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Impact of rs16928751 (+795 G/A) SNP in Adiponectin Receptor 2 on Type 2 Diabetes Risk in an Iraqi Population: A Case-Control Study

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Abstract

Adiponectin receptor 2 (ADIPOR2) contributes to adiponectin-mediated glucose and lipid metabolism. This case-control study investigated whether the ADIPOR2 single-nucleotide polymorphism (SNP) rs16928751 (+795 G/A) was associated with type 2 diabetes mellitus (T2DM) in an Iraqi study population. The study included 48 patients with T2DM and 32 controls. Genomic DNA was amplified by conventional polymerase chain reaction and analyzed by Sanger sequencing; fasting blood glucose (FBG) and glycated hemoglobin (HbA1c) were also measured. Genotype and allele frequencies were compared using categorical tests, while Hardy-Weinberg equilibrium was evaluated separately for each group. HbA1c and FBG were significantly higher in patients with T2DM than in controls (reported p-values below 0.001). The GG genotype occurred in 75.00% of patients and 78.12% of controls, the GA genotype in 18.75% of both groups, and the AA genotype in 6.25% and 3.13%, respectively. The A allele was observed in 15.63% of patient alleles and

12.50% of control alleles (reported odds ratio [OR] = 1.30; 95% confidence interval [CI]: 0.52–3.26). The AA genotype showed a numerically higher odds ratio (OR = 2.07; 95% CI: 0.21–20.80), whereas the GA genotype showed no increase in the reported estimate (OR = 1.00; 95% CI: 0.32–3.15). None of the reported genotype or allele comparisons reached statistical significance; similarly, the dominant (OR = 1.19) and recessive (OR = 2.07) model comparisons were not statistically significant. The findings therefore do not establish an association between rs16928751 and T2DM in this sample, although the confidence intervals are wide and do not exclude potentially meaningful effects. Larger, adequately powered investigations incorporating additional demographic and metabolic covariates are needed to clarify the possible contribution of this variant to T2DM susceptibility in Iraqi populations.

Keywords: adiponectin receptor 2, fasting blood sugar, polymorphism, type 2 diabetes mellitus

1. INTRODUCTION

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia arising from impaired insulin secretion, insulin action, or both. Disrupted glucose regulation is accompanied by alterations in lipid and protein metabolism and, over time, may contribute to microvascular and macrovascular complications [1, 2]. Of the major clinical forms of diabetes, including type 1, type 2, and gestational diabetes, type 2 diabetes mellitus (T2DM) accounts for the largest proportion of cases [3]. Its development is multifactorial, involving interactions among genetic susceptibility, epigenetic regulation, environmental exposures, and lifestyle-related factors. Pancreatic β -cell dysfunction, insulin resistance, and chronic low-grade inflammation contribute to progressive impairment of glycemic control [4]. The public-health burden continues to increase: the International Diabetes Federation estimated that 589 million adults aged 20–79 years were living with diabetes worldwide in 2024, with a projected increase to approximately 853 million by 2050 [5]. These trends strengthen the rationale for investigating genetic and metabolic factors associated with T2DM susceptibility. Adipose tissue participates in systemic metabolism through the secretion of adipokines, including adiponectin, whose circulating concentrations tend to be lower in individuals with obesity, insulin resistance, and T2DM [6, 7]. Adiponectin is an adipocyte-derived protein involved in the regulation of glucose homeostasis, lipid metabolism, and inflammatory signaling. Previous studies have examined variation in the adiponectin gene, *ADIPOQ*, and its possible association with metabolic phenotypes, including T2DM [8]. Adiponectin circulates in distinct oligomeric forms and exerts biological effects through the membrane receptors adiponectin receptor 1 (AdipoR1) and adiponectin receptor 2 (AdipoR2). Genetic variation in these receptors has been investigated in relation to insulin sensitivity and diabetes risk [9]. The human *ADIPOR2* gene is located at chromosome 12p13.33 [10]. In particular, the *ADIPOR2* variant rs16928751, also described as +795 G/A, has been investigated in populations with different genetic backgrounds. Iranian studies evaluated its genotype distribution and its relationship with insulin resistance and clinical responses to pioglitazone [11, 12]. Evidence from other populations suggests that reported associations may differ according to study design, phenotype, and ancestry, emphasizing the need to interpret findings within their population context [13, 14]. Against this background, the present case–control study examined the distribution of *ADIPOR2* rs16928751 (+795 G/A) genotypes and alleles among Iraqi patients with T2DM and healthy controls and assessed their statistical association with disease status. The study also compared fasting glucose and HbA1c measurements between groups to characterize the sampled metabolic profiles.

2. MATERIALS AND METHODS

This case–control study included 80 participants, comprising 48 patients with a diagnosis of T2DM and 32 healthy controls. Participants were recruited from outpatient clinics in Baqubah City and Baqubah General Hospital between March and October 2025. The participants were 20–70 years of age. Before any study-related procedures, all individuals provided written informed consent. According to the authors' account, the study was approved by the ethics committee of the University of Diyala and the Council of the College of Education for Pure Sciences.

After a 12-hour overnight fast, 5 mL of venous blood was collected from each participant. Approximately 3 mL was transferred into an ethylenediaminetetraacetic acid (EDTA)-containing tube for HbA1c measurement and genomic DNA extraction. The remaining sample was transferred to a tube without anticoagulant and centrifuged at $3,000 \times g$ for 10 minutes to separate serum. Serum specimens were stored at -20°C until analysis of fasting blood glucose (FBG). Sample identification and storage were maintained according to the laboratory procedures described by the investigators.

Genomic DNA was isolated from EDTA-anticoagulated blood using the ReliaPrep™ Blood gDNA Miniprep System (Promega, USA), according to the manufacturer's protocol. The reported DNA concentrations ranged from 50 to 100 ng/ μL , and the reported purity ratios were 1.7–2.0. The region encompassing the *ADIPOR2* rs16928751 (+795 G/A) variant was amplified by conventional polymerase chain reaction (PCR). Forward and reverse primers were designed with Primer3Plus to obtain a 322-bp amplicon; the sequences and annealing temperature are presented in Table 1. The amplified fragments were subsequently processed for Sanger sequencing to determine the genotypes.

Table 1. Primers used to amplify rs16928751 (+795 G/A) of the adiponectin 2 receptor

Primer	Sequence (5'–3')	Product size (bp)	Annealing temperature ($^{\circ}\text{C}$)	Source
Forward primer 1	AGCAACCCAGTTTGGCTCTT	322	58	Current study
Reverse primer 2	CCGTGCTCTTACCTGCTCTT			

Primers were supplied in lyophilized form by Bioneer (Republic of Korea) and reconstituted in nuclease-free water to prepare stock solutions at 100 pmol/ μL , following the manufacturer's instructions. Working solutions at 10 pmol/ μL were obtained by combining 10 μL of the stock with 90 μL of nuclease-free water. These working solutions were used for conventional PCR amplification of the target region.

Conventional PCR was performed in 25- μL reactions containing 5 μL of Bioneer PCR premix, 1.5 μL of each primer, 3

μL of genomic DNA template, and 14 μL of distilled water. The amplification protocol consisted of an initial denaturation at 94 °C for 5 minutes; 35 cycles of denaturation at 94 °C for 30 seconds, annealing at 58 °C for 30 seconds, and extension at 72 °C for 30 seconds; followed by a final extension at 72 °C for 5 minutes. PCR products were separated by electrophoresis on a 1.5% agarose gel stained with ethidium bromide at 90 V for 90 minutes. Fragment sizes were compared with a DNA ladder supplied by Intron (Republic of Korea), and the amplified bands were visualized using a UV transilluminator.

Successfully amplified PCR products were purified and submitted for Sanger sequencing using the same forward and reverse primers. Sequencing was performed by Macrogen (Republic of Korea) on an ABI 3730XL DNA Analyzer. The resulting chromatograms were inspected and analyzed with Geneious Prime, version 2020.1 (Biomatters Ltd., Auckland, New Zealand), to determine the rs16928751 (+795 G/A) genotypes. Sequence-based genotype calls formed the basis of the subsequent genotype and allele frequency analysis.

Statistical analyses were performed using IBM SPSS Statistics, version 27. Continuous variables were summarized using means and standard deviations, whereas categorical variables were described as counts and percentages. Between-group comparisons were assessed using Student's *t*-test for continuous variables and chi-square tests for categorical variables, as described in the original analysis. Hardy–Weinberg equilibrium (HWE) was evaluated separately in the patient and control groups using an exact test. Genotype and allele frequencies were compared between groups, and odds ratios (ORs) with 95% confidence intervals (CIs) were used to describe the associations of ADIPOR2 rs16928751 with T2DM under codominant, dominant, and recessive inheritance models. A two-sided significance threshold of $p < 0.05$ was applied. Because of the small number of participants with the AA genotype, exact or sparse-data methods should be considered when independently validating the reported model-specific significance tests.

3. RESULTS

Table 2 summarizes the participants' demographic characteristics and biochemical measurements. The patient group included 48 participants and the control group included 32 participants. The reported comparisons did not identify statistically significant differences in the proportion of male participants or in mean age within the sex strata. In contrast, HbA1c and FBG values were markedly higher in patients with T2DM than in healthy controls, with reported *p*-values below 0.001 for both measures in the male and female strata. These biochemical differences are consistent with the clinical classification of the study groups, but do not in themselves establish an association between the investigated SNP and T2DM.

Table 2. Demographic characteristics and blood measurements of the study groups, stratified by sex

Variables	Gender	Patients (N=48)	Control (N=32)	P-value
Gender	Male n (%)	26 (54.17)	15 (46.88)	0.52 NS
	Female n (%)	22 (45.83)	17 (53.13)	
Age (year)	Male	53.88±9.76	53.40±9.85	0.88 NS
	Female	56.81±10.75	51.29±9.14	0.09 NS
HbA1c (%)	Male	7.05±1.28	5.41±0.13	P<0.001 **
	Female	7.67±1.01	5.42±0.14	P<0.001 **
Fasting blood sugar (FBG) mg/dL	Male	173.69±45.90	115.60±4.82	P<0.001 **
	Female	196.00±36.03	115.82±5.21	P<0.001 **

* (P<0.05), ** (P<0.01), NS: non-significant.

The amplification of the target ADIPOR2 fragment is illustrated by the representative agarose-gel electrophoresis image in Figure 1; the image demonstrates the PCR product, rather than Hardy–Weinberg equilibrium. The observed and expected genotype distributions and the originally reported HWE exact-test results are presented separately in Table 3. The reported HWE *p*-values were 0.068 for patients and 0.392 for controls, both above the prespecified significance threshold. Accordingly, no statistically significant departure from HWE was detected in either group; this finding alone does not establish population representativeness or guarantee the validity of individual genotype calls. The GG genotype occurred in 36 of 48 patients (75.00%) and 25 of 32 controls (78.12%). The GA genotype was observed in 9 patients (18.75%) and 6 controls (18.75%), while the AA genotype occurred in 3 patients (6.25%) and 1 control (3.13%). The frequencies of the G and A alleles were 84.38% and 15.63%, respectively, among patients, compared with 87.50% and 12.50% among controls. As summarized in Table 4, the observed A-allele odds ratio was 1.30 (95% CI: 0.52–3.26), whereas the G-allele odds ratio was 0.77 (95% CI: 0.31–1.94). The reported allele comparison was not statistically significant ($\chi^2 = 0.104$, $p = 0.747$), and neither allele can be classified as a proven risk or protective factor on the basis of these data.

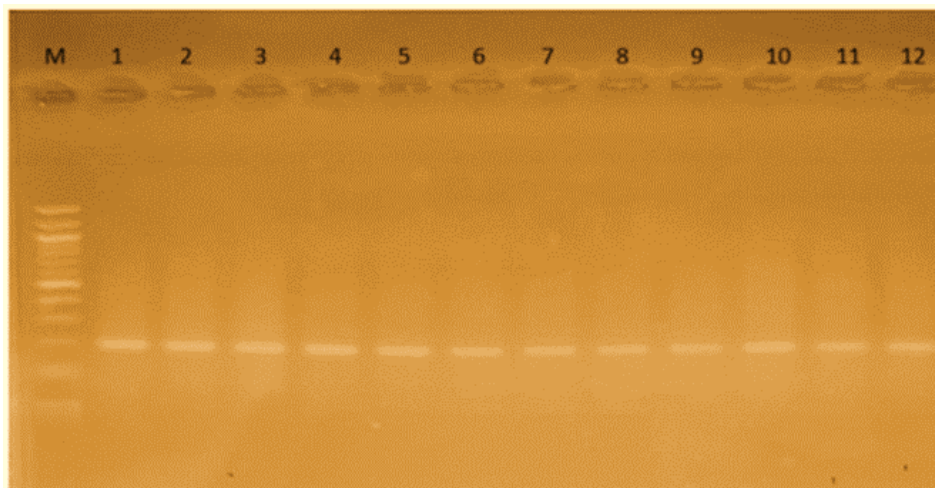


Figure 1. Agarose-gel electrophoresis of an amplified ADIPOR2 fragment in patients with T2DM (lanes 1–6) and healthy controls (lanes 7–12). Products were separated on a 1.5% agarose gel at 90 V for 90 minutes, stained with ethidium bromide, and visualized under ultraviolet illumination.

Table 3. Observed and expected ADIPOR2 rs16928751 genotype distributions, allele frequencies, and reported Hardy–Weinberg equilibrium results

Genotype rs16928751	Patients (N=48)				Control (N=32)			
	Observed		Expected		Observed		Expected	
	No.	%	No.	%	No.	%	No.	%
Genotype G/G	36	75	34.17	71.18	25	78.12	24.5	76.56
Genotype G/A	9	18.75	12.66	26.38	6	18.75	7	21.88
Genotype A/A	3	6.25	1.17	2.44	1	3.13	0.5	1.56
Alleles								
Allele G	81	84.38	Not Estimated		56	87.5	Not Estimated	
Allele A	15	15.63			8	12.5		
Exact HWE p-value	0.068				0.392			
Outcome	The population is in Hardy-Weinberg equilibrium							

Table 4. Comparison of ADIPOR2 rs16928751 allele frequencies between the T2DM and control groups

Alleles Frequency rs16928751	study groups		χ^2	P-value	OR	
	Patients (N=48)	Control (N=32)			value	95% CI
Allele G n(%)	81 (84.38)	56 (87.5)	0.104	0.747	0.77	0.31 - 1.94
Allele A n(%)	15 (15.63)	8 (12.5)		NS	1.30	0.52 - 3.26

The genotype distributions and the authors' reported results for the codominant, dominant, and recessive inheritance models appear in Tables 5, 6, and 7, respectively. Under the reported codominant analysis, the AA genotype had an OR of 2.07 (95% CI: 0.21–20.80), while the GA genotype had an OR of 1.00 (95% CI: 0.32–3.15). Neither estimate indicated a statistically significant association, and the confidence interval around the AA estimate was particularly wide because that genotype occurred in only four participants. Under the dominant model, the combined GA+AA genotypes had an OR of 1.19 (95% CI: 0.41–3.46; reported $p = 0.957$). Under the recessive model, the reported AA-genotype OR was 2.07 (95% CI: 0.21–20.78; reported $p = 0.917$). The nominal differences did not reach statistical significance. The reported chi-square statistic for the codominant comparison was $\chi^2 = 0.340$ ($p = 0.527$); the authors did not specify the degrees of freedom or the precise test underlying this statistic.

Table 5. ADIPOR2 rs16928751 genotype frequencies and reported statistics under the codominant inheritance model

Genotype rs16928751	study groups		χ^2	P-value	OR	
	Patients (N=48)	Control (N=32)			value	95% CI
Genotype G/G n(%)	36 (75)	25 (78.12)	0.340	0.527 NS	0.84	0.29 - 2.43
Genotype G/A n(%)	9 (18.75)	6 (18.75)			1.00	0.32 - 3.15
Genotype A/A n(%)	3 (6.25)	1 (3.13)			2.07	0.21 - 20.80

* (P<0.05), ** (P<0.01), NS: non-significant.

Table 6. ADIPOR2 rs16928751 genotype frequencies and reported statistics under the dominant inheritance model

Genotype rs16928751	study groups		χ^2	P-value	OR	
	Patients (N=48)	Control (N=32)			value	95% CI
Genotype G/G n(%)	36 (75)	25 (78.12)	0.003	0.957 NS	0.84	0.29 - 2.43
Genotype G/A + A/A n(%)	12 (25)	7 (21.88)			1.19	0.41 - 3.46

* (P<0.05), ** (P<0.01), NS: non-significant.

Table 7. ADIPOR2 rs16928751 genotype frequencies and reported statistics under the recessive inheritance model

Genotype rs16928751	study groups		χ^2	P-value	OR	
	Patients (N=48)	Control (N=32)			value	95% CI
Genotype G/G+G/A n(%)	45 (93.75)	31 (96.87)	0.011	0.917 NS	0.48	0.05 - 4.87
Genotype A/A n(%)	3 (6.25)	1 (3.13)			2.07	0.21 - 20.78

* (P<0.05), ** (P<0.01), NS: non-significant.

4. DISCUSSION

Type 2 diabetes mellitus is a common multifactorial metabolic disease whose progression is influenced by genetic susceptibility, environmental exposures, and metabolic risk factors. Long-term clinical management is needed to reduce the risk of microvascular and macrovascular complications [15]. Sex-related differences in its development have been reported, including differences in age at diagnosis, adipose distribution, and the metabolic conditions associated with insulin resistance [16, 17]. These published findings provide context for the present sample, although the absence of statistically significant between-group age and sex differences here prevents stronger inferences about sex-specific susceptibility. As expected, HbA1c and FBG were substantially higher in participants with T2DM than in controls, reflecting impaired glycemic regulation. Insulin resistance, increased hepatic glucose production, and dysfunction in insulin secretion may contribute to such differences. The observed biochemical pattern is compatible with evidence relating glucose and glycated hemoglobin to diabetes detection and monitoring [18–21]. These measures characterize the sampled groups but should not be interpreted as direct evidence that the ADIPOR2 variant alters glucose concentrations.

In both the T2DM and control groups, the reported exact tests found no statistically significant deviation from Hardy–Weinberg equilibrium. The GG genotype was slightly more frequent in controls (78.12%) than in patients (75.00%), whereas the GA genotype occurred at the same frequency in both groups (18.75%). The AA genotype was observed in 6.25% of patients and 3.13% of controls, but this difference was based on only three patients and one control and was not statistically significant. The A allele was also numerically more frequent in the T2DM group; however, its confidence interval included the null value, making it inappropriate to infer increased susceptibility from this sample alone. Previous work has reported heterogeneous results across populations. Shramko et al. examined adiponectin and adiponectin-receptor variants in a Russian population and described associations with metabolic characteristics, while also noting the lack of a consistent association in other genetic investigations [14]. Potapov et al. observed an association between the rs16928751 A allele and T2DM in a separate Russian sample (OR = 1.88, $p = 0.018$) [13]. Differences between studies may reflect population structure, sampling variability, measured phenotypes, statistical power, or analytical methods rather than a reproducible biological effect in every population. The present study adds population-specific descriptive data but does not replicate a statistically significant association.

Across the reported genetic models, the association estimates were inconclusive: the AA genotype produced a numerically elevated estimate, the GA estimate was approximately null, and the combined GA+AA and recessive comparisons were not statistically significant. The wide intervals surrounding the less frequent genotypes indicate imprecision and

suggest that the sample was too small to characterize modest effects reliably. In a Finnish cohort of individuals with impaired glucose tolerance, Siitonen et al. reported an association of the rs16928751 A allele with cardiovascular disease risk under a dominant model; this cardiovascular outcome should not be conflated with an association with T2DM onset [22]. Khodyrev et al. likewise examined ADIPOQ, ADIPOR1, and ADIPOR2 variation in relation to T2DM in a Russian population and reported findings that differed across variants and analyses [23]. Namvaran et al. documented the distribution of the ADIPOR2 +795 G/A variant in an Iranian sample, further illustrating that allele frequencies differ by population [11]. Taken together, the available studies support continued investigation of adiponectin signaling as a possible metabolic pathway of interest, but the present case–control data provide no statistically significant evidence that rs16928751 independently predicts T2DM in the sampled Iraqi participants.

Interpretation of the observed associations. The direction of an estimated odds ratio should not be interpreted independently of its confidence interval and the underlying genotype counts. An odds ratio above one suggests higher estimated odds in the exposed or variant group, whereas a confidence interval spanning one indicates that the data are also compatible with no association. This distinction is particularly important for the AA genotype, because only three patients and one control carried this genotype in the study. A small difference in the number of carriers would materially alter the estimate, and the resulting uncertainty cannot be resolved by the apparent magnitude of the point estimate alone. The same reasoning applies to the allelic analysis: the estimated A-allele odds ratio is greater than one, but the evidence is not sufficient to identify the allele as an independent determinant of diabetes risk. Moreover, the allele-based test considers two alleles per individual, and its uncertainty assessment must account appropriately for the genetic sampling structure.

Contextualization of the clinical and genetic observations. The study compares participants who already had T2DM with individuals assigned to the control group, and its case–control design cannot determine the temporal sequence between metabolic abnormalities and the development of disease. Likewise, the measured fasting glucose and HbA1c values provide cross-sectional clinical characteristics rather than estimates of future diabetes incidence. These distinctions matter when interpreting the word “risk,” which is used here to describe the odds of existing case status across genotype categories and not a directly measured prospective probability of developing T2DM. Although a genetic variant precedes the development of the disease in a biological sense, a retrospective association analysis remains susceptible to selection effects, population stratification, and unmeasured confounding. The result should therefore be interpreted as an initial population-level comparison rather than a mechanistic demonstration of causation.

The investigated receptor is part of a wider signaling system in which adiponectin, its circulating molecular forms, receptor abundance, and downstream pathways interact with metabolic state. A polymorphism examined in isolation may capture only a limited portion of this pathway’s variation. Moreover, the biological consequence of rs16928751 was not measured directly in this study: neither ADIPOR2 receptor expression nor protein activity was assessed. Consequently, the absence of a statistically significant association in the sample does not establish that adiponectin signaling has no role in metabolic disease, just as a numerically elevated odds ratio does not demonstrate that this specific variant is functional. Distinguishing the observed genotype distribution from proposed biological mechanisms is necessary for a balanced interpretation of the results.

Implications for subsequent research. Further work should distinguish exploratory genetic associations from clinically useful risk prediction. Replication in independent samples is essential, because allele frequencies, linkage disequilibrium patterns, and distributions of metabolic risk factors may differ between populations. Future protocols should prespecify genotype quality-control procedures, assess potential departures from Hardy–Weinberg equilibrium in controls, and provide transparent statistical-model specifications, including the exact reference genotype and adjustment covariates. Reporting absolute genotype counts alongside percentages and confidence intervals would facilitate comparisons across studies and allow independent assessment of whether an observed association is robust. These methodological improvements would make it possible to determine whether variation at ADIPOR2 rs16928751 adds meaningful information beyond established T2DM risk factors, rather than merely reflecting chance differences in small samples.

5. CONCLUSION

The rs16928751 (+795 G/A) polymorphism in the ADIPOR2 gene was examined in 48 Iraqi patients with T2DM and 32 healthy controls. Although the AA genotype and the A allele were numerically more frequent in the patient group, the genotype and allele comparisons did not reach statistical significance. In particular, the estimates derived from the less common AA genotype were imprecise, as reflected in their wide 95% confidence intervals. Therefore, these findings neither confirm a risk effect nor demonstrate a protective effect of the alternative allele. Larger case–control and prospective investigations, preferably incorporating ancestry information and relevant clinical covariates, are needed to assess whether rs16928751 contributes to T2DM susceptibility and whether any observed effect is consistent across Iraqi populations.

6. STUDY LIMITATIONS

This study has several limitations. First, the relatively small number of participants, particularly the four individuals carrying the AA genotype across both groups, limits statistical power and leads to wide confidence intervals for some associations. Second, participants were drawn from a limited geographical and clinical setting, which restricts the generalizability of the findings to other Iraqi communities. Third, only one ADIPOR2 variant was evaluated, and the study did not assess other variants or gene–environment interactions. Fourth, the available summary data do not permit evaluation of potential confounding by body mass index, medication use, family history, or other metabolic factors. Finally, although the reported genotype frequencies were compatible with Hardy–Weinberg equilibrium, this statistical result does not replace independent inspection of sequencing chromatograms or detailed genotyping quality control. Multicenter studies with larger samples and appropriately adjusted analyses are required to corroborate or refute the associations considered here.

Conflict of interests

The author declares no conflicts of interest related to this research.

Data availability

Aggregate genotype frequencies and summary clinical measurements supporting the findings are presented in the manuscript. Participant-level genetic and clinical records are not included in the article because they may contain sensitive personal information. Any request for access to de-identified data should be directed to the author and would require applicable institutional and ethical authorization.

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