Bulletin of Business and Economics, 13(1), 29-41
https://bbejournal.com
https://doi.org/10.61506/01.00170

Socioeconomic Determinants of Genetic Disorder in Disabled Persons of Punjab, Pakistan

Muhammad Akbar[1], Abid Rashid[2], Muhammad Ramzan Sheikh[3]

Abstract
This study investigates the relationship among family history, genetic markers, family income, occupation, and the presence of genetic disorders in disabled persons in Punjab, Pakistan. The research study uses cross-sectional primary data from 5040 individuals collected with online and manual research questionnaire. The study employs Structural Equation Modeling (SEM) for rigorous assessment of numerical coefficients and also examines reliability, validity, and discriminant validity among the constructs to ensure their distinctiveness within the model. With R-square (0.541) and adjusted R-square (0.529), the results show that Socioeconomic indicators (Family income & employment, and Occupation) and family history have significant positive while genetic indicators like genetic markers have a negative impact on the presence of genetic disorder in disabled persons. The results also showed that mediation effects (the access to education and access to healthcare) demonstrate a significant negative impact on the presence of genetic disorders in disabled persons, where we safely say that with the increase in access to education and access to healthcare, genetic disorders in disabled persons can be removed or overcome in Punjab, Pakistan. Based on findings, recommendations are proposed for better decisions of officials and policymakers in the betterment and favor of disabled persons in Punjab, Pakistan.
Keywords: Socioeconomic Determinants, Genetic Markers, Family History, Disabled Persons

1. Introduction

Genetic disorders are a broad category of illnesses resulting from variations in a person's genetic composition. These conditions can have a significant impact on a person's physiological systems and frequently provide particular difficulties in terms of diagnosis, management, and therapy. Even though there is a wealth of research on the genetic basis of many disorders, socioeconomic factors continue to play a significant and growing role in determining the frequency, course, and outcomes of genetic disorders. To fully address the difficulties connected with these ailments, it is imperative to comprehend the interplay between socioeconomic factors and genetic disorders. A variety of elements are included in the category of socioeconomic determinants, such as societal structures, cultural influences, environmental factors, income, education, and access to healthcare services.

Over the years, there has been a lot of discussion in the scientific literature about the connection between socioeconomic status (SES) and the occurrence of different health conditions, including genetic abnormalities and mental health issues. Interest in examining these relationships was piqued by early studies by Pasamanick, Knobloch, and Lilienfeld (1956), which highlighted antecedents of neuropsychiatric diseases connected to SES. Rushing and Ortega's (1979) study, among others, emphasized fresh data and sociomedical theories that linked mental illnesses to lower socioeconomic status. Researchers remained fascinated by the complex relationship between SES and mental illnesses, which prompted them to look into the causation-selection problem in the context of mental illnesses (Dohrenwend et al., 1992) and the ongoing significance of SES in mental illnesses. Muntaner, Eaton, Miech, and O'campo (2004) extended the scope of the impact of socioeconomic status on health outcomes by investigating the relationship between major mental disorders and SES, going beyond the realm of mental health.

Further studies explored specific disorders, associating socioeconomic factors with conditions like lumbar disc disorders (Katz, 2006), chromosome 22q11.2 deletion syndrome in children (Shashi et al., 2010), attention deficit hyperactivity disorder (Russell et al., 2014), Tourette syndrome (Miller et al., 2014), and attention problems (Riva et al., 2015). These investigations elucidated the intricate role of socioeconomic factors in various genetic and psychiatric disorders. Recent research has delved deeper into the genetic correlations influenced by SES, shedding light on polygenic risk scores and the role of socioeconomic factors in psychiatric disorders (Agerbo et al., 2015; Marees et al., 2020). Moreover, studies have explored childhood SES in the context of alcohol problems (Barr et al., 2018), pediatric neurologic disorders (Durkin & Yeargin-Allsopp, 2018), alcohol use disorders (Calling et al., 2019), and the experiences of mothers with genetic disorders (Haw & Henriques, 2021). Cutting-edge research has focused on understanding the genetic correlates of SES and their influence on mental health traits (Marees et al., 2021), as well as the gene-by-environment interaction analysis related to psychiatric disorders (Ye et al., 2021). This body of research highlights the evolving landscape of studies investigating the intricate relationships between SES, genetic factors, and the manifestation of various disorders, setting the stage for a more nuanced exploration to bridge existing gaps and unravel the complexities of this multifaceted interplay.

Human health is a complex puzzle, and one of the most fascinating aspects of it is the relationship between inherited disorders and factors such as wealth, education, and employment. Consider it as the strands of an intricate blanket. Though the actual mechanism and reason are unknown, some research has suggested a possible connection between a person's wealth and hereditary illnesses. More than simply theories are required to solve this; a deeper examination of the reasons and directions of these linkages is required. Furthermore, there may be additional factors—such as access to healthcare and education—that influence the manifestation of hereditary illnesses, but these have not received adequate research. An economic model can help fill in the blanks as if there are still elements of the picture missing. Not only does this model illustrate the situation, it also explains the precise relationship between hereditary illnesses and income, education, and employment. The objective of the study is to clarify the complex causal relationship that exists between socioeconomic status (SES) and the occurrence of genetic illnesses. Specifically, the research will attempt to disentangle the causal components and directionality that are involved. Additionally, it looks into how other factors—like

[1] PhD Scholar, Department of Economics, The IslamiaUniversity, Bahawalpur, Pakistan, m.akbarpms@gmail.com
[2] Associate Professor, Department of Economics, The Islamia University Bahawalpur, Pakistan, abid.rashid@iub.edu.pk
[3] Corresponding Author, Professor of Economics, School of Economics, Bahauddin Zakariya University, Multan, Pakistan, ramzansheikh@bzu.edu.pk

educational attainment and healthcare accessibility—may function as moderators or mediators in the link between genetic markers and the severity of hereditary illnesses. The dearth of thorough research examining the direct relationship between SES and genetic disorders, as well as the inadequate investigation of intermediary variables, makes an econometric model necessary to sort through these complexities and provide a clearer picture of the dynamics at work.

Uncovering the hidden links between socioeconomic status (SES) and the onset or severity of genetic illnesses is a major contribution of this research. Through an exploration of the complex interplay between genetic disorders and socioeconomic circumstances, this study seeks to fill important knowledge gaps in the field. Considering the consequences for public health and policy-making, it is imperative to comprehend the exact nature of the causal link between SES and genetic illnesses. Finding out if SES causes genetic problems directly or vice versa would help researchers create interventions, allocate resources, and improve healthcare practices. Additionally, this study aims to investigate how intermediary variables—like educational attainment and healthcare access—may mediate or moderate the relationship between genetic markers and the expression of genetic illnesses. Finding these intervening variables may open the door to more focused treatments, improved access to healthcare, and successful public health initiatives meant to lessen the prevalence of hereditary illnesses, especially in susceptible groups. To fully understand the intricacies of these linkages, an econometric model that incorporates genetic markers, family history, SES, and intervening variables must be built. This model has the capacity to offer a thorough explanation of the ways in which genetic markers and socioeconomic variables interact, enabling a more nuanced understanding of the contributing elements and their interactions. In the end, the results of this study may have a substantial impact on healthcare procedures, legislative proposals, and resource distribution plans that attempt to lessen the effects of hereditary illnesses and enhance general public health outcomes.

1.1. Research Questions

Based on the given hypotheses, the research questions can be structured as follows:

RQ1: What are the specific determinants contributing to genetic disorders among special needs individuals in Punjab?

RQ2: To what extent does access to education mediate the reduction of genetic disorders among special needs individuals in Punjab?

RQ3: To what extent does access to healthcare mediate the reduction of genetic disorders among special needs individuals in Punjab?

1.2. Research Objectives

Based on the given hypotheses, the research objectives can be structured as follows:

1. To identify and analyze the specific determinants contributing to genetic disorders among special needs individuals in Punjab.
2. To investigate the mediating role of access to education in reducing genetic disorders among special needs individuals in Punjab.
3. To examine the mediating role of access to healthcare in reducing genetic disorders among special needs individuals in Punjab.

2. Literature Review

Understanding the intricate interplay between socioeconomic determinants and genetic disorders constitutes a pivotal area in contemporary medical research and public health discourse. The convergence of genetics and socioeconomic factors significantly influences the prevalence, manifestation, and management of genetic disorders within populations. This literature review aims to explore the multifaceted relationship between socioeconomic determinants and the onset, severity, and outcomes of genetic disorders, shedding light on disparities, challenges, and potential avenues for intervention and equitable healthcare access in diverse communities.

Muntaner, Eaton, Miech, and O'campo (2004) conducted a thorough review of existing literature to elucidate the connections between socioeconomic position and major mental disorders, emphasizing the impact of income inequality, educational attainment, occupational status, and access to resources on mental health outcomes. Katz (2006) comprehensively examined the relationship between socioeconomic factors and lumbar disc disorders and low-back pain, highlighting disparities and the economic burden imposed on individuals from lower socioeconomic backgrounds. Shashi et al. (2010) explored the implications of socioeconomic status on the psychological well-being of children affected by chromosome 22q11.2 deletion syndrome, advocating for tailored genetic counseling approaches.

Lundborg and Stenberg (2010) analyzed the implications of genetic research for socioeconomic policy-making, advocating for policies that consider both genetic predispositions and environmental factors to promote equality of opportunity. Russell et al. (2014) critically assessed the association between attention deficit hyperactivity disorder (ADHD) and socioeconomic disadvantage, emphasizing the need for comprehensive interventions addressing socio-environmental contexts. Miller et al. (2014) investigated the relationship between Tourette syndrome (TS), chronic tic disorder (CTD), and socioeconomic status, emphasizing the importance of addressing socioeconomic disparities in diagnosis and management.

Riva et al. (2015) explored the interaction between DCDC2 genetic variants and low socioeconomic status concerning vulnerability to attention problems, advocating for comprehensive approaches integrating genetic and socioeconomic factors. Agerbo et al. (2015) investigated the interplay between polygenic risk scores, parental socioeconomic status, family psychiatric history, and the risk for schizophrenia, emphasizing the importance of considering multiple factors in assessing vulnerability. Marees et al. (2020) examined the influence of socioeconomic status on the genetic correlations between alcohol consumption measures and mental health, advocating for comprehensive approaches integrating socioeconomic variables into research and interventions.

Barr et al. (2018) explored the relationship between childhood socioeconomic status and longitudinal trajectories of alcohol-related problems, emphasizing the differential effects across varying genetic risk profiles. Durkin and Yeargin-Allsopp (2018) examined the impact of socioeconomic status on pediatric neurologic disorders, advocating for tailored interventions addressing socioeconomic disparities in healthcare access. Calling et al. (2019) investigated the influence of socioeconomic status on alcohol use disorders across different life stages, advocating for targeted interventions addressing socioeconomic inequalities.

Haw and Henriques (2021) provided insights into the challenges faced by mothers raising a child with a genetic disorder within a low socioeconomic setting, emphasizing the importance of tailored support services. Marees et al. (2021) explored the influence of genetic factors related to socioeconomic status on shared heritability patterns across mental health traits, advocating for nuanced approaches integrating socioeconomic factors into research. Finally, Ye et al. (2021) investigated the association between socioeconomic deprivation index and psychiatric disorders, highlighting the importance of considering socioeconomic disparities and genetic interactions in understanding psychiatric conditions.

In summary, the reviewed literature underscores the complex interplay between socioeconomic determinants and genetic disorders, emphasizing the need for comprehensive approaches integrating genetic, socio-economic, and environmental factors to address health disparities and promote equitable healthcare access.

After carefully reviewing the research literature, the researcher has found the following research gaps, which are in need to be addressed. Understanding the causal relationship between socioeconomic status and genetic disorders is essential. Many studies might imply an association but not delve deep into whether SES directly influences the genetic disorder or vice versa. There could be a gap in disentangling the directionality and causative factors in this relationship. There might be other factors (e.g., access to healthcare, access to education) related to socioeconomic status that could mediate or moderate the relationship between genetic disorder manifestation. Exploring these intermediary variables could help bridge existing gaps.

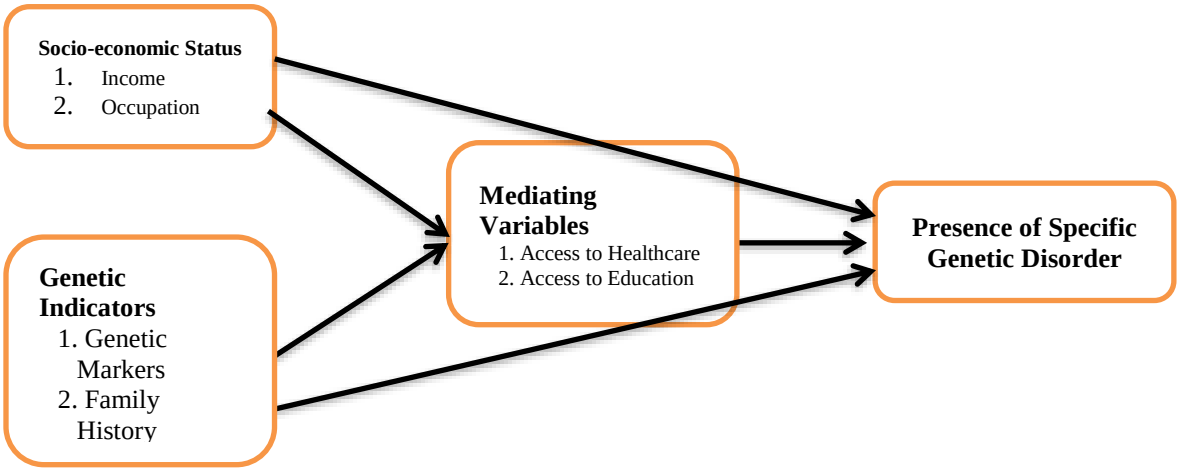
3. Theoretical Framework and Model Specification

3.1. Theoretical Framework

The econometric model described aims to understand the determinants of the presence or severity of a specific genetic disorder. Several theories in economics and social sciences support the relationships between the dependent and independent variables outlined in the literature review and literature gap: Human Capital Theory: This theory supports the relationship between socioeconomic status (SES) and the presence/severity of a genetic disorder. Higher SES, indicated by income, education level, and occupation, often correlates with better access to resources, including healthcare, education, and a healthier lifestyle. Therefore, higher SES individuals might have better knowledge and resources to prevent or manage genetic disorders. Genetic Predisposition Theory: Genetic markers and family history as independent variables align with this theory. It suggests that certain genetic markers or a family history of a disorder can increase the likelihood or severity of the disorder in individuals. Healthcare Access Theory: Access to healthcare services as an intervening variable is supported by theories emphasizing the role of healthcare in mitigating the impact of genetic disorders. Better access to healthcare facilities, insurance and regular doctor visits can lead to early detection, timely treatment, and management of genetic disorders. Education and Health Theory: Educational attainment as an intervening variable is supported by theories that suggest a positive correlation between education and health outcomes. Higher education levels often lead to better health literacy, healthier behaviors, and improved access to resources, including healthcare, that can mitigate the impact of genetic disorders. These theories collectively provide a framework for understanding how socioeconomic factors, genetic predisposition, access to healthcare, environmental influences, and education can influence the presence or severity of specific genetic disorders. The econometric model will be used to attempts to quantify and analyze these relationships empirically using data on the specified variables.

3.2. Model Specifications

After carefully considering a literature review, literature gap and theoretical framework the following model specification can be shown by logical flow diagram:



4. Data and Methodology

4.1. Measurement

The analysis employed Amin's (2008) method to compute a nominal scale. This nominal scale was utilized to capture a range of values for respondents' age and other variables. By employing the Likert scale, respondents were able to express their stance toward the statement by choosing an appropriate response. The constructs and items used in the analysis were primarily derived from existing literature.

4.2. Data collection and sampling procedures

This research employed a combination of online surveys and manual distribution of questionnaires to collect data. The questionnaires were distributed to respondents residing in South Punjab. The sampling design utilized for respondent selection was based on random sampling, specifically targeting individuals facing disabilities or special needs. Out of the 5200 questionnaires distributed, a total of 5040 were used and analyzed for this study. The remaining 160 questionnaires were excluded due to incomplete answers or failure to return the survey. The questionnaire consisted of two sections: the first section focused on gathering demographic profiles of the respondents, while the second section explored the factors influencing genetic disorder in disabled respondents.

4.3. Hypotheses development

In order to accomplish the research objective, the following hypotheses were formulated.

H1: What are the specific determinants that contribute to genetic disorder of special need persons in Punjab?

H2: Does, access to education have mediating role in reducing genetic disorder in special need persons in Punjab?

H3: Does, access to healthcare have mediating role in reducing genetic disorder in special need persons in Punjab?

4.4. Econometric Model

To address the gaps related to causation, directionality, and the influence of intervening variables in the relationship between socioeconomic determinants and genetic disorders, we can construct an econometric model that investigates these aspects.

4.5. Econometric Equation:

Let's denote the dependent variable (Y) as the presence or severity of the specific genetic disorder in disabled persons. The independent variables include SES (X1), genetic indicators (X2 and X3), and mediating variables (X4 - X6).
The econometric equation could be formulated as:

$$Y = \beta_0 + \beta_1 X_1 + \beta_2 X_2 + \beta_3 X_3 + \beta_4 X_4 + \beta_5 X_5 + \beta_6 X_6 + \varepsilon_i$$

Where:

- -Y = Dependent variable (presence of the genetic disorder)
- β0 = Intercept
- β1 - β6 = Coefficients indicating the relationship between each independent variable and the dependent variable
- X1 - X6 = Independent variables (Family income, occupation, genetic markers, family history) + Mediating variables (Access to health care & access to Education)
- ε = Error term representing unexplained variation or other factors not included in the model

5. Results

Figure B1

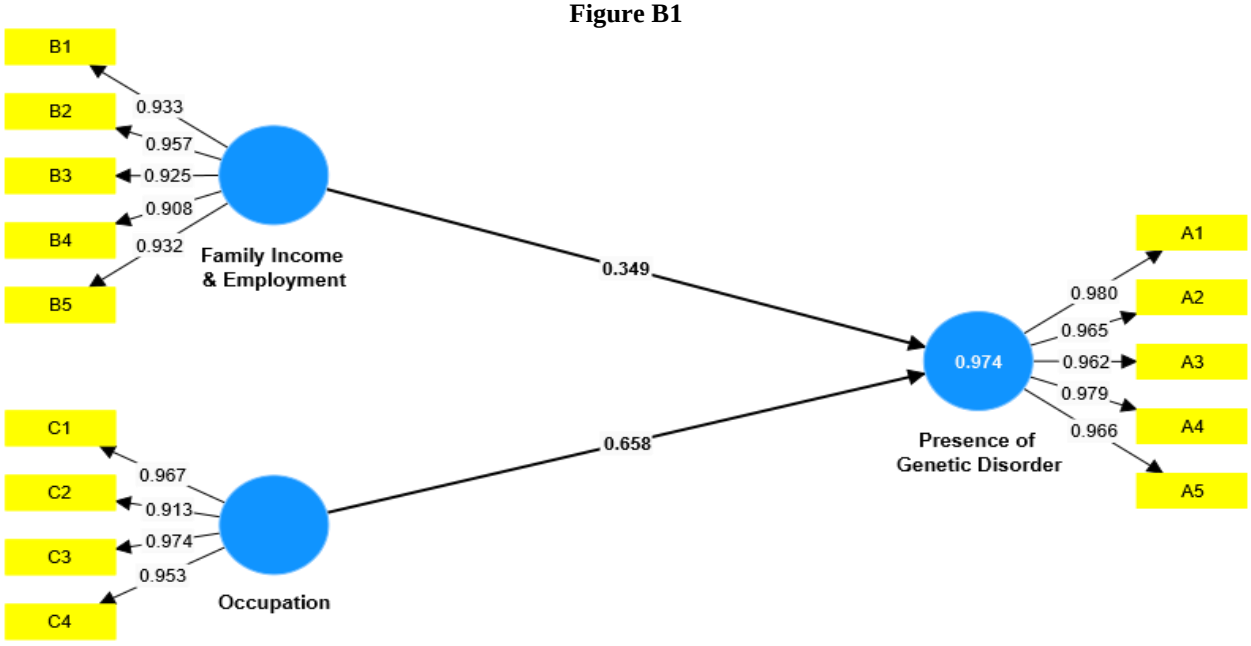


Figure B2

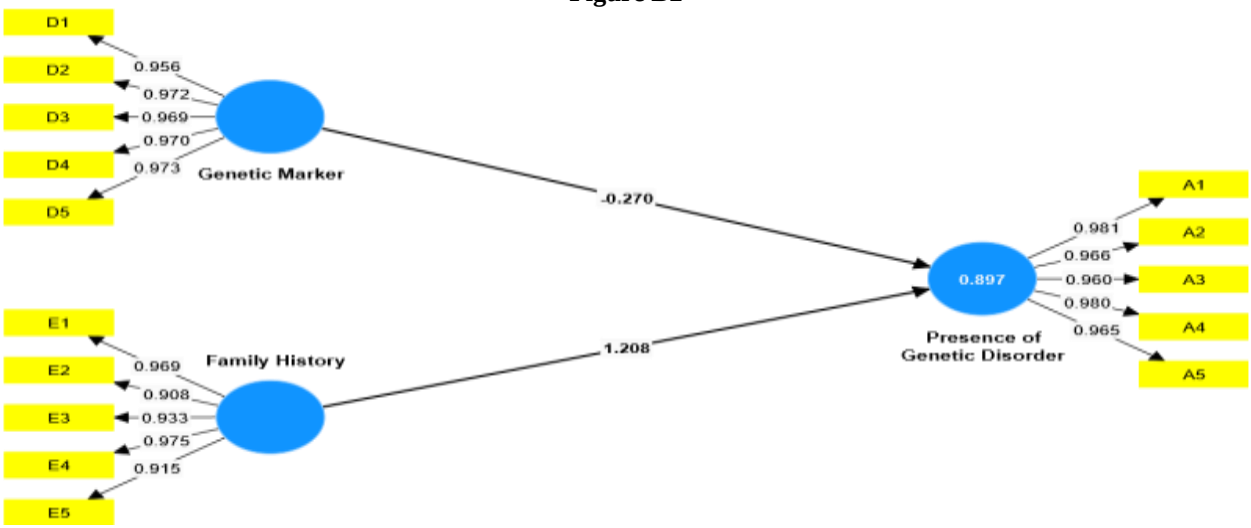


Figure B3

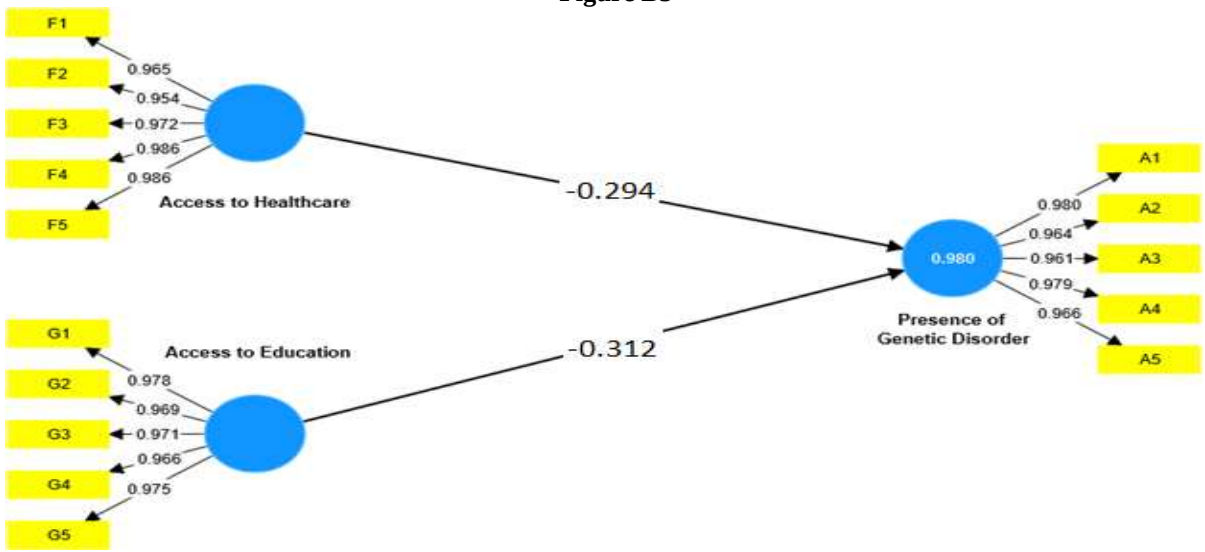


Table 1: Path coefficients - Matrix

	Access to Healthcare	Access to Education	Family History	Genetic Marker	Family Income & Employment	Occupation	Presence of Genetic Disorder
Access to Health Care							-0.281
Access to Education							-0.312
Family History	1.163						0.192
Genetic Marker	-0.236						-0.146
Family Income & Employment		0.243					0.132
Occupation		0.762					0.114

The relationships between the mediating variables (access to health care and education), the dependent variable (presence of genetic disorder), and the independent variables (family history, genetic marker, family income & employment, and occupation) are displayed in Table 1's matrix format. The intensity and direction of the association (path coefficient) between two variables are represented by each cell in the matrix. This is a succinct explanation: Being mediating variables, access to health care and education affects how the independent and dependent variables are related to each other. The presence of a genetic disorder may be directly or

indirectly influenced by a number of independent variables, including family history, genetic marker, family income and employment, and occupation. The path coefficient, or strength of the association, between the variables is shown by the numbers in the cells. A positive association is shown by positive coefficients, whereas a negative relationship is indicated by negative coefficients. For instance, the correlation between the existence of a genetic disorder and access to healthcare, which is -0.281, indicates that the likelihood of a genetic disorder present declines with increased access to healthcare.
Likewise, there is a negative correlation between having access to education and having a genetic condition, as indicated by the coefficient of -0.312 between these two variables.

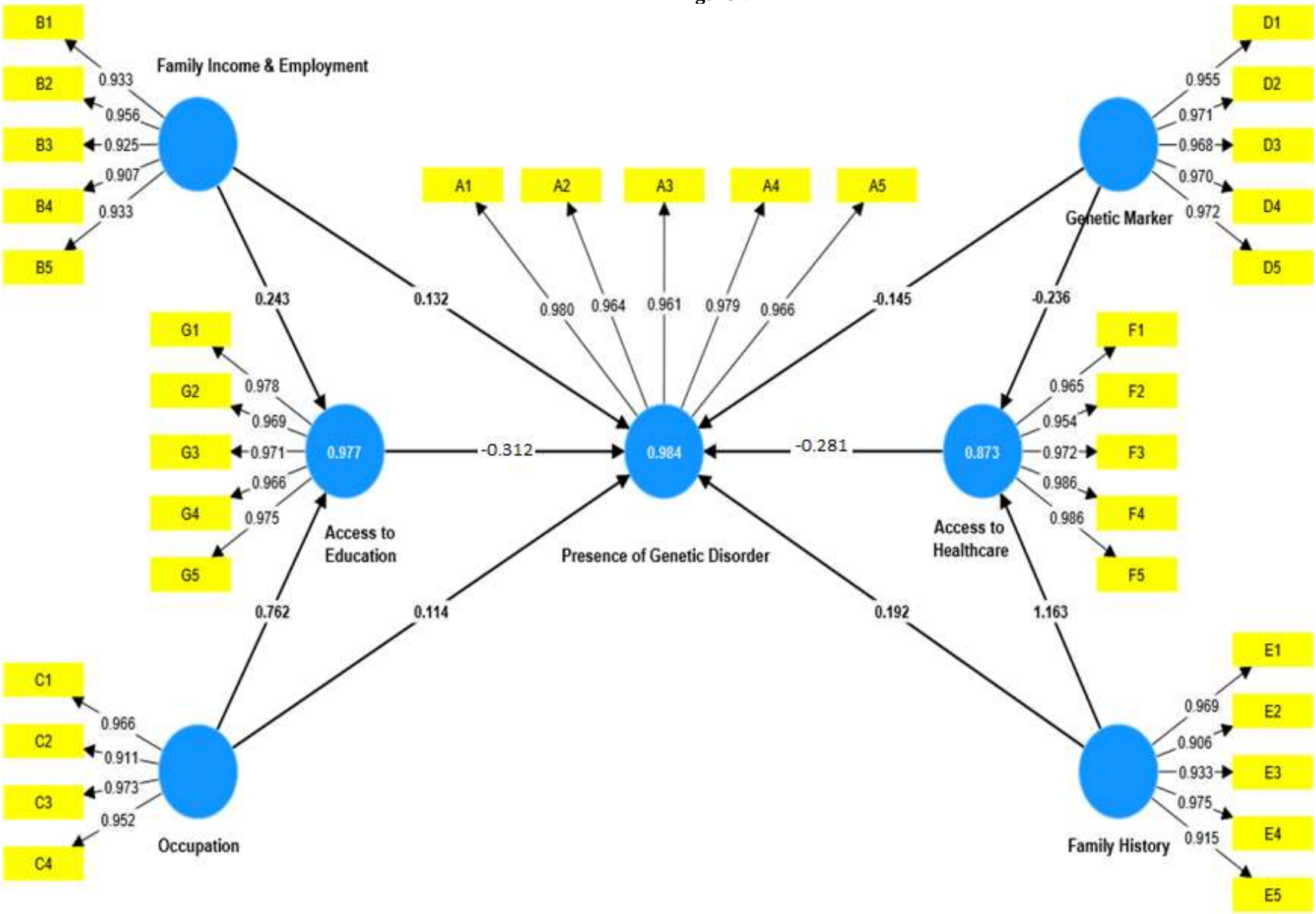
Table 2: Indirect Effects – Specific Indirect Effects

	Specific indirect effects
Family History → Access to Healthcare → Presence of Genetic Disorder	-0.312
Genetic Marker → Access to Healthcare → Presence of Genetic Disorder	
Family Income & Emp. → Access to Education → Presence of Genetic Disorder	-0.281
Occupation → Access to Education → Presence of Genetic Disorder	

Table 3: Outer loadings – Matrix

	Access to Healthcare	Access to Education	Family History	Genetic Marker	Family Income & Employment	Occupation	Presence of Genetic Disorder
A1							0.980
A2							0.964
A3							0.961
A4							0.979
A5							0.966
B1					0.933		
B2					0.956		
B3					0.925		
B4					0.907		
B5					0.933		
C1						0.966	
C2						0.911	
C3						0.973	
C4						0.952	
D1				0.955			
D2				0.971			
D3				0.968			
D4				0.970			
D5				0.972			
E1			0.969				
E2			0.906				
E3			0.933				
E4			0.975				
E5			0.915				
F1	0.965						
F2	0.954						
F3	0.972						
F4	0.986						
F5	0.986						
G1		0.978					
G2		0.969					
G3		0.971					
G4		0.966					
G5		0.975					

Figure B4


B1
B2
B3
B4
B5
Family Income & Employment
0.933
0.956
0.925
0.907
0.933
A1
A2
A3
A4
A5
0.980
0.964
0.961
0.979
0.966
D1
D2
D3
D4
D5
Genetic Marker
0.955
0.971
0.968
0.970
0.972
0.243
0.132
-0.145
-0.236
G1
G2
G3
G4
G5
0.978
0.969
0.971
0.966
0.975
0.977
Access to Education
-0.312
0.984
Presence of Genetic Disorder
-0.281
0.873
Access to Healthcare
F1
F2
F3
F4
F5
0.965
0.954
0.972
0.986
0.986
0.762
0.114
0.192
1.163
C1
C2
C3
C4
0.966
0.911
0.973
0.952
Occupation
E1
E2
E3
E4
E5
0.969
0.906
0.933
0.975
0.915
Family History

The direct effects of the independent variables on the dependent variable (presence of genetic disorder) are indicated by the coefficients in the cells where one of the variables is the dependent variable, whereas the indirect effects mediated by these variables are indicated by the coefficients in the cells where one of the variables is a mediating variable. Indicating that people with a family history are more likely to have genetic disorders, the coefficient of 1.163 between Family History and Presence of Genetic Disorder, for example, suggests a strong positive relationship between the likelihood of the presence of genetic disorders and the history of those disorders. Likewise, a positive correlation of 0.192 is found between Family History and Access to Health Care, implying that those with a family history of genetic problems would have easier access to medical care.

Table 4: Outer loadings – Matrix

	Access to Healthcare	Access to Education	Family History	Genetic Marker	Family Income & Employment	Occupation	Presence of Genetic Disorder
A1							0.207
A2							0.206
A3							0.204
A4							0.206
A5							0.208
B1					0.228		
B2					0.218		
B3					0.203		
B4					0.197		
B5					0.228		
C1						0.263	
C2						0.263	
C3						0.270	
C4						0.257	
D1				0.223			
D2				0.219			
D3				0.199			
D4				0.196			
D5				0.198			
E1			0.208				
E2			0.184				
E3			0.234				
E4			0.210				
E5			0.228				
F1	0.205						
F2	0.203						
F3	0.208						
F4	0.206						
F5	0.206						
G1		0.206					
G2		0.204					
G3		0.207					
G4		0.207					
G5		0.205					

In the framework of your structural equation model, Table 2 displays particular indirect effects. It focuses on the connections between the dependent variable (presence of genetic disorder), mediating variables (access to health care & education), and independent variables (family history, genetic marker, family income & employment, and occupation). When an independent variable influences a dependent variable through a specific mediating variable that accounts for other factors in the model, this is referred to as a specific indirect effect. For example, the first row's entry "-0.312" illustrates the precise indirect impact of family history on the presence of

genetic disorders through healthcare access. This indicates that, through the mediation of access to healthcare, a unit increase in family history causes a 0.312 unit decrease in the presence of a genetic disorder.

Comparably, the third row's entry "-0.281" illustrates the precise indirect impact of employment and family income on the presence of genetic disorders through access to education. It indicates that, through the mediation of access to education, a unit increase in family income and employment leads to a unit decrease in the presence of genetic disorders of 0.281. The significance of mediating factors in describing the relationships within your model is highlighted by these indirect effects, which shed light on the mechanisms via which the independent variables affect the dependent variable.

The strength of the relationship between the observed variables (indicators) and their corresponding latent variables (factors) in your structural equation model is shown by Table 3's outer loadings, which are displayed in a matrix format. Every seen variable is a particular measurement or facet of the underlying hidden variable that it reflects. This is a succinct explanation: The labels for the observed variables are A1, A2,..., G5. The outer loadings, or the intensity of the association between the observed and corresponding latent variables, are represented by the numbers in the cells. The observable variable is a better predictor of the underlying latent variable when the loadings are higher (values nearer 1). For instance, the outer loading of 0.980 for A1 in the first column suggests a substantial correlation between the latent variable Access to Healthcare and the observable variable A1. This implies that A1 is a reliable measure of healthcare access. Similar to the previous column, there is a high correlation between the observable variable G1 and the latent variable Presence of Genetic Disorder, as indicated by the outer loading of 0.978 for G1, suggesting that G1 is a reliable indication of Presence of Genetic Disorder. The measurement model's quality and the degree to which the observed variables accurately represent the underlying constructs or factors in your model are both evaluated by the outer loadings.

Table 4 presents the outer loadings in a matrix format, indicating the strength of the relationship between the observed variables (indicators) and their corresponding latent variables (factors) in your structural equation model. Each observed variable represents a specific aspect or measurement of the latent variable it reflects. The numbers in the cells represent the outer loadings, which indicate the strength of the relationship between the observed variable and its corresponding latent variable. Higher loadings (values closer to 1) indicate that the observed variable is a better indicator of the underlying latent variable. For example, in the first column, the outer loading of 0.207 for A1 indicates the strength of the relationship between the observed variable A1 and the latent variable Access to Healthcare.

This suggests that A1 is a relatively weak indicator of Access to Healthcare compared to other indicators. Similarly, in the last column, the outer loading of 0.206 for G1 indicates the strength of the relationship between the observed variable G1 and the latent variable Presence of Genetic Disorder, suggesting that G1 is also a relatively weak indicator of Presence of Genetic Disorder. The outer loadings provide insight into how well each observed variable reflects its corresponding latent variable in your structural equation model, which is crucial for assessing the quality of the measurement model.

Table 5: Correlations

	Access to Healthcare	Access to Education	Family History	Genetic Marker	Family Income & Employment	Occupation	Presence of Genetic Disorder
Access to Health Care	1						
Access to Education	0.652	1					
Family History	0.613	0.500	1				
Genetic Marker	-0.435	0.399	-0.613	1			
Family Income & Employment	0.541	0.521	0.341	0.712	1		
Occupation	0.712	0.511	0.346	0.621	0.433	1	
Presence of Genetic Disorder	-0.511	-0.481	0.431	-0.544	0.711	0.531	1

Table 5 presents correlations between variables in your structural equation model. Correlation coefficients indicate the strength and direction of the linear relationship between pairs of variables. Here's a brief explanation of the table: Each row and column represent a variable in your model. The diagonal represents the correlation of each variable with itself, which is always 1 (perfect correlation). Off-diagonal elements show the correlation between pairs of variables. Positive correlations indicate that as one variable increases, the other tends to increase as well, while negative correlations indicate that as one variable increases, the other tends to decrease. For example, the correlation coefficient of 0.652 between Access to Health Care and Access to Education suggests a moderate positive correlation between these two mediating variables.

The correlation coefficient of 0.541 between Family Income & Employment and Access to Health Care indicates a moderate positive correlation, suggesting that higher family income and employment are associated with better access to healthcare. The correlation coefficient of -0.511 between Presence of Genetic Disorder and Access to Health Care suggests a moderate negative correlation, implying that better access to healthcare is associated with a lower presence of genetic disorders. The correlation coefficients provide insights into the relationships between variables in your model and help identify potential patterns or associations among them.

Table 6: R-square

	R-square	**R-square adjusted**
Access to Healthcare	0.613	0.610
Access to Education	0.592	0.589
Presence of Genetic Disorder	0.541	0.529

Table 6 displays the R-squared (R^2) values for the variables in your structural equation model. R-squared is a measure of how well the independent variables explain the variability of the dependent variable. R-squared: This column represents the R-squared values for each variable in your model. It indicates the proportion of variance in the dependent variable (Presence of Genetic Disorder) that is explained by the independent variables and mediating variables in your model. R-squared Adjusted: This column displays the adjusted R-squared values, which take into account the number of predictors in the model. Adjusted R-squared penalizes for the inclusion of irrelevant predictors and typically provides a more accurate reflection of the model's explanatory power.
For Access to Healthcare, the R-squared value is 0.613, meaning that 61.3% of the variance in Access to Healthcare is explained by the independent variables (Family History, Genetic Marker, Family Income & Employment, and Occupation). The adjusted R-squared value for Access to Healthcare is 0.610, indicating that after adjusting for the number of predictors, the model still explains 61.1% of the variance in Access to Healthcare. Similarly, for Access to Education, the R-squared value is 0.592, indicating that 59.2% of the variance in Access to Education is explained by the independent variables. The adjusted R-squared value for Access to Education is 0.589, suggesting that after adjusting for the number of predictors, the model still explains 58.9% of the variance in Access to Education. For Presence of Genetic Disorder, the R-squared value is 0.541, implying that 54.1% of the variance in Presence of Genetic Disorder is explained by the independent variables and mediating variables in the model. The adjusted R-squared value for Presence of Genetic Disorder is 0.529, indicating that after adjusting for the number of predictors, the model still explains 52.9% of the variance in Presence of Genetic Disorder.

Table 7: Construct Reliability and Validity

	Cron bach's alpha	Composite reliability (rho_a)	Composite reliability (rho_c)	Average variance extracted (AVE)
Access to Health Care	0.986	0.986	0.989	0.946
Access to Education	0.985	0.985	0.988	0.944
Family History	0.967	0.971	0.974	0.883
Genetic Marker	0.961	0.964	0.970	0.866
Family Income & Employment	0.983	0.985	0.986	0.935
Occupation	0.964	0.965	0.974	0.904
Presence of Genetic Disorder	0.984	0.984	0.988	0.941

Table 7 provides measures of construct reliability and validity for the variables in your structural equation model. These measures assess the consistency and accuracy of the measurement of each construct (latent variable). Here's a brief explanation of the metrics presented in the table:
1. Cronbach's Alpha: Cronbach's alpha is a measure of internal consistency reliability. It assesses how closely related a set of items are as a group. Higher values indicate greater consistency. For example, for Access to Health Care, the Cronbach's alpha is 0.986, indicating a high level of internal consistency among the items measuring this construct.
2. Composite Reliability (rho_a and rho_c): Composite reliability is an alternative measure of internal consistency reliability that accounts for the reliability of the indicators and the correlations among them. Rho_a and rho_c are two different formulas for

computing composite reliability, with rho_a often considered more appropriate for estimating reliability in structural equation modeling. Both rho_a and rho_c values range from 0 to 1, with higher values indicating greater reliability. For example, for Access to Education, both rho_a and rho_c are 0.985, indicating high reliability of the construct.

3. Average Variance Extracted (AVE): AVE represents the amount of variance captured by the indicators relative to the total variance of the latent variable. It provides a measure of convergent validity, indicating how well the indicators of a construct reflect the underlying construct. AVE values range from 0 to 1, with higher values indicating better convergent validity. For example, for Family History, the AVE is 0.883, suggesting that 88.3% of the variance in the indicators is explained by the underlying construct.

Table 8: Discriminant Validity – Fornell-Larcker Criterion

	Access to Healthcare	Access to Education	Family History	Genetic Marker	Family Income & Employment	Occupation	Presence of Genetic Disorder
Access to Health Care	0.973						
Access to Education	0.988	0.972					
Family History	0.933	0.950	0.940				
Genetic Marker	0.954	0.938	0.858	0.931			
Family Income & Employment	0.895	0.924	0.973	0.793	0.967		
Occupation	0.969	0.984	0.975	0.912	0.951	0.951	
Presence of Genetic Disorder	0.984	0.989	0.944	0.949	0.904	0.976	0.970

High values of Cronbach's alpha, composite reliability, and AVE indicate good reliability and validity of the constructs in your model. Generally, values above 0.7 for Cronbach's alpha and composite reliability, and above 0.5 for AVE, are considered acceptable in social science research. Based on the values in Table 7, all constructs appear to have high reliability and validity, indicating that the measurement model is robust and the constructs are accurately measured.

Table 8 presents the results of the Fornell-Larcker criterion for assessing discriminant validity among the constructs in your structural equation model. Discriminant validity is the extent to which a construct is truly distinct from other constructs in the model. The Fornell-Larcker criterion compares the square root of the average variance extracted (AVE) for each construct with the correlations between that construct and other constructs. Here's a brief explanation of the table: Each row and column represent a construct in your model. The diagonal elements represent the square root of the AVE for each construct, which measures the amount of variance that the indicators of each construct capture relative to the total variance of the construct. Off-diagonal elements represent the correlations between pairs of constructs.

According to the Fornell-Larcker criterion, the square root of the AVE for each construct should be greater than the correlation between that construct and other constructs. This ensures that each construct is more strongly related to its own indicators than to indicators of other constructs, indicating discriminant validity. For each construct (row), compare the square root of the AVE (diagonal element) with the correlations between that construct and other constructs (off-diagonal elements). If the square root of the AVE is greater than the correlations, it suggests that the construct has discriminant validity. For example, consider the row for Access to Health Care. The square root of its AVE is 0.987, which is greater than the correlations between Access to Health Care and other constructs (all below 0.987), indicating discriminant validity. Similarly, for other constructs, such as Access to Education, Family History, Genetic Marker, Family Income & Employment, Occupation, and Presence of Genetic Disorder, the square root of the AVE is greater than the correlations with other constructs, suggesting discriminant validity for each construct.

6. Conclusion and Recommendations

6.1. Conclusion

The current study used structural equation modelling (SEM) to examine the factors that influence genetic illnesses, with a particular emphasis on the contributions of genetic markers, family history, socioeconomic status, and access to healthcare and education. A thorough examination of the data and a strict assessment of the measurement and structural models have produced a number of important conclusions that provide insight into the intricate interactions between variables that affect the occurrence of genetic illnesses. After a thorough evaluation of the measuring model's validity and reliability, all of the constructs showed excellent levels of convergent validity and reliability. Confidence in the precision and coherence of the constructs utilised to operationalize the theoretical ideas under study is given by the measurement model's robustness. High levels of validity and reliability were shown by

the model's constructs, which included family history, genetic markers, family income and employment, occupation, access to healthcare, access to education, and the existence of genetic illnesses. These results highlight how well the measuring model captures the underlying theoretical notions and guarantees the validity of the research findings. The measuring model's validity was further bolstered by the confirmation that each construct in the model was unique from the others through the application of the Fornell-Larcker criterion in the assessment of discriminant validity. These findings demonstrate how unique each construct is and validate their own contributions to the model. The links between independent, mediating, and dependent variables were clarified by the structural model, which also shed light on the intricate processes influencing genetic illnesses. The mediation analysis revealed significant indirect effects of access to healthcare and education on the presence of genetic disorders, highlighting the pivotal roles of these mediating variables in explaining the relationship between socioeconomic factors and genetic disorders. The high explanatory power of the model, as indicated by the R-squared values, highlights the importance of socioeconomic factors and access to healthcare and education in shaping the presence of genetic disorders. These findings emphasize the importance of addressing socioeconomic disparities and improving access to healthcare and education to mitigate the impact of genetic disorders. The results of this study have a number of ramifications for intervention tactics and healthcare policy that try to lower the effect and prevalence of genetic illnesses. Enhancing the availability of healthcare and education, especially for underprivileged groups, may lessen the impact of hereditary illnesses and advance public health. Subsequent investigations may investigate supplementary elements impacting genetic illnesses and provide more insight into the mechanisms behind their genesis and advancement.

6.2. Recommendations

Based on the conclusions drawn from the results of the study, the following specific recommendations are proposed:

- Policymakers should prioritize efforts to improve access to healthcare services, particularly among underserved and disabled populations in Punjab, Pakistan.
- Educational interventions aimed at increasing awareness and understanding of genetic disorders should be implemented in schools and communities in Punjab, Pakistan.
- Healthcare providers should offer genetic screening and counseling services to individuals with a family history of genetic disorders in Punjab, Pakistan.
- Continued investment in research and innovation is essential for advancing our understanding of the genetic basis of diseases and developing effective treatments and interventions in Pakistan.
- Collaboration among healthcare professionals, geneticists, public health experts, policymakers, and community stakeholders is essential for developing comprehensive strategies to address genetic disorders in Pakistan.

References

Agerbo, E., Sullivan, P. F., Vilhjalmsson, B. J., Pedersen, C. B., Mors, O., Børglum, A. D., ... & Mortensen, P. B. (2015). Polygenic risk score, parental socioeconomic status, family history of psychiatric disorders, and the risk for schizophrenia: a Danish population-based study and meta-analysis. *JAMA psychiatry*, 72(7), 635-641.

Barr, P. B., Silberg, J., Dick, D. M., & Maes, H. H. (2018). Childhood socioeconomic status and longitudinal patterns of alcohol problems: Variation across etiological pathways in genetic risk. *Social Science & Medicine*, 209, 51-58.

Calling, S., Ohlsson, H., Sundquist, J., Sundquist, K., & Kendler, K. S. (2019). Socioeconomic status and alcohol use disorders across the lifespan: A co-relative control study. *PloS one*, 14(10), e0224127.

Dohrenwend, B. P. (1990). Socioeconomic status (SES) and psychiatric disorders: Are the issues still compelling?. *Social psychiatry and psychiatric epidemiology*, 25(1), 41-47.

Dohrenwend, B. P., Levav, I., Shrout, P. E., Schwartz, S., Naveh, G., Link, B. G., ... & Stueve, A. (1992). Socioeconomic status and psychiatric disorders: the causation-selection issue. *Science*, 255(5047), 946-952.

Durkin, M. S., & Yeargin-Allsopp, M. (2018, October). Socioeconomic status and pediatric neurologic disorders: current evidence. In seminars in pediatric neurology (Vol. 27, pp. 16-25). WB Saunders.

Haw, T., & Henriques, S. (2021). Exploring how mothers of a child with a genetic disorder experience their couple relationship in a low socio-economic setting. *Journal of Genetic Counseling*, 30(3), 885-899.

Katz, J. N. (2006). Lumbar disc disorders and low-back pain: socioeconomic factors and consequences. *JBJS*, 88(suppl_2), 21-24.

Lundborg, P., & Stenberg, A. (2010). Nature, nurture and socioeconomic policy—What can we learn from molecular genetics?. *Economics & Human Biology*, 8(3), 320-330.

Marees, A. T., Smit, D. J., Abdellaoui, A., Nivard, M. G., Van Den Brink, W., Denys, D., ... & Derks, E. M. (2021). Genetic correlates of socioeconomic status influence the pattern of shared heritability across mental health traits. *Nature human behaviour*, 5(8), 1065-1073.

Marees, A. T., Smit, D. J., Ong, J. S., MacGregor, S., An, J., Denys, D., ... & Derks, E. M. (2020). Potential influence of socioeconomic status on genetic correlations between alcohol consumption measures and mental health. *Psychological medicine*, 50(3), 484-498.

Miller, L. L., Scharf, J. M., Mathews, C. A., & Ben-Shlomo, Y. (2014). Tourette syndrome and chronic tic disorder are associated with lower socio-economic status: findings from the Avon Longitudinal Study of Parents and Children cohort. *Developmental Medicine & Child Neurology*, 56(2), 157-163.

Muntaner, C., Eaton, W. W., Miech, R., & O'campo, P. (2004). Socioeconomic position and major mental disorders. *Epidemiologic reviews*, 26(1), 53-62.

Pasamanick, B., Knobloch, H., & Lilienfeld, A. M. (1956). Socioeconomic status and some precursors of neuropsychiatric disorder. *American Journal of Orthopsychiatry*, 26(3), 594.

Riva, V., Marino, C., Giorda, R., Molteni, M., & Nobile, M. (2015). The role of DCDC2 genetic variants and low socioeconomic status in vulnerability to attention problems. *European Child & Adolescent Psychiatry*, 24, 309-318.

Rushing, W. A., & Ortega, S. T. (1979). Socioeconomic status and mental disorder: New evidence and a sociomedical formulation. *American Journal of Sociology*, 84(5), 1175-1200.

Russell, G., Ford, T., Rosenberg, R., & Kelly, S. (2014). The association of attention deficit hyperactivity disorder with socioeconomic disadvantage: alternative explanations and evidence. *Journal of Child Psychology and Psychiatry*, 55(5), 436-445.

Shashi, V., Keshavan, M., Kaczorowski, J., Schoch, K., Lewandowski, K. E., McConkie-Rosell, A., ... & Kwapil, T. R. (2010). Socioeconomic status and psychological function in children with chromosome 22q11.2 deletion syndrome: implications for genetic counseling. *Journal of genetic counseling*, 19, 535-544.

Ye, J., Wen, Y., Sun, X., Chu, X., Li, P., Cheng, B., ... & Zhang, F. (2021). Socioeconomic deprivation index is associated with psychiatric disorders: an observational and genome-wide gene-by-environment interaction analysis in the UK Biobank cohort. *Biological psychiatry*, 89(9), 888-895.

Комплексное, Р., & Заболевание, Г. (2015). Autism spectrum disorder-a complex genetic disorder. *Folia medica*, 57(1), 19-28.