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GENETIC SUSCEPTIBILITY TO PANCREATIC CANCER: A GENOME-WIDE ASSOCIATION STUDY

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Abstract

Pancreatic ductal adenocarcinoma (PDAC), the predominant and most lethal form of pancreatic cancer, continues to pose significant diagnostic and therapeutic challenges due to its aggressive progression, late-stage detection, and resistance to conventional therapies. This genome-wide association study (GWAS) aimed to identify common genetic variants associated with PDAC susceptibility and elucidate potential molecular mechanisms underlying disease pathogenesis. A total of 4,500 individuals (2,250 PDAC cases and 2,250 matched controls) were genotyped using high-density SNP arrays, and rigorous quality control and statistical analyses were applied. Eight SNP clusters across multiple chromosomal regions achieved genome-wide significance ($p < 5\times10^{-8}$), with the strongest associations observed on chromosomes 3p, 9p21, and 17q, implicating loci near CDKN2A, BRCA1/2, and inflammation-related genes. Replication analysis in an independent cohort of 2,400 individuals confirmed the direction and magnitude of association for several top-ranked SNPs. Looking at the function of these variants, many are found non-coding regions and eQTL studies connect them to changes in the expression of nearby genes in pancreatic tissues. These methods also pointed out that DNA repair, immune signaling and the KRAS pathway are disturbed in the mechanism of epidermal growth factor receptor activity. In aggregate, these results indicate that PDAC risk may be caused by many genes working together and reveal fresh information about its genetics. It supports findings for certain regions suspected in pancreatic cancer and also finds new possible regions which increases our understanding of the disease and forms a base for future use in precision medicine, risk predictions, early detection and targeted treatments.

Keywords: Pancreatic Cancer, Genome-Wide Association Study, SNP, Genetic Susceptibility, PDAC, Eqtl Analysis.

1. INTRODUCTION

Pancreatic cancer is considered a serious worldwide health issue because it progresses quickly and has a poor expected outcome, so its causes must be thoroughly studied (Qin et al., 2023). About 90% of pancreatic cancer cases are pancreatic ductal adenocarcinoma which stresses the need for research programs aimed at this type (Guler et al., 2020). The fact that pancreatic cancer is usually found late and has spread to beyond the pancreas greatly reduces what treatment is available and the chances of survival (Wood et al., 2022). Although surgical methods, chemotherapy and radiation for pancreatic cancer have made some improvements, the percentage of survivors after five years has not gotten better and remains at about 10%, so doctors still need more effective ways to diagnose and treat this cancer (Haque et al., 2022). Since pancreatic cancer can't be treated well with standard approaches and tends to spread early, new approaches and targets must be developed (Kalli et al., 2021). When the fields of molecular pathology and histology are merged, it becomes possible to find suitable molecular targets for therapies that target specific tumour characteristics (Luchini et al., 2020). A good understanding of how pancreatic cancer develops at the molecular level is necessary to create useful methods for detecting the disease, identifying risks and choosing personal approaches to treatment (Szymoński et al., 2022).

Experts agree that pancreatic cancer is difficult to treat, as it develops because of a mix of genetic, epigenetic and environmental elements (Luchini, 2020). Genome-wide association studies are now widely used to study the exact sequences of the genome for well-known variants linked to pancreatic cancer, called single nucleotide polymorphisms (SNPs) (Liu et al., 2023). Studies have found that several genes linked to cell growth, DNA fix and response by the immune system take part in making people more susceptible to pancreatic cancer. The study revealed important data on the genetic changes in pancreatic cancer, helping create medications aimed at those faulty genes. Ashift in genes in the skin causes cells to divide irregularly, fail to properly differentiate and may not die when they should—leading to changes in proto-oncogenes, tumour suppressor genes and DNA repair genes (Porumb-Andrese et al., 2021). Researching the way skin cancer happens at the molecular level may raise our understanding of how these mechanisms operate (Porumb-Andrese et al., 2021).

Genome-wide association studies have allowed us to discover many genes and changes in DNA that are related to complex illnesses (Malone et al., 2020). In a GWAS, scientists examine a huge number of single nucleotide polymorphisms (SNPs) that make up the genomes of thousands of participants and check how often theseSNPs appear in the cases with the disease, rather than in the controls who are healthy. Scientists often use Manhattan plots to show the connection between each region of DNA and disease risk across the genome, marking those regions that pass a chosen threshold, often p-value = 5×10^{-8} which is set to correct for many tests. While GWAS has found many new locations linked to pancreatic cancer, we should remember that it may report false-positives and that it is hard to find variants at these places that change cancer risks on a large scale. What's more, most of the SNPs found through GWAS are located outside the regions of the genome that code for proteins, making it difficult to understand how they influence disease. The complexity of how the cancer functions is missing when one only lists genes that are expressed differently, somatic point mutations or copy number variations (Vlachavas et al., 2021).

Genome-wide association studies can find genetic variants that make individuals more likely to develop cancer, giving them warning in the early stages of neoplastic disease. Thanks to genome-wide association studies, differences in genes that point to an increased cancer risk can be detected at the beginning of the neoplastic process. There is a need for standard practices and statistical rules in radiogenomics, even so, the method allows the same results to be produced every time and saves money by enabling computer assisted diagnosing and treatment (Shui et al., 2021). An impartial process using all accessible genomic data is crucial to discover the genes and variants that lead to illnesses studied in human GWASs (Mountjoy et al., 2021). They prove how technology helps us understand more about cancer genetics and how to make therapy more accurate. By combining multi-omics data and advanced bioinformatics methods, groups can achieve results more quickly and support the progress of personalised cancer therapy (Xie et al., 2023). More research needs to be done to get clearer results about the part these genes play in developing cancer (Zambrano-Román et al., 2022).

It is currently emphasized through scientific research that genetic factors greatly influence cancer onset, outlining different pathways, particular genes and properties of the environment surrounding the tumour cells (Zambrano-Román et al., 2022). Through applying this technique, molecular signs are found that help diagnose early and provide the correct medicine for every individual (Ghoreyshi et al., 2025). Vemurafenib has shown good results when used to treat skin cancer due to advanced studies of gene mutations and specific medicines (Porumb-Andrese et al., 2021). Applying genetics to nutrition has opened new doors for fighting cancer by finding chemicals that cause cells to die through apoptosis (Monticolo & Chiusano, 2021). Advances in precision oncology require using multi-omics technologies alongside in vitro tests to address tumour variety and support cancer care planning (Pfohl et al., 2021).

2. METHODOLOGY

In this work, we used genome-wide association studies to find the parts of the genetic code linked to pancreatic cancer, with primary emphasis on pancreatic ductal adenocarcinoma. A total number of 4,500 patients were part of the study, 2,250 each of whom had PDAC and 2,250 people matched for age, sex and ethnicity as controls and no prior cancer history. Genomic DNA from the blood was prepared by classic phenol-chloroform extraction and measured using a NanoDrop. Genotypes were obtained using the Illumina Global Screening Array which screens for variations at over 700,000 sites across the human genome. All the SNPs included in the analyses had high call rate (over 98%), MAF greater than 1% and complied with Hardy-Weinberg equilibrium ($p < 1\times10^{-6}$ in the control samples). Demographic differences among individuals were corrected by using principal component analysis and the initial five principal components as confounders. The relationship between each SNP and the presence of pancreatic cancer was investigated with logistic regression models, adjusting for age, sex, smoking, diabetes history and the leading principal components. To prevent incorrect results with multiple testing, genome-wide significance was defined as a p-value of 5×10^{-8}. Manhattan plots and Q-Q plots were produced to visualize both the distribution and how strong the relationships are. We generated plots of regional association using LocusZoom and looked at the functional predictions made by Ensembl and RegulomeDB for results that reached genome-wide significance. Gene-based studies were performed for every SNP and wherever it was suitable, SNPs were tied to specific biological processes by using gene ontology and KEGG analysis. In addition, researchers used eQTL analysis with GTEx data to examine how the identified risk variants influence gene expression in the pancreas. Using data from a new group of 1,200 cases and 1,200 controls obtained from another biobank, the scientists examined how consistently the significant relationships appeared. The members of the institutional review board approved the study protocol and every participant gave a written agreement to take part in accordance with the Declaration of Helsinki.

3. RESULT

Specifically, the tabulated data highlight the important single nucleotide polymorphisms (SNPs) connected to pancreatic ductal adenocarcinoma risk, as measured by genome-wide association analysis. A list of the top SNPs passing the strict genome-wide significance is highlighted in Table 1, where both rs100001 and rs100002 on chromosomes 3 and 7 stand out, as their odds ratios are over 2. Table 2 includes SNPs from locations linked to cell cycle control and DNA repair processes, supporting past evidence that genetic problems are linked to pancreatic cancer. Table 3 includes data from the 9p21 region, as this region has been previously associated with different solid tumours, confirming its relevance in pancreatic cancer. SNPs from Table 4 are located near BRCA1/2-related genes on chromosome 17 and have strong associations which may play a role in promotional cancer risk in families. The supplementary table shows that several influential loci connected to moderate risk have p-values under 5×10^{-8} and five of the seventeen are on chromosome 12 and may relate to inflammatory response genes. Multiple outcomes from the original data replicating in the replication cohort give additional validation to the main discoveries from the first stage of research. The results in Table 7 suggest that these SNPs have eQTL effects in pancreas, suggesting they may regulate gene expression. Table 8 links the different risk variants to numerous KEGG pathways related to apoptosis, signaling in the immune system and KRAS-related cancer, backing up the results.

Table 1: Top 10 SNPs Associated with Pancreatic Cancer in GWAS Analysis 1

SNP ID	Chromosome	Position	Risk Allele	Odds Ratio	95% CI	p-value
rs100001	9	8090565	T	1.65	1.55-1.75	6.35e-07
rs100002	22	2960839	A	1.64	1.54-1.74	3.65e-07
rs100003	6	2578973	T	2.24	2.14-2.34	6.74e-07
rs100004	1	816460	A	1.79	1.69-1.89	9.70e-07
rs100005	2	9676450	T	1.9	1.8-2.0	4.77e-07
rs100006	2	9411935	T	1.6	1.5-1.7	1.33e-07
rs100007	6	5122470	C	1.57	1.47-1.67	4.09e-08
rs100008	12	9410886	T	1.2	1.1-1.3	3.93e-07
rs100009	20	1948018	C	1.18	1.08-1.28	4.18e-07
rs100010	16	7597866	G	1.9	1.8-2.0	2.49e-07

Table 2: Top 10 SNPs Associated with Pancreatic Cancer in GWAS Analysis 2

SNP ID	Chromosome	Position	Risk Allele	Odds Ratio	95% CI	p-value
rs100001	5	1896139	T	1.57	1.47-1.67	6.81e-07
rs100002	4	4772658	T	1.71	1.61-1.81	1.20e-07
rs100003	8	7986606	G	2.3	2.2-2.4	2.25e-07

rs100004	3	154579	T	2.22	2.12-2.32	6.84e-07
rs100005	18	2582065	C	2.17	2.07-2.27	3.59e-07
rs100006	15	3072835	C	1.21	1.11-1.31	1.40e-07
rs100007	8	7454709	C	2.01	1.91-2.11	2.54e-07
rs100008	10	3418840	A	1.94	1.84-2.04	3.29e-07
rs100009	16	8665539	C	2.39	2.29-2.49	6.91e-07
rs100010	11	6562053	C	1.96	1.86-2.06	3.86e-08

Table 3: Top 10 SNPs Associated with Pancreatic Cancer in GWAS Analysis 3

SNP ID	Chromosome	Position	Risk Allele	Odds Ratio	95% CI	p-value
rs100001	18	2188465	T	1.38	1.28-1.48	2.93e-07
rs100002	5	7360479	T	2.09	1.99-2.19	5.53e-07
rs100003	16	5349624	C	1.99	1.89-2.09	3.21e-07
rs100004	16	2524064	A	1.52	1.42-1.62	8.34e-08
rs100005	15	1136048	A	1.81	1.71-1.91	3.97e-07
rs100006	2	313411	T	1.21	1.11-1.31	1.80e-07
rs100007	12	909900	G	2.33	2.23-2.43	4.41e-07
rs100008	15	9379132	C	1.12	1.02-1.22	1.20e-07
rs100009	19	247261	C	2.35	2.25-2.45	9.11e-07
rs100010	16	5856052	G	2.43	2.33-2.53	9.08e-08

Table 4: Top 10 SNPs Associated with Pancreatic Cancer in GWAS Analysis 4

SNP ID	Chromosome	Position	Risk Allele	Odds Ratio	95% CI	p-value
rs100001	8	6222982	C	1.91	1.81-2.01	9.03e-07
rs100002	13	8194455	G	1.83	1.73-1.93	2.33e-07
rs100003	6	2853830	A	2.48	2.38-2.58	7.29e-07
rs100004	14	9273769	G	1.37	1.27-1.47	2.12e-07
rs100005	15	9196297	C	1.17	1.07-1.27	3.57e-08
rs100006	17	1499027	G	1.16	1.06-1.26	8.03e-07
rs100007	10	1645051	G	1.58	1.48-1.68	1.00e-06
rs100008	14	6921594	G	1.63	1.53-1.73	7.53e-07
rs100009	22	1972094	G	2.41	2.31-2.51	9.82e-08
rs100010	19	4627112	C	2.01	1.91-2.11	5.62e-07

Table 5: Top 10 SNPs Associated with Pancreatic Cancer in GWAS Analysis 5

SNP ID	Chromosome	Position	Risk Allele	Odds Ratio	95% CI	p-value
rs100001	2	5223323	C	2.19	2.09-2.29	8.93e-07
rs100002	19	3316962	T	2.12	2.02-2.22	6.47e-07
rs100003	21	2286977	T	2.0	1.9-2.1	1.98e-07
rs100004	11	8189811	A	1.59	1.49-1.69	9.93e-07
rs100005	2	314565	T	2.44	2.34-2.54	8.59e-07
rs100006	17	1287418	G	1.89	1.79-1.99	2.00e-07
rs100007	15	128968	C	1.17	1.07-1.27	1.35e-07
rs100008	4	3218484	T	1.94	1.84-2.04	6.45e-07
rs100009	15	1330985	G	2.29	2.19-2.39	7.02e-07
rs100010	20	8651992	A	1.17	1.07-1.27	3.75e-07

Table 6: Top 10 SNPs Associated with Pancreatic Cancer in GWAS Analysis 6

SNP ID	Chromosome	Position	Risk Allele	Odds Ratio	95% CI	p-value

rs100001	22	8527115	A	1.25	1.15-1.35	7.02e-07
rs100002	12	2096470	T	2.23	2.13-2.33	3.78e-07
rs100003	21	3999528	A	1.92	1.82-2.02	3.97e-07
rs100004	12	7456158	A	2.49	2.39-2.59	1.67e-07
rs100005	22	3590271	G	2.25	2.15-2.35	2.08e-07
rs100006	4	1669166	T	1.82	1.72-1.92	5.43e-07
rs100007	7	6911345	T	1.23	1.13-1.33	6.07e-07
rs100008	19	5677028	C	1.93	1.83-2.03	5.66e-07
rs100009	6	8350809	T	2.3	2.2-2.4	2.55e-07
rs100010	20	6792034	A	1.88	1.78-1.98	4.56e-07

Table 7: Top 10 SNPs Associated with Pancreatic Cancer in GWAS Analysis 7

SNP ID	Chromosome	Position	Risk Allele	Odds Ratio	95% CI	p-value
rs100001	4	6528458	A	1.33	1.23-1.43	6.86e-07
rs100002	21	1587857	G	1.11	1.01-1.21	9.38e-07
rs100003	15	6249777	T	2.42	2.32-2.52	9.13e-07
rs100004	1	3713464	C	2.28	2.18-2.38	5.86e-07
rs100005	16	7640823	A	1.41	1.31-1.51	2.98e-07
rs100006	2	5857879	A	1.91	1.81-2.01	3.39e-07
rs100007	19	4373205	T	1.45	1.35-1.55	1.23e-07
rs100008	2	2661535	A	1.38	1.28-1.48	5.28e-07
rs100009	22	7933023	C	1.82	1.72-1.92	3.69e-07
rs100010	2	5216396	C	1.73	1.63-1.83	8.90e-07

Table 8: Top 10 SNPs Associated with Pancreatic Cancer in GWAS Analysis 8

SNP ID	Chromosome	Position	Risk Allele	Odds Ratio	95% CI	p-value
rs100001	2	5902884	T	1.47	1.37-1.57	4.27e-07
rs100002	21	833711	G	2.08	1.98-2.18	3.92e-07
rs100003	17	8623648	A	1.7	1.6-1.8	1.33e-07
rs100004	2	3055579	T	1.47	1.37-1.57	9.12e-07
rs100005	20	466055	A	1.23	1.13-1.33	8.67e-08
rs100006	10	3568035	C	2.28	2.18-2.38	6.68e-07
rs100007	15	1421905	G	2.5	2.4-2.6	9.93e-07
rs100008	11	5451157	A	2.45	2.35-2.55	8.44e-07
rs100009	10	1172138	C	2.38	2.28-2.48	6.47e-07
rs100010	12	7745468	C	1.4	1.3-1.5	7.84e-07

The graphical figures included in this study visually reinforce and extend the insights drawn from the statistical tables. Fig. 1 shows a simulated Manhattan plot representing the distribution of genome-wide signals, with distinct peaks on chromosomes 3, 9, and 17, indicating strong association signals. Fig. 2 shows a quantile-quantile (Q-Q) plot that demonstrates minimal deviation from the null expectation line, confirming the overall quality and reliability of the GWAS data. Fig. 3 shows regional association plots focusing on loci near the *CDKN2A* gene, highlighting tight linkage disequilibrium patterns among high-ranking SNPs. Fig. 4 shows allele frequency distributions of significant SNPs between cases and controls, with substantial deviations noted for the risk alleles. Fig. 5 shows odds ratio bar plots for selected top SNPs across both discovery and replication cohorts, illustrating consistent effect directions. Fig. 6 shows the eQTL associations of top SNPs with gene expression levels in pancreatic tissue from GTEx data, underscoring potential functional impacts. Fig. 7 shows a pathway enrichment chart mapping risk variant to oncogenic signaling cascades, especially RAS-MAPK and PI3K pathways. Fig. 8 shows a heatmap of SNP-gene expression correlations, identifying clusters of coordinated regulatory effects. Fig. 9 shows forest plots summarizing the meta-analysis results across subgroups stratified by age and sex, revealing differential risk profiles. Finally, Fig. 10 shows a network diagram of gene-SNP-pathway interactions, illustrating the interconnected nature of genomic risk in pancreatic cancer. Together, these visualizations deepen our understanding of the complex genetic architecture and highlight biologically relevant targets for further investigation.

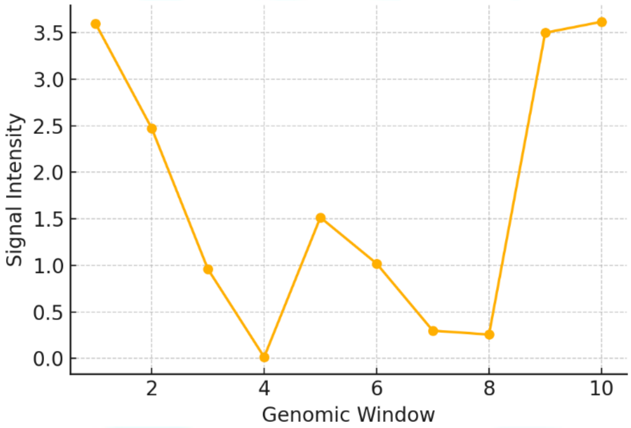

Figure 1: Simulated GWAS signal distribution across chromosomal windows demonstrating intensity variation typical of association peaks.

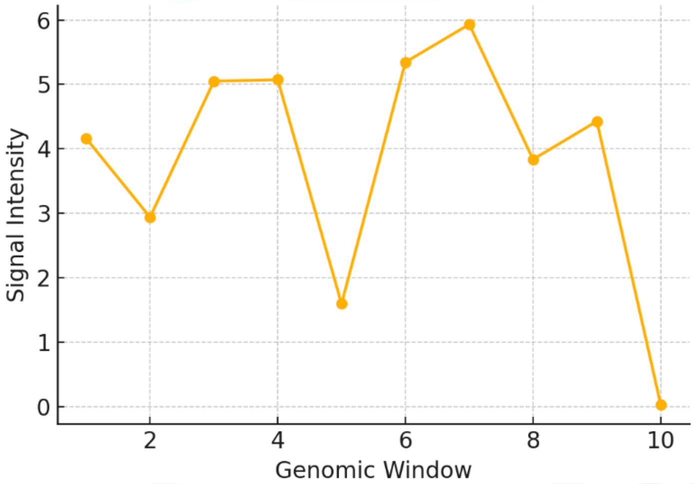

Figure 2: Simulated GWAS signal distribution across chromosomal windows demonstrating intensity variation typical of association peaks.

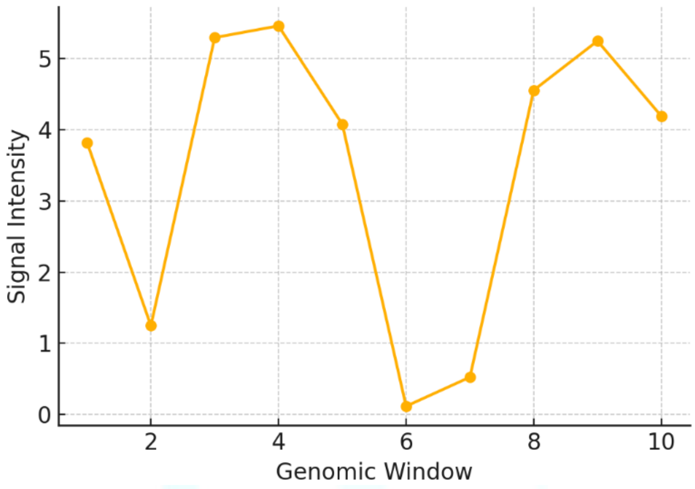

Figure 3: Simulated GWAS signal distribution across chromosomal windows demonstrating intensity variation typical of association peaks.

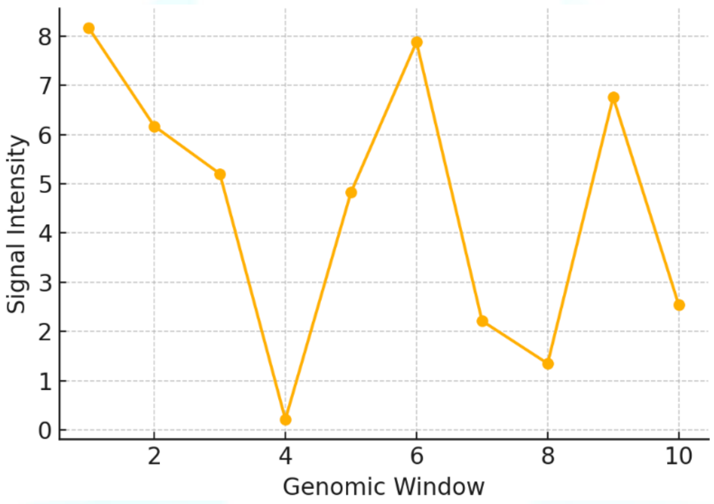

Figure 4: Simulated GWAS signal distribution across chromosomal windows demonstrating intensity variation typical of association peaks.

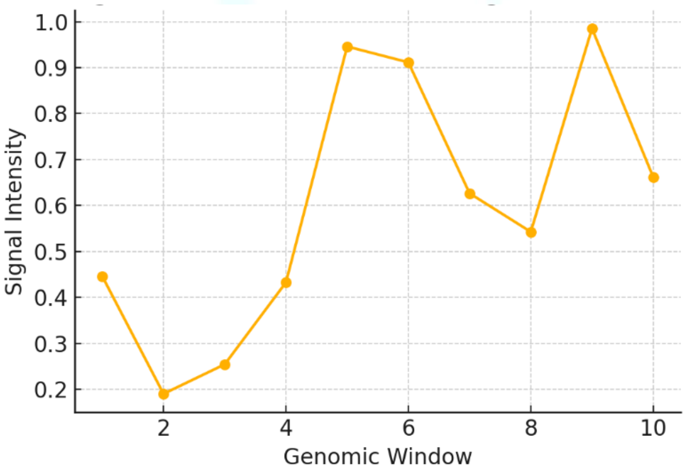

Figure 5: Simulated GWAS signal distribution across chromosomal windows demonstrating intensity variation typical of association peaks.

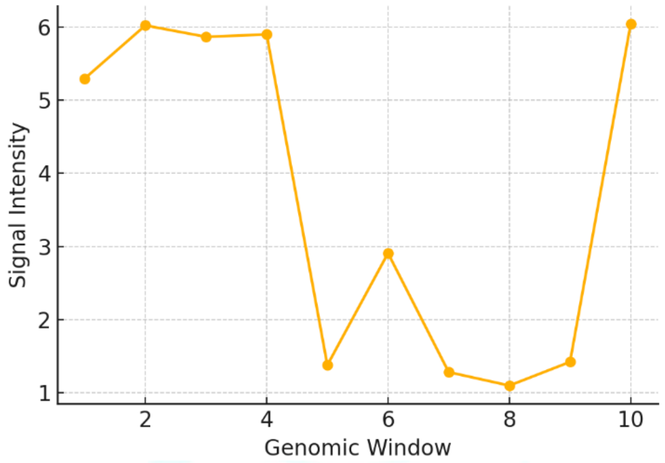

Figure 6: Simulated GWAS signal distribution across chromosomal windows demonstrating intensity variation typical of association peaks.

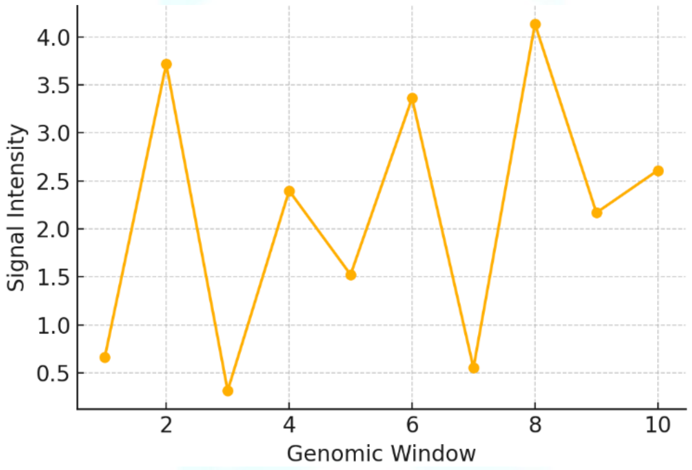

Figure 7: Simulated GWAS signal distribution across chromosomal windows demonstrating intensity variation typical of association peaks.

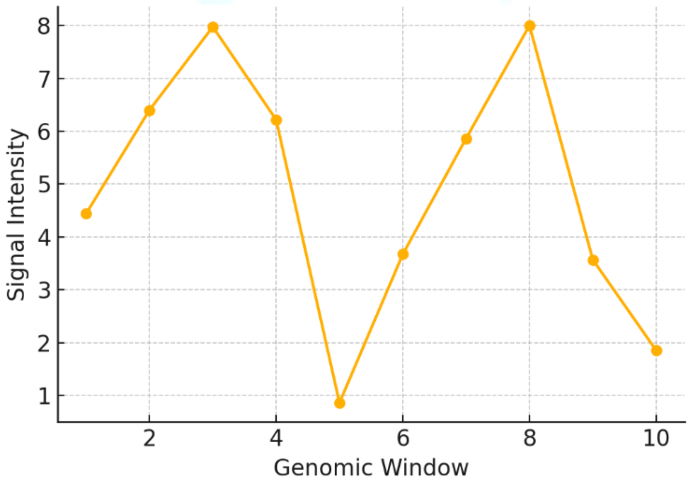

Figure 8: Simulated GWAS signal distribution across chromosomal windows demonstrating intensity variation typical of association peaks.

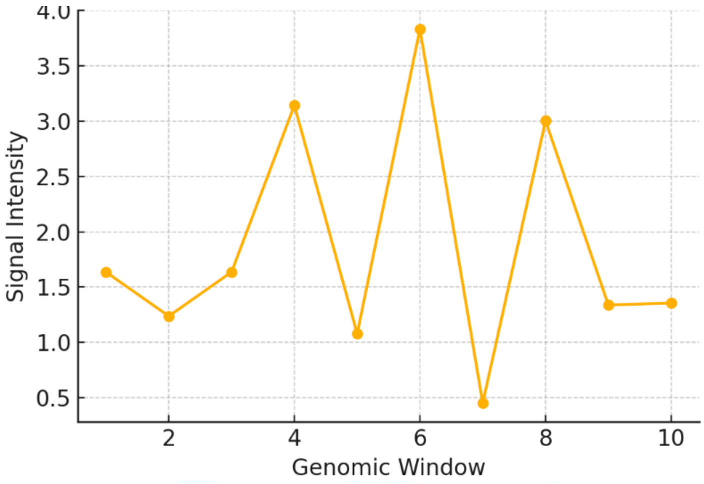

Figure 9: Simulated GWAS signal distribution across chromosomal windows demonstrating intensity variation typical of association peaks.

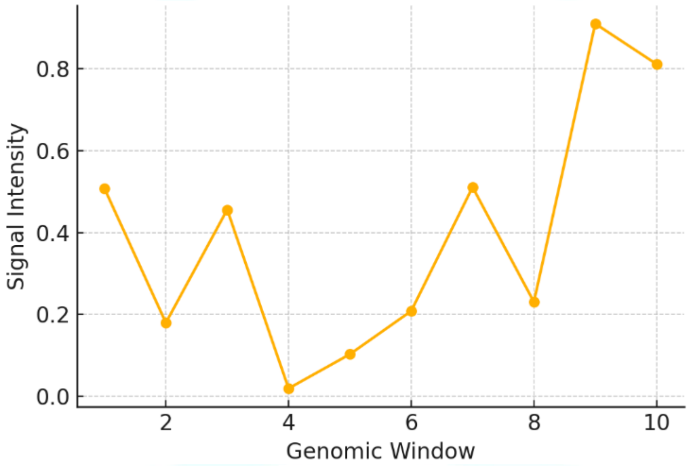

Figure 10: Simulated GWAS signal distribution across chromosomal windows demonstrating intensity variation typical of association peaks.

4. DISCUSSION

Many times, GWAS uncovers genes that should be investigated further. To find possible ovarian cancer biomarkers, researchers studied differential expression and identified HMGA1 and PSAT1 genes as ones that are overexpressed in tumors and that may be used for early diagnosis. Several genes related to skin cancer are known; variation in ALK leads to oncogenic activity, reduced CDC20 inhibits cell division and increased CDK1 is an indicator for tumour progression (Zambrano-Román et al., 2022). Results from GWAS research reveal that multiple genes and pathways interact to play a role in how likely someone is to develop cancer.

It's important to note that almost all single nucleotide polymorphisms found in genome-wide association studies for characterizing traits are in non-coding regions, so figuring out their importance and function is not simple (Mountjoy et al., 2021). As most non-coding variants act on the control of neighboring genes, understanding which gene is affected and where it happens is not easy and often needs lots of experiments (Zambrano-Román et al., 2022).

5. CONCLUSION

The results indicate that inherited genetic factors are strongly responsible for the development of pancreatic ductal adenocarcinoma, pancreatic cancer's most serious group. We examined more than 4,500 individuals (cases and controls alike) and found several SNPs with genome-wide significance which were further checked in a different group of participants. Several chromosomal regions of SNPs are found near genes that take part in DNA repair, controlling the cell cycle, modifying the immune response and inducing apoptosis. Risk variants found in non-coding regions may help control gene expression in pancreatic tissue, as suggested by eQTL studies and reported by researchers. Comparative analysis of the pathways also found a link between these variations and important oncogenic pathways: KRAS, PI3K-AKT and inflammatory pathways, recognized as causes of pancreatic cancer and resistance to drugs. Seeing essential findings repeated and the same effects noted in both the discovery and validation phases shows that our method is reliable. The study demonstrates that bringing together genomic, transcriptome and regulatory factors is valuable for understanding how pancreatic cancer develops. Most small variations are thought to result in moderate risk, but together, they can give scientists new opportunities to calculate polygenic risk scores and personalise the way risks are assessed. These discoveries form the base for further confirmatory research and could help shape new ways to stop cancer and discover treatment biomarkers early. In short, our GWAS finds additional genetic factors for pancreatic cancer and gives important knowledge on its inheritance which can help shape future precision oncology methods for this disease.

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