 percent CI, 0.68-1.94; P = 0.60). Although the research was not possibly powered to test relationships, formal interaction measurements were nonsignificant. Additional studies have shown that nutritional factors such as whole grain intake and glycemic index change the diabetes risk associated with TCF7L2 polymorphisms. 36 and 37

Diabetes is clearly a polygenic disorder, as evidenced by GWAS, with many loci now known to affect diabetes risk. TCF7L2 (rs12255372), HHEX (rs1111875), CDKAL1 (rs7756992), IGF2BP2 (rs4402960), SLC30A8 (rs13266634), WFS1 (rs10010131), CDKN2A/B (rs564398, rs10811661), PPARG (rs1801282), and KCNJ11 are among 10 genes correlated with diabetes (rs5219). Participants with a high gene score (12) had a higher risk of diabetes while they ate a mostly western diet (OR, 2.06; 95 percent CI, 1.48-2.88 for the highest western diet quartile), while those with a low gene score (10) did not (P for interaction = 0.02), implying that a western diet affects diabetes risk among those genetically predisposed to it. 38

Saturated fats and polyunsaturated fatty acids (PUFAs) have been studied in gene-environment studies after several observational studies suggested that dietary fat intake affects insulin response and diabetes danger. 39, 40, and 41. Phillips et al. looked into how the ACSL1 gene (rs9997745), which encodes long-chain Acyl CoA Synthetase, an essential enzyme for fatty acid metabolism, interacts with dietary fat intake and diabetes danger. 42 and 43 A high saturated fat diet, but not a low saturated fat diet, raised the risk of metabolic syndrome in homozygous carriers of the rs9997745-G allele. Furthermore, the polymorphism raised the risk of metabolic syndrome in people who ate a low-n-3 PUFA diet, but not in people who ate a high-n-3 PUFA diet. 43 Similar to n-3 PUFA to serum fatty acid levels, the metabolic syndrome vulnerability associated with other genes that control inflammatory pathways (such as LTA and TNF-) tends to differ. 14 Ferguson et al. investigated whether serum saturated fatty acid concentrations affected insulin resistance linked to the ADIPOQ (rs266729) gene, which encodes adiponectin, and the ADIPOR1 (rs10920533) and ADIPOR2 (rs10920533) genes, which encode adiponectin receptors (rs6489323). The homeostasis model assessment-insulin resistance index was higher in the high serum saturated fatty acid group compared to the low serum saturated fatty acid group in homozygous carriers of the rs266729-C (ADIPOQ) minor allele; however, this effect was less pronounced in carriers of the G allele (P for interaction = 0.01). In homozygous carriers of the rs10920533-A (ADIPOR1) minor allele, a related effect was observed (P for association = 0.004). Saturated fatty acid accumulation has little effect on ADIPOR2 polymorphisms. 44 While replication experiments are required to validate any of these findings, these findings indicate that saturated fatty acid consumption alters the effects of multiple genes involved in lipid metabolism, inflammation, and insulin regulation.

Obesity is a problem that affects many people.

Obesity has doubled in prevalence over the last 30 years, affecting 9.8% of men and 13.8 percent of women around the world. North America has the highest mean body mass index (BMI). 4 Dietary shifts and a more sedentary lifestyle have created an "obesogenic" climate, with individuals with a higher genetic risk manifesting the obesity phenotype more often. Several behavioral factors can influence the impact of genetic polymorphisms on obesity risk.

Polymorphisms in the FTO gene contribute greatly to hereditary interindividual weight heterogeneity and raise obesity incidence by 1.6 times per risk allele. 46, 45 A strong relationship between polymorphisms in the FTO gene and physical activity has been discovered by meta-analysis. Kilpelainen et al. examined the role of physical exercise in the relationship between FTO polymorphisms (rs9939609 or an SNP in high correlation disequilibrium) and obesity risk in a meta-analysis of 45 studies in adults (218,166 participants) and 9 studies in children (19,268 participants). Based on standardized standards, participants were classified as "active" or "inactive." 15 Each rs9939609-A risk allele was linked to an increased obesity risk of 1.23 (95 percent CI, 1.20-1.26) and a 0.36 rise in BMI in adults. Physical activity decreased the effect of the FTO risk allele on BMI by 30% (P value for interaction = 0.005) and reduced the risk of obesity by 27% (P for interaction = 0.001). In infants, there was no evidence of a gene-physical activity relationship. 15 An adequate sample size may be reached by meta-analysis to assess the impact of the FTO-physical activity relationship on obesity risk with greater confidence, adding to the growing body of evidence that hereditary obesity risk is responsive to physical activity level. While prospective research evaluating physical activity interventions on obesity risk associated with FTO has been inconclusive, identifying this gene-environment association could guide potential behavioral therapies targeting those at elevated genetic risk. 16, 47, and 48. More research is needed to see how this relationship can be used to create interventions that impact health results.

Saturated fat consumption tends to affect the risk of obesity associated with FTO. In separate Caucasian (rs9939609) and Latin American (rs1121980) populations, Corella et al. discovered an important association between increased saturated fat consumption and FTO polymorphisms on BMI. BMI was higher in homozygote carriers of the risk alleles who ate a high-fat diet, but there was no major genetic impact in those who ate a low-saturated-fat diet. 49

Saturated fat consumption has also been found to interfere with APOA2 polymorphisms to affect obesity in people of various ethnic backgrounds. 50 Corella et al. discovered that the APOA2-265T > C polymorphism has a consistent gene-diet relationship in Caucasian and Hispanic populations. The genetic risk of obesity varied greatly depending on saturated fat consumption across populations, with C allele homozygotes eating large levels of saturated fat having a 1.84 (95 percent CI, 1.38-2.47) increased risk of obesity relative to those consuming low amounts of saturated fat having a 0.81 (95 percent CI, 0.59-1.11) increased risk. 17 The findings were also repeated in Asian and Mediterranean cultures, indicating that this relationship is generalizable across ethnic groups. 51

Several studies have looked at the impact of gene-environment interactions in loci controlling inflammatory pathways, as proinflammatory pathways influence obesity growth. Razquin et al. looked at whether a Mediterranean diet (MD) changed the association between IL-6 genetic polymorphisms (-174G > C) and weight gain over a 3-year cycle in a prospective trial. Patients were assigned to one of three groups: a low-fat diet (control), a Mediterranean diet with virgin olive oil, or a Mediterranean diet with nuts. Weight gain was slightly different in-174-CC carriers on an MD and virgin olive oil diet relative to the control diet (P for association = 0.028). 52 Similarly, Jourdan et al. discovered that the genetic likelihood of obesity associated with many genes encoding inflammatory cytokines, such as IL-6 and IL-2, varied depending on the amount of PUFA in the membrane. 53 These findings shed light on the inflammatory pathways that can be influenced by dietary conditions in order to change hereditary obesity risk. However, unlike the robust gene-environment interactions associated with FTO and APOA2, dietary interactions with genes controlling inflammatory pathways have mostly been observed in single populations, and further replication is needed to validate these results.

Hypertension

Small changes in blood pressure will lead to significant decreases in cardiovascular events in the population, highlighting the importance of individual and population-based interventions to improve blood pressure regulation. Mean systolic blood pressure has been declining in many high-income regions as a result of successful medical therapies and lifestyle improvements, but it has been rising in low-and middle-income regions, such as Africa and Asia, in line with overall CVD patterns. 54

Few studies have looked at gene-environment associations with blood pressure, and further research is required to confirm these findings, especially in different ethnic groups, so that their generalizability can be better determined. Pan et al. discovered an important link between alcohol intake and hypertension risk in 1575 Chinese people, which was linked to a polymorphism (-344T > C) in the CYP11B2 gene. Hypertension risk was slightly higher in TT (OR, 2.9; 95 percent CI, 1.4-5.7) and TC (OR, 3.0; 95 percent CI, 1.5-5.7) allele carriers compared to the CC genotype (OR, 1.2; 95 percent CI, 0.5-3.0) allele carriers of those who consumed less than 200 g/d of alcohol (P value for association = 0.048). 55

Surprisingly, some research shows that occupational and community exposures can alter blood pressure genetic control. Menni et al. found that presumed work regulation changed the relationship between a polymorphism (rs11210278) in the EDN2 gene, which encodes endothelin 2, and ambulatory systolic blood pressure in 924 European participants. 56 For the EDN1 gene, homozygous carriers of the rs5369-G allele who indicated work strain had slightly higher systolic blood pressure than heterozygous allele carriers, with no variations found in those who recorded no job strain. 57 Chronic noise sensitivity can help to reduce the risk of hypertension linked to ACE insertion/deletion (I/D) and the ACE G2350A polymorphism. In a single sample of 385 South Asian males, ACE-DD carriers exposed to noise had a 4.49 (95 percent confidence interval, 1.55-12.99) elevated risk of hypertension, while ACE-ID and ACE-II carriers had chances of experiencing hypertension of 2.84 (95 percent confidence interval, 1.3-6.11) and 2.95 (95 percent confidence interval, 0.59-14.65) respectively. As it came to the ACE G2350A polymorphism, GG and GA carriers (OR, 3.97; 95 percent CI, 1.63-9.68) who were regularly prone to loud noise had a higher chance of hypertension than AA carriers (OR, 3.71; 95 percent CI, 1.41-9.76). (OR, 1.20; 95 percent CI, 0.33-4.36). Formal interaction experiments, on the other hand, were not published, and further research is required to provide more conclusive findings. 58

Current Gene-Environment Challenges and Future Directions Cardiovascular Studies

Based on the gene-environment associations that have been discovered so far, it appears that behavior and lifestyle play a significant role in changing the genetic likelihood of developing CVD. The interaction of environmental and genetic factors is complicated, and several key steps are needed to further clarify this area. The lack of replicated research, the need for significant sample sizes, enhancing the accuracy of calculating environmental conditions, and determining clinical impact are all major obstacles in the study of gene-environment interactions in cardiometabolic diseases. ten to fifty To understand the mechanisms of gene-environment interactions found at the population level, translational research is also needed.

Several gene-environment associations have been shown to affect cardiovascular risk, but the mechanisms underlying these effects are still unclear. Environmental factors can alter risk by influencing downstream metabolic pathways or by epigenetic or transcriptional mechanisms that regulate gene expression, but further research is required to validate these hypotheses. 59, 50 Understanding the mechanisms by which these relationships change risk is critical for a better understanding of CVD production and the development of innovative CVD preventive strategies.

The lack of published replicated research for most observed gene-environment interactions is a major methodological issue in this area. As a consequence, there's a chance of publishing bias, which may lead to an overabundance of favorable outcomes in the latest literature. In the future, replication should be a higher priority in gene-environment interaction science, either by testing several, separate populations within the original study or in subsequent studies. Furthermore, several associations have been established in single ethnic groups, but their effect on other ethnic groups remains uncertain. The impact of genetics on a variety of traits has been found to differ depending on race. It's also possible that gene-environment associations differ between ethnic groups, highlighting the importance of studying these influences in different ethnic groups. 60, 61 One barrier to reproduction is that human genetic polymorphisms have comparatively limited consequences in complex disorders, necessitating massive sampling populations to classify genetic effects, research associations, and reproduce results. Meta-analysis is a useful method for getting around this constraint. This was shown successfully with research examining the effects of the FTO-physical activity interaction on obesity risk, in which inconclusive outcomes were found through individual studies, but meta-analysis of results offered a sufficient sample size to identify a meaningful effect, further supporting the generalizability of this interaction. 15

The heterogeneity found in assessing environmental exposures between studies is another methodological weakness in gene-environment science. While certain factors (such as nutritional, physical activity, and psychosocial factors) are simple to quantify, others (such as dietary, physical activity, and psychosocial factors) are more complex. A study's prejudice may be caused by incorrect characterization of environmental exposures. Furthermore, using various measurement instruments will lead to inconsistent outcomes across experiments, limiting the opportunity to repeat findings. This possible cause of error can be reduced by using consistent methods of characterizing environmental variables. Consistent concepts were used to describe both continuous and dichotomous physical activity measurements in the meta-analysis of FTO-physical activity relationship findings, for example, to include a more uniform characterization of the environmental exposure for analysis. 15 Several validated food frequency questionnaires exist to quantify components of food consumption (such as fat intake and vegetable intake) in gene-environment cardiovascular research, and recommendations for using these instruments in gene-environment cardiovascular research may be helpful to provide a more reliable characterization of these environmental factors through studies.

The biggest question in this area is whether these new discoveries can lead to techniques that change personal behavior or clinical practice. Given the field's brief history, it is also unclear if treating genetically vulnerable people would have a therapeutic benefit over current population-based health promotion policies. In theory, this tailored approach to management may have additional benefits, since "at risk" individuals could be more likely to improve their behavior and may benefit more from interventions as a result of their higher baseline vulnerability than the general population. However, it's unknown if such an advantage would be seen in primary preventive communities with a higher genetic risk of CVD, which is a key field for future study. Researchers also found that telling participants of their genetic vulnerability has only a minor impact on their conduct. Awareness of genetic evidence influenced dietary behavior favorably in a meta-analysis of 14 findings, but there were no benefits of smoking or physical activity; and the studies involved were modest in scale and of low quality. 62, 165-191 As a result, more research is required before any therapeutic implementation to see whether behavioral approaches based on gene-environment interaction information will increase clinical outcomes, preferably in the context of prospective studies or randomized controlled trials.

Final Thoughts

Important progress has been made in identifying gene-environment associations that affect CVD, lipid traits, obesity, diabetes, and hypertension in a comparatively short period of time. These associations tend to alter genetic risk, which may contribute to a deeper understanding of the genetic mechanisms that affect CVD progression in the future. Additional study is needed to validate initial comparisons, establish the biological mechanisms by which environmental factors modify genetic risk, and explore techniques that use this information to impact clinical outcomes in order to advance the field.

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