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LIFESTYLE-MODIFIED GENETIC SUSCEPTIBILITY TO ACNE IN A SOUTHEAST ASIAN POPULATION

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ABSTRACT

Acne vulgaris is a polyetiological inflammatory disease that can be genetically predisposed and act as a response to environmental factors that can be modified. In this study, summary-level data of 4,976 participants, consisting of 2,741 acne cases and 2,235 controls, were used to assess the genetic susceptibility to acne and its modification by lifestyle-related factors in a Southeast Asian population. Replication results for 68 candidate single-nucleotide polymorphism (SNP) records were analysed along with 544 gene–environment interaction models for 8 lifestyle and environmental factors. Odds ratios, 95% confidence intervals, nominal p values, Benjamini–Hochberg false-discovery-rate adjustment and Bonferroni correction were used for evaluation of genetic associations. Ten candidate variants were nominally associated with acne, and rs738409 and rs1159268 were still significant after Bonferroni Correction. Of the interaction analyses, screen usage time yielded the most significant results with 58 nominally significant interactions, 43 interactions surviving the BH-FDR correction, and 7 interactions surviving Bonferroni correction. The corrected associations were weaker for housing type and alcohol intake, and the interactions were not strongly supported by correction for multiple testing with regard to the other factors assessed (fast food intake, milk intake, physical activity, income, and seafood intake). Given these results, lifestyle-related exposures, such as screen usage, could significantly impact, if not completely alter, the expression of genetic susceptibility to acne. The findings suggest a gene–environment model to explain acne heterogeneity and emphasize the need to incorporate behavioral factors in the genetic risk evaluation for acne in the Southeast Asian population.

Keywords: acne vulgaris; genetic susceptibility; gene–environment interaction; screen time; Southeast Asian population

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1. Introduction

Acne vulgaris is a chronic inflammatory disease of the pilosebaceous unit and is one of the most prevalent skin diseases in the world. It presents as comedonal lesions, inflammatory papules and pustules, nodules and scarring and has significant psychosocial and quality of life implications. However, globally, acne is a significant disease burden in adolescents and young people, though a variable prevalence and severity in geographic regions and population groups exists (Chen et al., 2022). The epidemiology of acne was also found to be highly variable by age, sex, ethnicity and environmental exposures, indicating that the development of acne is likely influenced by a number of biological and contextual factors (Heng & Chew, 2020).

Acne pathogenesis is complex and includes abnormal follicular keratinization, hyperactivity of the sebaceous glands, dysbiosis of microbes, androgenic signaling and inflammatory responses. The production of sebum is especially significant since it creates a lipid-rich micro-environment which can facilitate follicular obstruction and inflammatory activity (Del Rosso & Kircik, 2024). Differences in Cutibacterium acnes phylotypes may also play a role in the development and severity of the lesions (Dreno et al., 2024). Recent data further suggests association between lipid metabolism and sebaceous gland function with systemic metabolic and dietary factors, which we believe has provided a mechanistic link between biological susceptibility and lifestyle exposures (Jakubowicz-Zalewska et al., 2026).

Genetic susceptibility is a significant part of the pathogenesis of acne. Inherited variation has been shown to play a role in the variation in acne risk and severity by family aggregation and population-based genetic studies. Various genetic polymorphisms were discovered in a systematic review and meta-analysis study to support the polygenetic nature of acne (Heng et al., 2021). GWAS have greatly increased the number of associated loci; Mitchell et al. (2022) found 29 more acne susceptibility loci. Additional GWMA has identified additional acne risk loci, further confirming the genetic architecture's role in acne expression (Teder-Laving et al., 2024). More modern reviews therefore characterize acne as a genetically complex disease with a strong inflammatory, follicular, sebaceous and hormonal component (Van Steensel, 2025).

While genetics play a role, acne is not likely to be solely caused by genetic factors. Phenotypic expression of inherited susceptibility could be modified by lifestyle and environmental exposures. Diet has been of special interest, and evidence suggests that a high-glycemic dietary pattern, dairy consumption and other dietary factors may be associated with the development and severity of acne (Meixiong et al., 2022). In addition, dietary, family history, BMI and hormonal issues have been linked to severity of acne in adult patients (Anaba & Oaku, 2021). Lifestyle-related factors that can be modified like sleep habits, psychological stress, physical activity, screen time, and other daily routines could potentially affect acne through neuroendocrine, inflammatory, metabolic, and behavioral mechanisms (Hu et al., 2022).

The importance of these factors has been further reinforced by recent studies in an Asian population. The epidemiological risk factors were associated with the presentation, severity and scarring of acne in the Singapore Chinese population, highlighting the clinical heterogeneity and the importance of both modifiable and non-modifiable factors (Heng et al., 2022). The same was observed with Malaysian Chinese patients where lifestyle and inherited factors were linked with acne occurrence and severity (Say et al., 2021). The Singapore/Malaysia Cross-Sectional Genetics Epidemiology Study gave in-depth information on acne phenotypes, acne severity, scars, and risk factors among young adults of Chinese origin (Huang et al., 2025).

The relationship between genetic susceptibility and lifestyle exposure is therefore becoming a greater factor. Environmental influences can affect the intensity or the direction of genetic influences, rather than acting alone. The concept was directly supported by recent data of the Singapore–Malaysia study, which found that significant gene–environment interactions with screen time are observed, with the replication of acne susceptibility loci (Liu et al., 2026b). The results indicate that certain lifestyle factors act as risk or protective factors for acne and provide a more holistic understanding of the variation in risk associated with acne between individuals.

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Thus, the aim of the present study was to examine summary-level genetic data and gene–environment interaction data for the Southeast Asian population in the SMCGES cohort for lifestyle-modifiable genetic susceptibility to acne. The study examines whether replication of previously reported variants associated with acne, and a test of whether genetic associations with acne are modified by lifestyle and environmental factors like physical activity, dietary exposures, alcohol consumption, income, and housing characteristics, such as screen time.

2. Materials and Methods

2.1 Study Design and Data Source

Secondary summary-level genetic and gene–environment interaction data from the Singapore–Malaysia Cross-sectional Genetics Epidemiology Study (SMCGES) (Liu et al., 2026a) were used in this study. For the analytical data set, 2,741 cases of acne and 2,235 controls were included. All the deposited files included the summary statistics for previously reported single-nucleotide polymorphisms (SNPs) associated with acne, SNP summary statistics for gene–environment interactions, and SNP proxy-mapping information. No individual level participant information was provided in the deposited data set, limiting the analyses to reported summary data.

2.2 Genetic Variant Replication Analysis

This replication set consisted of 68 candidate-SNP records with 67 different rsIDs. Both directly genotyped SNPs and proxy SNPs found in the provided proxy-mapping file were included as variants. Replication summary statistics for all the candidate variants were downloaded and the effect allele, effect allele frequency, odds ratio (OR), 95% CI and P value for association with acne extracted.
Those with p values < 0.05 were regarded as nominally related to acne. The odds ratios were reported and values greater than 1 represented higher odds and values less than 1 represented lower odds of acne for the effect allele.

2.3 Gene–Environment Interaction Analysis

There were 544 SNP × environment models in the gene–environment interaction dataset. Eight environmental or lifestyle related factors were evaluated for each candidate SNP: screen usage time, physical activity frequency, income, housing type, milk intake, seafood intake, fast food intake, alcohol intake.
Summarized statistics (summary file) were downloaded for each interaction model and the interaction odds ratio, 95% confidence interval and the P value were extracted. The number of interaction signals were summarized individually for each environmental factor. Interactions that were not statistically significant were not mentioned after correction for multiple testing.

2.4 Multiple-Testing Correction

Multiple-testing correction was done separately for genetic replication and gene–environment interaction analyses. To account for the multiple comparisons made in the replication analysis with 68 candidate-SNP records, the significance level was Bonferroni adjusted to $P < 0.000735$. Additionally, false discovery rate correction done by Benjamini–Hochberg was applied and q values below 0.05 were considered statistically significant.
The Bonferroni correction was applied to the P-value for the 544 gene–environment interaction tests to obtain a threshold of $0.05/544 = P<0.0000919$. To determine significant interaction signals at an FDR threshold of 0.05, Benjamini–Hochberg adjusted q values were calculated.

2.5 Statistical Presentation

Odds ratios, 95% confidence intervals, P values and FDR adjusted q values were used to summarize genetic replication results. Significance plot for replication signals based on the chromosome and forest plot of nominally significant SNPs were generated. Findings from the gene–environment analysis were reported by environmental factor by nominal significance, FDR significance and

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Bonferroni significance. Furthermore, Bonferroni-significant SNP × screen-usage interactions were presented graphically with a forest plot of the ORs with 95 percent CIs.

2.6 Ethical Considerations

In this study, summary-level data from publicly accessible sources was used, which were reported and de-identified, and no direct recruitment, intervention, or access to personally identifiable information was conducted. This study was thus confined to the information included in the research files that were deposited. Because no individual level identifying data were available or used in the present analysis, confidentiality of participants was preserved.

3. Results

3.1 Analytical Dataset and Genetic Replication Profile

The summary-statistics dataset that was deposited was composed of 4,976 participants including 2,741 with acne and 2,235 without acne. There were 68 candidate-SNP records in the replication file, including 67 different rsIDs with 33 of these directly genotyped and 35 being proxy-SNPs. The gene–environment component involved 544 interaction models with eight environmental or lifestyle factors: screen usage time, frequency of physical activity, income, housing type, intake of milk, intake of seafood, intake of fast food and alcohol intake (Table 1).

Of the 68 candidate-SNPs, 10 had nominal evidence of association with acne ($P<0.05$). Three of these were significant following Benjamini–Hochberg false-discovery-rate correction and two were significant after the more conservative Bonferroni correction threshold of less than 0.000735. Figure 1 shows the distribution of association significance over chromosomes.

Table 1. Analytical coverage of the acne genetic and gene–environment dataset

Characteristic	Result
Acne cases	2,741
Controls	2,235
Total participants represented	4,976
Candidate-SNP records	68
Distinct rsIDs	67
Directly genotyped records	33
Proxy-SNP records	35
Environmental/lifestyle factors	8
SNP × environment models	544
Replication Bonferroni threshold	0.000735
G×E Bonferroni threshold	0.0000919

Note: The deposited replication summary contains 68 candidate-SNP records but 67 distinct rsIDs because rs9616391 occurs in more than one source entry.

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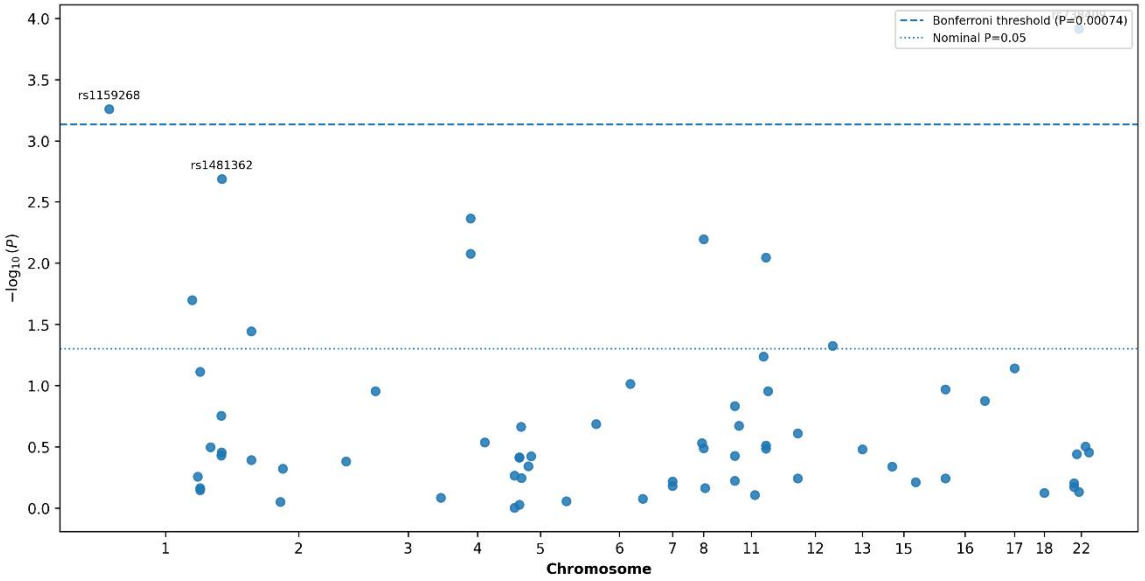

Figure 1. Replication significance of previously reported acne-associated SNPs
The chromosome-based plot displays −log10(P) values for the candidate variants. The dashed horizontal line represents the Bonferroni-corrected threshold, while the dotted line represents nominal P = 0.05. rs738409 and rs1159268 exceeded the Bonferroni threshold.

3.2 Replication of Acne-Associated Genetic Variants

The most significant replication signal was on chromosome 22 at rs738409. The G effect allele was associated with lower odds of acne (OR = 0.849, 95% CI 0.781–0.923, P = 0.000122). This association also persisted following BH-FDR correction (q = 0.0083) as well as Bonferroni correction. The second Bonferroni-significant variant was rs1159268 on chromosome 1, for which the G allele was likewise associated with reduced acne odds (OR = 0.870, 95% CI 0.803–0.941, P = 0.000553; q = 0.0188).

A third directly genotyped variant (rs1481362) was found to be associated with a higher risk for acne (OR = 1.133, 95% CI 1.046–1.226, P = 0.00206); while not significant at the Bonferroni threshold, it was significant at BH-FDR correction (q = 0.0466). Seven additional loci showed nominal associations, including rs2047583, rs712253, rs4487353, rs11602680, rs3917698, rs2901000, and rs7488044 (Table 2). The effect estimates and corresponding 95% confidence intervals for the ten nominally significant replications signals are shown in Figure 2.

Table 2. Candidate SNPs showing nominal replication of association with acne

SNP	Chr	Effect allele	EAF	OR (95% CI)	P value	BH-FDR
rs738409	22	G	0.376	0.849 (0.781–0.923)	0.000122	0.0083
rs1159268	1	G	0.487	0.870 (0.803–0.941)	0.000553	0.0188
rs1481362	1	A	0.500	1.133 (1.046–1.226)	0.002055	0.0466
rs2047583	4	C	0.458	1.125 (1.038–1.221)	0.004309	0.0733
rs712253	8	T	0.018	1.517 (1.125–2.048)	0.006375	0.0867
rs4487353	4	G	0.477	1.114 (1.027–1.207)	0.008363	0.0872

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rs11602680	11	G	0.487	0.897 (0.828–0.973)	0.008976	0.0872
rs3917698	1	A	0.365	1.103 (1.015–1.198)	0.020090	0.1708
rs2901000	2	A	0.295	1.100 (1.006–1.202)	0.035960	0.2717
rs7488044	12	A	0.005	0.527 (0.280–0.992)	0.047250	0.3213

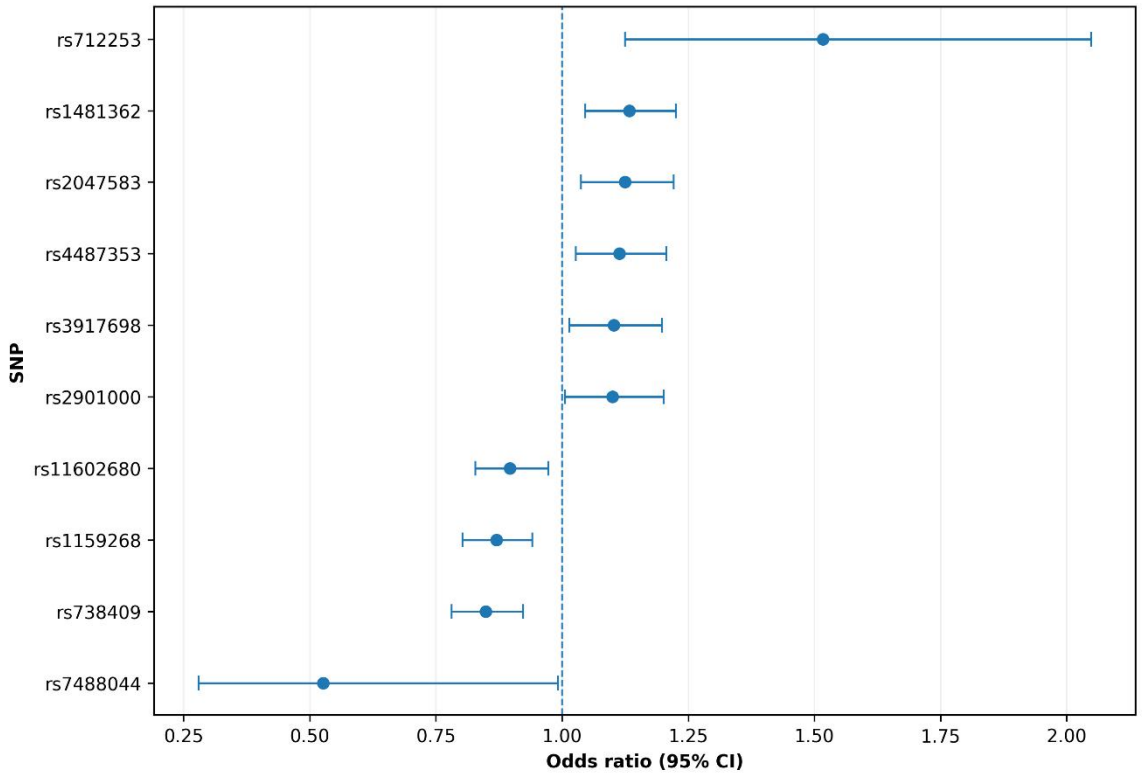

Figure 2. Forest plot of the ten strongest replicated acne-associated SNPs

Points represent odds ratios for the effect allele and horizontal bars represent 95% confidence intervals. The vertical reference line denotes OR = 1.0.

3.3 Gene–Lifestyle and Gene–Environment Interaction Patterns

There were 544 SNP × environment tests available, which equals 68 SNP records tested individually against 8 environmental/lifestyle factors. However, overall, 76 interactions showed nominal $P < 0.05$; 46 of these interactions remained significant after BH-FDR correction, and seven survived the Bonferroni threshold of $P < 9.19 \times 10^{-5}$.

The interaction resulted was highly focused in screen usage time. The 68 SNP×screen-time models yielded 58 nominally significant models, 43 of which survived BH-FDR correction, and seven Bonferroni-significant models, all of which included screen usage time. There were 5 nominal interactions and 2 FDR significant interactions for housing type, and 3 nominal and 1 FDR significant interactions for alcohol intake. Fast food consumption, milk consumption, physical activity and income level gave several nominal signals; however, none of this survived FDR correction. None of the seafood-intake interaction effects were nominally significant (Table 3; Figure 3).

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Table 3. Distribution of SNP × environmental/lifestyle interaction signals

Environmental/lifestyle factor	Tests	Nominal P<0.05	BH-FDR<0.05	Bonferroni significant
Screen usage time	68	58	43	7
Housing type	68	5	2	0
Alcohol intake	68	3	1	0
Fast-food intake	68	3	0	0
Milk intake	68	4	0	0
Income	68	1	0	0
Physical activity frequency	68	2	0	0
Seafood intake	68	0	0	0

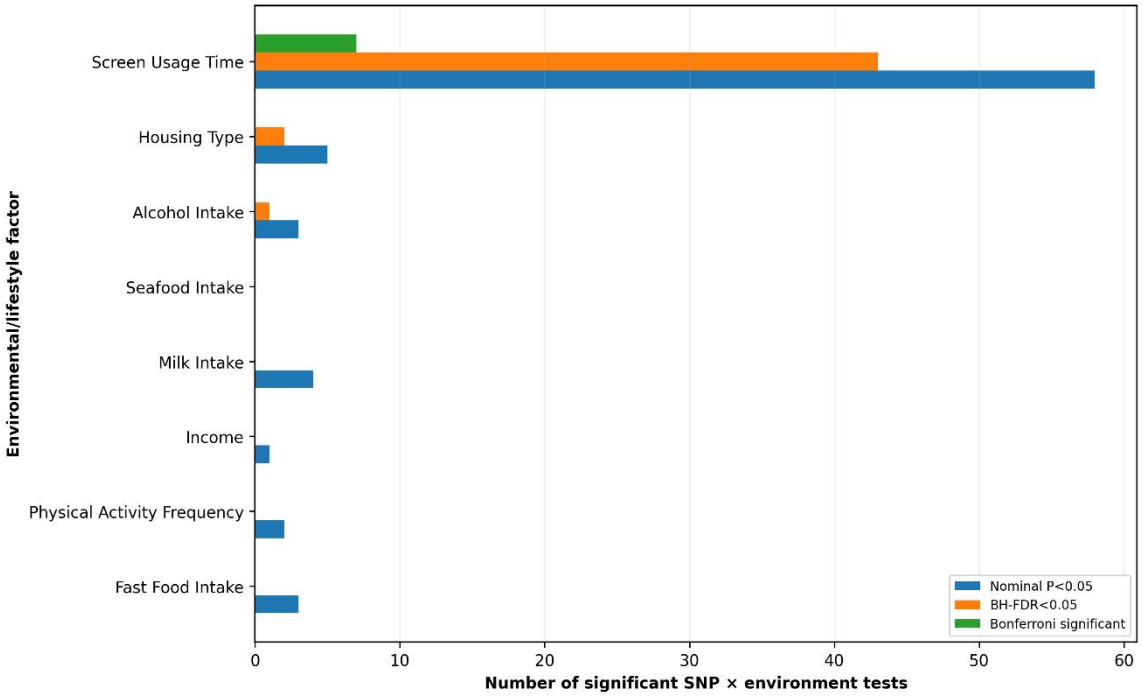

Figure 3. Number of significant SNP × environment associations according to environmental or lifestyle factor

The number of interactions that meet nominal, BH-FDR and Bonferroni significance criteria are indicated by bars. The top interaction signal was screen usage time.

3.4 Screen Usage as the Predominant Modifier of Genetic Susceptibility

There were 7 SNP×screen-usage interactions that remained after correction for all 544 interaction tests (Table 4). The most statistically significant interaction occurred for rs4723979, with an interaction OR of 1.119 (95% CI 1.066–1.175, P = 4.96×10^{-6}). Similarly strong interaction effects were observed for rs11602680 (OR = 1.117, 95% CI 1.064–1.172, P = 6.62×10^{-6}), rs6949092 (OR = 1.114, 95% CI 1.061–1.170, P = 1.27×10^{-5}), and rs13013349 (OR = 1.110, 95% CI 1.057–1.165, P = 2.42×10^{-5}).

The largest interaction effect among these corrected associations was observed for rs9650656 (OR = 1.132, 95% CI 1.066–1.202), closely followed by rs6698475 (OR = 1.131, 95% CI 1.066–1.199). The

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rs12321 interaction was also significant after Bonferroni correction (OR = 1.105, 95% CI 1.051–1.162, P = 8.97×10^{-5}). The seven loci have consistent estimates of their positive interactions, as shown in Figure 4.

The source dataset contains summary statistics that have been encoded rather than the actual variable of screen time, so the interaction ORs should be interpreted as effects per unit change in the environmental variable as coded in the source model and should not be specifically interpreted as effects per hour of screen time.

Table 4. Bonferroni-significant SNP × screen-usage interactions

SNP	Interaction OR	95% CI	P value	BH-FDR
rs4723979	1.119	1.066–1.175	0.00000496	0.0018
rs11602680	1.117	1.064–1.172	0.00000662	0.0018
rs6949092	1.114	1.061–1.170	0.0000127	0.0023
rs13013349	1.110	1.057–1.165	0.0000242	0.0033
rs6698475	1.131	1.066–1.199	0.0000415	0.0045
rs9650656	1.132	1.066–1.202	0.0000500	0.0045
rs12321	1.105	1.051–1.162	0.0000897	0.0069

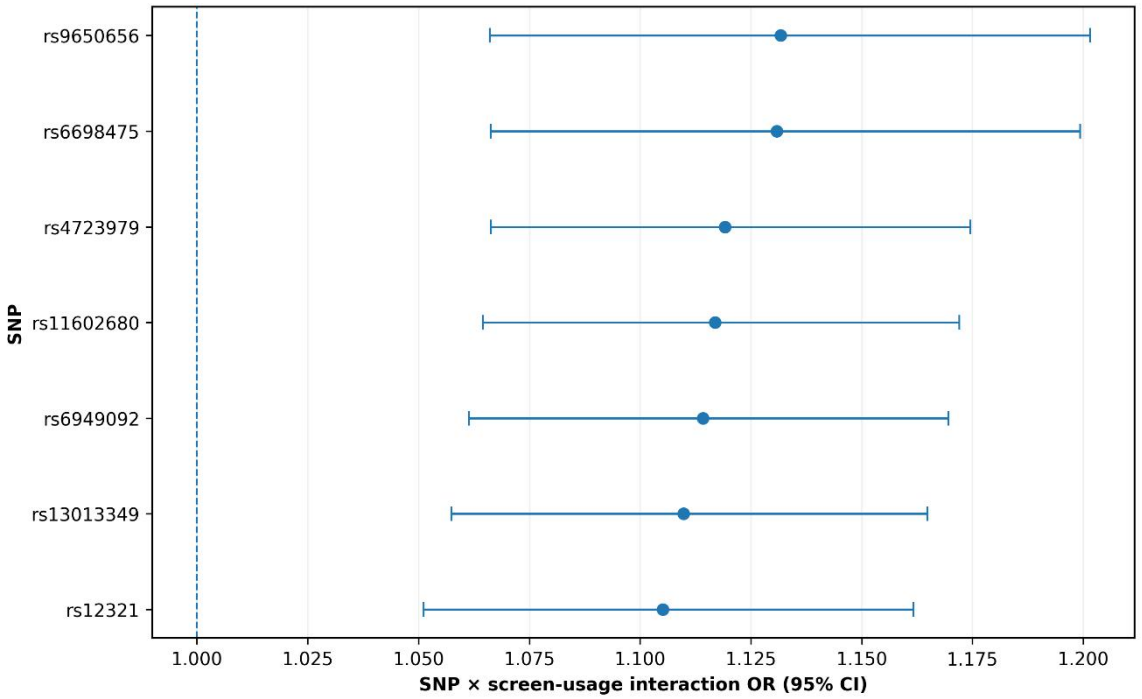

Figure 4. Forest plot of Bonferroni-significant SNP × screen-usage interactions

The seven associations that remained after correction are displayed using odds ratios and 95% confidence intervals for each association in the 544 G×E tests. None of the estimates were below an

OR of 1.0.

3.5 Secondary Lifestyle Interaction Signals

Outside screen usage, the strongest corrected signals involved socioeconomic/environmental and alcohol-related factors. rs16874036 × housing type showed an interaction OR of 1.275 (95% CI 1.097–1.480, P = 0.00149; q = 0.0219), while rs10941664 × housing type showed an OR of 1.261 (95% CI 1.087–1.464, P = 0.00227; q = 0.0317). The interaction between rs1342583 and alcohol intake was also FDR-significant (OR = 1.253, 95% CI 1.076–1.459, P = 0.00366; q = 0.0443).

There were several other lifestyle interactions that were nominally significant but not significant after multiple testing corrections. These included rs2727365 × fast-food intake (OR = 1.775, P = 0.00497), rs629725 × milk intake (OR = 0.840, P = 0.00700), and rs8042919 × physical-activity frequency (OR = 1.205, P = 0.0482). These observations are therefore to be understood as exploratory and not as statistically solid results.

4. Discussion

The present results confirm that the susceptibility to acne in the present population in the Southeast Asian region has genetic factors and measurable influences of lifestyle-related exposures. The strongest associations remained after multiple testing were obtained by rs738409 and rs1159268 after multiple testing, and rs1481362 after false-discovery-rate (FDR) adjustment, among the replicated variants. These observations are in line with the increasing evidence of acne's polygenetic nature, in which multiple genes play a role instead of a single gene (Mitchell et al., 2022; Van Steensel, 2025). The further nominal associations found at multiple loci support this notion that multiple genetic pathways are involved and contribute to acne expression.

The most salient result was the clustering of interactions between genes and environment around amount of screen time. Seven SNP×screen-time interactions passed the Bonferroni corrected threshold, while no other lifestyle factor was associated with as strong a threshold. This is very similar to the evidence found in Singapore–Malaysia that screen-related exposures are able to alter the effect of acne-associated genetic variants (Liu et al., 2026b). Lifestyle factors are increasingly acknowledged as a potential link to acne, especially through the neuroendocrine and inflammatory pathways involved with sedentary time, sleep pattern, stress, and daily habits (He et al., 2026).

Another indirect way of screen exposure could be through changes in sleep. Acne severity has been associated with poor sleep quality, and poor sleep quality may be a mediator for the association between psychological stress and acne in young adults (Chen et al., 2026). A wider look also found that sleep disturbance is a potentially significant but poorly recognized factor that plays a role in the persistence and pathogenesis of acne (Samaniego et al., 2025). The associations between these observations and strong interaction signals of screen time are biologically plausible, but can't be examined directly in the summary-level dataset.

Comparatively weaker interactions were found for diet-related. Several nominal associations were found with milk consumption and fast food consumption, but after correction of multiple tests, none of these associations were significant. This doesn't rule out a food influence in acne. Based on the previous study, dietary patterns have been linked to acne by glycemic load, insulin-like growth factor signaling, sebaceous activity and inflammatory mechanisms (Baldwin & Tan, 2021). Systematic reviews and meta-analysis published recently have also associated higher adherence to a Mediterranean diet pattern with reduced acne burden, with some consistency across populations (Taha et al., 2025). The case-control study also indicates a possible association between the severity of acne and the adherence to the Mediterranean diet (Aryanian et al., 2025). This may, however, be due to the fact that the diet interactions were not corrected in the present analysis and therefore the modifying effects of the diet interactions were probably smaller compared to the effects of screen-related behavior.

The biological interpretation of these results also fits with the current understanding of the pathophysiology of acne. Acne is a condition that occurs due to a series of interconnected

abnormalities related to hyperkeratinization, hyperseborrhea, inflammation, microbial growth and hormonal imbalance (Vasam et al., 2023). Recent studies on Sebaceous disorders also show that the dysregulation of lipid and sphingolipid metabolism is linked to inflammatory signaling and the function of the sebaceous gland (Miner et al., 2025). These mechanisms may be pathways by which inherited susceptibility may play a role in conjunction with external exposures.

Hormonal pathways are especially important when it comes to the susceptibility of acne among men and women. In the case of adult female acne, several factors are linked to an endocrine and clinical profile that differs from that of the adolescent acne (Branisteanu et al., 2022). In susceptible women, androgen excess can also have a role in the development of persistent acne by altering sebaceous activity and/or follicular function (Carmina et al., 2022). In the absence of sex-specific interaction analysis, these mechanisms may account for inter-individual variation in genetic effects in the current summary statistics but would not be directly detected in this analysis.

The interactions between housing type and alcohol consumption were weaker but FDR significant, indicating that there may be a broader socioeconomic/behavioural context that influences the susceptibility to acne. The use of medication, age, and behaviours of the day may have an impact on the acne risk over time, as suggested in the lifestyle oriented prospective research (Snyder et al., 2025). Clinically important factors include psychosocial and environmental factors, as acne can significantly impact well-being, especially among young adults and students (Sachdeva et al., 2021).

The results should be viewed in the context of the summary level data, however. Direct estimation of combined lifestyle-risk profiles, individual level confounder adjustment and mediation analyses were not feasible. Moreover, statistics on interactions are insufficient to draw conclusions about causal direction. There remains a significant heterogeneity in the nature of the triggers and in the response to treatment in modern-day review articles (Dagnelie et al., 2022; Dessinioti & Dreno, 2026). The overall results suggest that genetic susceptibility to acne does not appear to be fixed in expression but can be influenced by life exposures: the current results indicated that screen usage was the biggest factor modifying genetic susceptibility to acne in this Southeast Asian population.

5. Conclusion

This research has shown that acne susceptibility in a Southeast Asian population is influenced by both genetic and lifestyle related environmental modification. Several previously reported acne associated variants replicated, and two (rs738409 and rs1159268) were still significant after applying a rigorous multiple-testing correction. The gene–environment analysis also demonstrated that the time spent on screens emerged as the most prominent lifestyle-related modifier of genetic susceptibility, with the majority of significant interaction signals, and all interaction signals surviving the Bonferroni correction. Housing type and alcohol consumption had weaker false-discovery-rate adjusted correlations, whereas dietary factors, physical activity and income had only weak nominal associations.

The results are consistent with the notion that hereditary predisposition for acne does not manifest itself in isolation of environmental variables. Rather, lifestyle exposures can modify the effects of the genes and can also be a source of inter-individual variation in the incidence of acne. The positive screen-time interaction signals are significant for young Southeast Asian populations with whom digital-device use is becoming more a part of everyday life. But, due to the nature of the data analyzed at the summary level, individual-level confounding, mediating and causal pathways could not be directly explored. Combined genetic and lifestyle risk profiles should be explored in future research with individual-level longitudinal data, as should the possibility of reducing the risk of acne by altering screen-related and other lifestyle risk factors in individuals with genetic susceptibility.

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