

Bioinformatics Analysis of Genetic Mutations Associated with Sleep Disorders and Stress in High School Students

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1 Abstract

As many students suffer deeply from stress and sleep disorders in their high school years, we must be aware of the effects on young adults. So, this research paper aims to explore the relationship between sleep and stress and their impact on genetic mutations. This research is conducted by reviewing prior papers about the effects of sleep disorders and genetic mutations on the human body. We then surveyed 150 participants aged between 15 and 25, representing various regions worldwide to avoid environmental bias. The findings indicate that many students who suffer from both stress and sleep disorders are afflicted by one or more types of diseases, such as vision disorders and hypertension. Additionally, they may experience genital diseases like erectile dysfunction and infertility. However, our discussion reveals that the combined effect of sleep disorders and stress can affect the DNA repair process and may also cause oxidative stress, which damages DNA. In conclusion, stress and sleep disorders can lead to mutations in many genes, including the SIM1 gene, and hemoglobin beta gene (HBB0), or affect chromosome 3p25-26, and others. These findings provide a gateway for further research to identify potential cures for these diseases or ways to prevent their occurrence.

2 Introduction

2.1 Exploring the Underlying Factors of Stress

The transition to a new education system can be difficult for freshman high school students, as some of them experience stress, which can hurt their health [1]. A variety of factors, such as academic pressure, extracurricular activities, and social relationships, can engender stress [2]. The digital age has ushered in a new dimension of stressors, including cyberbullying, constant connectivity, and the pressure to present a curated online persona. The lack of adequate sleep [3], owing to academic demands and the allure of screens, can also exacerbate. Social factors also play a pivotal role. Adolescence is a time of identity exploration and forming peer relationships. Social pressures, the desire to fit in, and fear of rejection can intensify stress levels. Moreover, high school students are at a stage of transition, where they grapple with uncertainties about their future, col-

lege applications, and career choices. This uncertainty can amplify stress as they are expected to make life-altering decisions.

As shown in [4], The high school education system differs in countries like Egypt, Syria, and Jordan, particularly in the senior year (grade 12). Despite their academic history, because of this year, a student's major is determined, resulting in high pressure for students and consequently leading to stress. Additionally, parents contribute to the high stress levels of students as they insist on achieving high marks.

2.2 Unraveling the Hazards of Sleep Disorders

On the other hand, sleep disorders can be incurred by staying up late to study, completing tasks, and using social media. Additionally, Sleep disorders in older adults can be caused by a variety of factors, including stress, medical conditions, and medications [5]. They can also be caused by feeling unsafe, especially for students in boarding schools. Also in study [6] shows that 60 percent of junior high school students reported sleep problems, including difficulty falling asleep, staying asleep, and waking up early. In other studies Sleep disturbances are a hallmark of PTSD, affecting up to 80 percent of patients. They can be caused by a variety of factors, including nightmares, anxiety, and hyperarousal [7]. This study [8] shows the association between borderline personality disorder (BPD) and chronic sleep disturbances. BPD The prevalence of chronic sleep disturbances in people with borderline personality disorder (BPD) is estimated to percent between 50 and 80 percent [9] linked to difficulty initiating sleep, maintaining sleep, and waking earlier than desired. BPD symptoms combined with sleep problems predict higher levels of impairment. Assessing and improving sleep in BPD patients may enhance treatment outcomes. Furthermore, a sleep disorder may significantly impact functioning and well-being for teenagers as mentioned in [10].

2.3 The Dangerous Nexus of Stress and Sleep Disorders

Imagine the mind as a symphony conductor, orchestrating a complex harmony of cognitive functions. In the presence of sleep disorders and chronic stress, this maestro is besieged. Memory, once an agile virtuoso, stumbles, forgetting its lines. Concentration, a steady hand on the baton, falters,

causing discord in cognitive processes. Decision-making, the compass guiding daily choices, loses its way, disrupting the rhythm of life [11]. Thus, cognitive faculties fray under the duress of these afflictions, diminishing the quality of intellectual engagement and impairing performance.

The prevailing stress experienced by high school students can have numerous negative effects on their health and mental well-being. Also, Stress is associated with global health threats such as the coronavirus disease 2019 (COVID-19) as mentioned in [12] This raises concerns, especially considering their ongoing physiological development. These effects can significantly impact cellular and genetic integrity and also impact male reproductive health[13]. In addition, This experiment [14] investigated the harmful impact of sleep disorders on the regularity of the menstrual cycle and the negative effects on female reproductive health.

Scientific investigations have already established a link between sleep disorders and the occurrence of DNA abnormalities, which can result in cellular deformities leading to various disorders, such as defects of mutant DNMT1 resulting in neurological disorders[15]. One other specific example is the MTHFR C677T gene, which has been strongly associated with an increased vulnerability to bipolar disorder[16]. This mental health condition is characterized by extreme mood swings, including periods of euphoria (mania or hypomania) and periods of sadness (depression).

The intertwining of sleep disorders and chronic stress presents a formidable alliance, replete with an array of perils that significantly imperil both the physical and mental domains of human well-being. This intricate interplay unfolds a narrative of adversity and underscores the urgency of comprehensive intervention [17].

The toll, however, extends beyond the realm of intellect. Emotions, the hues that color our existence, undergo a transformation. Like a turbulent tempest, mood swings sweep through, leaving behind an emotional landscape marred by irritability and anxiety. The fabric of relationships is strained as the emotional tempest engulfs personal interactions. Furthermore, the correlation between sleep disorders, chronic stress, and mental

health disorders – the unholy trinity – amplifies the emotional maelstrom [18]. The once-subtle hues of joy are obscured, overshadowed by the looming clouds of despondency and apprehension.

Diving deeper, one uncovers the cardiovascular canvas daubed with the hues of vulnerability. The heart, a resilient metronome, finds itself ensnared by irregular rhythms. Sleep disorders and chronic stress tighten the grip [19], elevating blood pressure and nurturing a fertile ground for cardiovascular ailment [20]. The orchestra of arteries and veins, once in harmonious synchrony, plays a discordant tune, escalating the risk of heart disease and stroke. The narrative of bodily harm is not limited to the cardiovascular overture – it cascades into the terrain of the immune system. A weakened defense mechanism is collateral damage, rendering the body susceptible to infections, impairing healing, and prolonging recovery.

Perchance one might think these afflictions remain within the domains of the abstract, the repercussions starkly materialize. The labyrinth of sleep disorders and chronic stress intersects with the highway of accidents. Sleep deprivation, akin to a fogged windshield, blurs the driver's vision of safety, increasing the probability of accidents. Stress, the phantom co-passenger, compromises reaction times, reducing the ability to avert peril. The outcome – a sobering reality where the roadways become theaters of preventable tragedies.

Embarking on a visceral expedition, one confronts the physical manifestations. Muscles, once supple, are now tense and constricted, responding to the relentless demands of stress. The head, home to cognition, becomes a crucible of pain, yielding headaches and migraines as a testament to the relentless grip of these afflictions. Gastrointestinal rhythms are disrupted, stirring a tempest within, as stress and sleep disorders converge to exacerbate conditions such as irritable bowel syndrome and acid reflux.

In culmination, the narrative of sleep disorders and chronic stress forms a tapestry where each thread is interwoven with the next, forming a fabric of multi-dimensional turmoil. The body's systems, once an orchestra harmonizing in health, now perform discordant symphonies under the ba-

ton of adversity. This narrative is not one of doom but of the imperative for proactive intervention. Strategies encompassing medical treatments, lifestyle modifications, and psychotherapeutic interventions are the tools at our disposal to transform this cacophonous narrative into a melody of resilience and well-being [21]. The journey towards mitigation is as intricate as the afflictions themselves, demanding the alliance of healthcare professionals, researchers, and societal stakeholders.

2.4 The Objectives

This scholarly research aims to elucidate the question: "How does the intricate interplay between sleep disorders and stress engender genetic mutations within high school students?" By collecting data, we will explore the relationship between stress, sleep disorders, cellular deformities, altered gene expression, and the potential occurrence of harmful diseases not present in the family history among high school students.

3 literature review

This section aims to provide a comprehensive overview of the literature relevant to this research paper. It will summarize the detrimental impacts of sleep disorders and stress, as well as the potential role of genetic mutations. Each paragraph in **Subsections 3.2**, and **3.1** will reference a specific paper and present a concise summary of its findings. Additionally, **subsection 3.3** will highlight the unique contributions of our work.

To conduct the literature search, Google Scholar was employed as the search engine. The keywords "stress," "mutation," and "sleep disorder" were utilized to retrieve relevant publications. The identified papers were then categorized based on their relevance, with the least relevant paper listed first and the most relevant paper listed last.

3.1 The Intricate Effects of Stress

In [22], the influence of stress, on the process of self-assessment and peer assessment in higher education, is exposed. The study highlights the significant impact of stress on assessment outcomes. It suggests that stress

can impair students' abilities to accurately assess their own work or evaluate their peers' work effectively. This emphasizes the detrimental effect of stress on the assessment process in higher education.

The study [23] examines the effects of stress on behavior related to metabolism and energy balance. The study highlights that stress triggers a cascade of physiological responses that disrupt normal metabolic regulation, leading to food intake, energy expenditure, and body weight alterations. Chronic stress can contribute to overeating, particularly high-calorie and palatable foods, and promote the development of obesity. Additionally, stress can impair physical activity and reduce energy expenditure, further exacerbating weight gain. These findings underscore the significant impact of stress on behavior, particularly in relation to eating habits and energy regulation, with potential implications for the development and management of obesity.

This study [24] examines the impact of stress on the human body, discussing how both intrinsic and extrinsic stimuli can trigger stress responses. Stress can lead to various effects on the body, ranging from disturbances in homeostasis to severe consequences and even mortality. The pathophysiological complications of diseases are often linked to stress, and individuals exposed to stressful environments are at a higher risk of developing disorders. Stress can act as a trigger or exacerbating factor for many diseases and pathological conditions. The study provides a review of the significant effects of stress on the primary physiological systems in humans.

This study [25] indicates that Stress hormones, such as cortisol, can have a negative impact on eating disorders. Cortisol can increase appetite and cravings for unhealthy foods, leading to weight gain. Stress can also trigger binge eating episodes and make it difficult to control food intake. People with eating disorders are often under a lot of stress, which can make their symptoms worse. Treatment for eating disorders should address the underlying stress in addition to the symptoms of the disorder.

In [26] Foster (2005) delves into the connection between stress responses and genetic variability in bacteria. The article explores how stress trig-

gers adaptive genetic changes, influencing bacterial evolution. Foster discusses the role of stress-induced mutagenesis in shaping genetic diversity and the potential consequences for bacterial populations. By investigating stress-related genetic variation, the study enhances our understanding of bacterial adaptation mechanisms and their implications for evolution and survival.

The study [27] investigates the consequences of receiving genetic mutation results for women with an elevated risk of hereditary breast cancer. The study delves into both short-term and enduring effects. It reveals that learning about genetic mutations induces heightened emotional distress and anxiety among affected individuals, underlining the significant psychological impact. Additionally, the research highlights that these effects persist over the long term, emphasizing the enduring psychological burden carried by those at risk. This study underscores the intricate interplay between stress and genetic mutations, spotlighting the need for comprehensive psychological support to mitigate the potentially profound and lasting effects of such revelations on mental well-being.

the study [28] shows how Stress-induced mutation is a significant aspect of cancer development, as highlighted by Cisneros et al. (2017). This ancient genetic phenomenon establishes stress as a key factor leading to genetic mutations in cancer. The study emphasizes how stress contributes to mutations that drive cancer progression, shedding light on the intricate relationship between stress and genetic alterations. This insight underscores the importance of understanding stress-induced mutagenesis in cancer research and treatment strategies.

In [29] investigates the potential genetic markers and mutations associated with depression and anxiety. It conducted a systematic review in the Journal of Affective Disorders. The research explores the link between stress and genetic mutations contributing to depression and anxiety. While analyzing a range of genetic factors, the study highlights the complex relationship between stress and genetic mutations that may influence the development of these mental health conditions. The systematic review emphasizes the need for further investigation into the underlying genetic

mechanisms of depression and anxiety, shedding light on the intricate interplay between genetics and stress in their etiology. This is different from our research, as our study targets high school students

The study by Cui et al [30] delves into the relationship between stress and genetic mutations in various Charcot-Marie-Tooth (CMT2) neuropathies. The research reveals that abnormal interactions of G3BP in stress granules are associated with diverse CMT2 neuropathies. This implies that stress-induced disruptions in cellular processes can lead to genetic mutations contributing to neuropathies. The findings underscore the intricate interplay between stress responses and genetic integrity. Our paper diverges from this perspective, as its focal point lies in examining the impact specifically on high school students, rather than a broad analysis encompassing the general population.

This study [31] explores the psychological well-being of individuals carrying BRCA1/2 mutations, focusing on their experiences of distress, anxiety, and depression. The study systematically reviews the literature and finds that these mutation carriers often face elevated levels of psychological distress, stemming from the genetic mutation's association with an increased risk of cancer. The research underscores the intricate link between stress and genetic mutations, highlighting the need for comprehensive psychological support for affected individuals due to the potential psychological impact of these mutations. Our paper diverges from this perspective, as its focal point lies in examining the impact specifically on high school students, rather than a broad analysis encompassing the general population, and our paper will identify the mechanisms by which gene expression and DNA translocation can lead to mutations as a result of stress.

In the study [32] examines the genetic underpinnings of anxiety disorders, particularly focusing on gender-associated genetic mutation. Investigating anxiety's impact on genetics, the research highlights potential links between anxiety and gender-specific mutation rates. Through comprehensive analysis, the authors elucidate mechanisms influencing such mutations. This work advances insights into anxiety's role in shaping ge-

netic stability and variability, paving the way for a deeper exploration of psychological effects on genetic traits. Our paper stands distinct, diverging from it, as our focus lies on examining the impact of stress and other forms of genetic mutation, such as CTSH, LEPREL1, and others.

3.2 Sleep Disorders' Impacts

The study [33] examined the relationship between homework and sleep disorders in elementary-aged children. Surveys were conducted with students, parents, and teachers to understand their perceptions of homework and its impact on sleep habits. Preliminary findings suggest that homework has a modest impact on sleep, with 36.8 percent of surveyed children reporting getting less sleep sometimes. The study also revealed that homework interferes with family time and creates a power struggle between parents and children. These findings emphasize the importance of considering the potential negative effects of homework on sleep and the need for balanced homework policies that prioritize children's well-being.

The study [34] investigates the impact of sleep disturbances on behavioral issues in preschool children with autism spectrum disorder (ASD). The findings suggest a significant association between sleep disturbances and behavioral problems in this population. Sleep problems contribute to increased hyperactivity, impulsivity, aggression, and social withdrawal. These results highlight the detrimental effect of sleep disturbances on behavior in preschool children with ASD, emphasizing the importance of addressing sleep issues to improve overall behavioral outcomes.

The Study [35] explores the relationship between sleep disorders and eating disorders. The study highlights that sleep disturbances, such as insomnia or sleep apnea, are associated with an increased risk of developing eating disorders, including binge eating disorder and night eating syndrome. Sleep problems can disrupt appetite regulation, alter hormone levels, and influence food choices, leading to disordered eating behaviors. Conversely, eating disorders can also contribute to sleep disturbances. This research emphasizes the bidirectional relationship between sleep disorders and eating disorders, suggesting the need for comprehensive treatment strategies that address both aspects to improve overall outcomes.

The impact of sleep disorders on genetics is explored in the study [36] by Pont-Sunyer et al. (2015), specifically focusing on parkinsonian and nonparkinsonian LRRK2 mutation carriers. The research investigates sleep disorders' prevalence and effects in these individuals, shedding light on the relationship between genetics and sleep disruptions. The study, conducted on a diverse group of individuals, highlights the age-related aspects by examining sleep disorders in LRRK2 mutation carriers. This insight into the genetic basis of sleep disorders underscores the importance of understanding genetic factors that contribute to sleep disturbance. This differs from our paper, as our study focuses on the correlation between sleep disorders and stress in inducing genetic mutations. Additionally, our research specifically targets high school students.

In the study [37]. Their research delves into the intricate relationship between sleep problems, neurobiological changes, core symptoms of autism spectrum disorder (ASD), and psychiatric comorbidities. The study emphasizes how sleep disturbances can influence neurobiological alterations that may contribute to genetic mutations. Notably, the research focuses on individuals with ASD, highlighting a young group (under 12 years old) where sleep-related genetic implications are investigated. This underscores the significance of understanding sleep-related factors in influencing genetic mutations, particularly within the context of ASD and specific age ranges. This distinction sets our paper apart, as it not only concentrates on individuals with ASD but also encompasses all individuals within the high school age range.

The study [38] explores the genetic aspects of insomnia and the potential involvement of epigenetic mechanisms. It discusses how sleep disorders, like insomnia, might impact genetic mutations through epigenetic processes. The study suggests that disrupted sleep patterns and insomnia could potentially influence genetic mutation formation via epigenetic modifications, thereby providing insights into the intricate relationship between sleep disorders and genetic mutation susceptibility. Our paper distinguishes itself by centering its focus on the intricate interplay between sleep and stress, with a specific emphasis on the context of high school

students

The study [39] explores the influence of disrupted sleep on the expression of metabolism-associated genes controlled by the Per1 and Per2 clock genes. The study reveals that sleep disruption impacts the regulation of these genes, potentially contributing to metabolic dysregulation. This underscores the link between sleep disorders and genetic mutations, suggesting that sleep disturbances could potentially influence the formation of genetic mutations and contribute to broader genomic instability. Our paper stands distinct, diverging from it, as our focus lies on examining the impact of stress and other forms of genetic mutation, such as CTSH, LEPREL1, and others.

In [40], The research discusses how disrupted sleep patterns might exacerbate cellular stress, impacting DNA repair mechanisms and increasing the risk of mutations. Additionally, the article highlights that sleep disorders may further exacerbate oxidative stress and impair the body's ability to manage DNA damage, potentially leading to genetic mutations over time. By exploring the intricate interplay between sleep disorders and genetic mutations in the context of primary mitochondrial diseases, the study emphasizes the need to consider sleep quality and its potential impact on genetic stability. Our paper diverges from this perspective, as its focal point lies in examining the impact specifically on high school students, rather than a broad analysis encompassing the general population, and our paper will identify the mechanisms by which gene expression and DNA translocation can lead to mutations as a result of stress.

3.3 Unveiling New Insights: Our Contributions

Our paper will address the paucity of literature on the deleterious effects of the interplay between sleep disturbances and stress on the incidence of genetic mutations. While there have been numerous studies on this topic (**subsection 3.1 and 3.2**), our research paper will make the following contributions:

- We will focus on a limited population of subjects, namely high school students.

- We will employ computational biology to analyze data and suggest potential therapeutic interventions.
- We will focus on the nexus between sleep disorders and stress as both agents in the etiology of genetic mutations.
- We will identify the mechanisms by which gene expression and DNA translocation can lead to mutations as a result of sleep disorders and stress.

To the best of our knowledge, our contributions are unique and add new knowledge to the literature. This is supported by the review conducted in **subsections 3.1 and 3.2**

4 Methodology

4.1 Unveiling the Study Design and Participant Selection

This study employs a mixed methods research design, integrating quantitative and qualitative data collection and analysis techniques. The research approach adopted is predominantly quantitative, aimed at measuring and analyzing numerical data to establish statistical relationships and patterns. However, qualitative data will also be gathered to provide additional insights and contextual understanding.

In subsection 4.2, the data collection method will be scrutinized, as we utilized Google Forms to administer a comprehensive survey with multiple questions. The survey aimed to explore the relationship between sleep hours, stress levels, and sleep disorders during high school period. The objective was to investigate potential connections between stress and sleep disorders, which may contribute to the development of genetic mutations. These endeavors enable us to draw more robust conclusions regarding their effectiveness.

Furthermore, data analysis was conducted using the Python programming language, as discussed **in subsection 4.3**, to ensure high accuracy. The Matplotlib library was utilized to facilitate detailed visualization and interpretation of the findings.

Moreover, a stratified random sampling technique was employed to select participants from the target population, ensuring representation from diverse demographic groups. The target group of this study comprises young adults aged 15-26, who can provide insights about their experiences during the high school period. The sample size of 150 individuals was determined through power analysis calculations.

Participants were included in the study if they met specific criteria, such as falling within the specified age range, having experienced the high school period, and having no prior history of diseases. Exclusion criteria encompassed individuals with pre-existing medical conditions that could potentially influence the study outcomes.

To enhance the representativeness of the sample, participants were recruited from various countries, including Egypt, Latvia, Syria, Saudi Arabia, Latin American countries, and Eastern Asian regions, although the majority were from Egypt. Extensive efforts were made to reach out to high school students from different countries through engagement in social media platforms such as Discord and Telegram. Despite these endeavors to ensure a representative sample across different age groups, there may be a potential bias towards individuals aged between 15 and 17.

Additionally, prior to the main study, valuable feedback from senior students was collected and utilized to refine the study procedures and make necessary adjustments to the study design and participant selection process.

4.2 Data collection

Firstly, we engaged in an extensive literature review, delving into books that encompassed the topic of genetic mutation, such as "Mutants: On Genetic Variety and the Human Body"[41] and "Principles of Genetics." [42] This endeavor aimed to foster a profound understanding of genetics and mutation. Subsequently, we delved into articles and books centered around sleep disorders and stress, including "Why Zebras Don't Get Ulcers." [43] These resources provided us with intricate details concerning stress and

its associated ailments.

To facilitate data collection, we devised a survey that was administered through the online platform Google Forms. This platform streamlined the distribution of the survey link to potential participants and ensured a convenient means of data collection. Each participant received a unique survey link, affording them the flexibility to complete the questionnaire at their preferred pace.

[44]

A comprehensive survey questionnaire was meticulously crafted to extract pertinent data from the participants. It encompassed five sections, comprising multiple-choice questions, Likert scale items, and open-ended queries, thereby capturing a wide spectrum of information. The initial section focused on gathering demographic details, such as age, gender, country, and average sleep hours. Additionally, participants were posed with the question, "During your high school period, have you experienced symptoms of stress or sleep disorder?" This query aimed to discern whether participants encountered sleep disorders, stress, or neither during their high school years. The multiple-choice format furnished five response options: "No" (leading to response submission), "Yes" (indicating the presence of both sleep disorders and stress), "Maybe," "Only stress," and "Only sleep disorder." Selecting any of these four options would direct participants to section 2.

Section 2 comprised a solitary question: "Did sleep disorders and stress during that time affect your health and increase the risk of developing certain diseases (such as hypertension, diabetes, shortsightedness, weight loss, and others), or were there any genetic disorders that may have contributed to your health issues?" Participants were presented with four choices: "No" (leading to response submission), "Yes and maybe" (directing them to section 3), and "I did not visit a doctor or undergo any medical analysis during high school, so I am not aware of whether or not I contracted any diseases" (progressing to section 5). The latter choice aimed to emphasize the importance of detecting potential genetic variations and ailments that participants may have been unaware of, especially if they experienced symptoms suggestive of genetic mutations, such as mood swings, which

may be attributed to mutations in genes like C9ORF72, MAPT, or GRN.

Section 3, prompted by the selection of "Yes and maybe" in section 2, contained inquiries pertaining to the specific health issues encountered during the high school period. This section comprised three questions to further explore the participants' experiences.

The first question was an open-ended query: "During that time, what kinds of diseases did you experience as a result of sleep disorders and stress, and have you recovered from them?" This question aimed to gather detailed information regarding the specific diseases participants may have encountered and whether they have since recovered.

The second question was an optional attachment: "Could you provide medical analysis or a prescription?" Participants had the choice to provide information on any medical analyses or prescriptions they had received during the high school period. This question acknowledged that some participants may not have undergone medical analyses or retained their prescriptions.

The final question in this section was a multiple-choice question: "Is there a history of these diseases in your family, or not?" This question sought to ascertain if the diseases experienced by participants were hereditary. For example, if the disease experienced by a participant is not hereditary but is a genetic disease, it is likely caused by a mutation in specific genes. For instance, if a participant experiences short-sightedness, a condition known to be hereditary, and there are no instances of this condition in the entire family, it suggests that the participant's short-sightedness may be caused by a mutation in genes such as CTSN or GJD2, the Participants were presented with three response options: "No" (leading to response submission), "I don't have awareness of that" (also leading to response submission), and "Yes" (which directed them to section 4).

Section 4, triggered by selecting "Yes" in section 3, aimed to gather more information about the family history of diseases. Participants were asked, "Which members of your family have a history of these diseases, are they on your mother's or father's side of the family?" This query aimed to elicit additional details regarding the presence of the diseases in participants' families and determine whether they were prevalent on the maternal

or paternal side.

Lastly, section 5, triggered by the choice in section 2 indicating a lack of medical analysis or awareness of diseases contracted during high school, consisted of a single question: "Please provide any symptoms that you felt then (e.g., fever, loss of memory, inability to focus and comprehend, depression, anorexia, etc....)." This question sought to ascertain if participants experienced any symptoms suggestive of genetic mutations that they may not have been aware of, such as mood swings, which could be linked to mutations in genes like C9ORF72, MAPT, or GRN.

4.3 Data Analysis

The research paper used Python for data analysis to ensure high accuracy. Matplotlib, known for its advantages in data analysis, was utilized due to its wide range of plot types, customization options, and seamless integration with Pandas. This library enables the creation of publication-quality plots and facilitates interactive exploration. In the **sub-subsection 4.3.1** code description was provided to emphasize the precise sizing of each data point. Additionally, **sub-subsection 4.3.2** displays the corresponding code for reference.

4.3.1 Code Description

The provided code snippet demonstrates a data analysis and visualization workflow using the Python libraries "pandas" and "matplotlib". The code can be divided into several distinct sections, each serving a specific purpose.

Firstly, the code imports the necessary libraries, "pandas" and "matplotlib", using the "import" statements. These libraries are widely used for data manipulation and plotting, respectively.

Next, the code reads data from an external source using the `pd.read_csv()` function. It fetches the data from a CSV file hosted on Google Sheets by providing the corresponding URL. The data is stored in a "pandas" DataFrame object named "df", which allows for efficient data manipulation and analysis.

The code proceeds to analyze the relationship between sleep hours and biological gender. It groups the data by the "biological gender" column and calculates the mean sleep hours using the "mean()" function. The results are stored in the "mean-sleep" variable.

To visualize this relationship, the code employs the "plot()" function on the "mean-sleep" object, specifying the plot type as a bar chart using the "kind='bar'" parameter. This generates a bar plot showing the average sleep hours categorized by biological gender.

Moving on, the code focuses on exploring the relationship between stress/sleep disorder symptoms and biological gender. It modifies the values in the "During your high school period, have you experienced symptoms of stress or sleep disorder" column, collecting the value "Maybe" with "Yes". This ensures consistent categorization.

The code then performs a group-by analysis using the "group by ()" function, grouping the data by biological gender and the presence of stress/sleep disorder symptoms. It counts the occurrences of each category using the "value-counts()" function, resulting in a series object named "stress-resp".

To visualize this relationship, another bar chart is generated using the "plot()" function with the "kind='bar'" and "stacked=True" parameters. This creates a stacked bar chart illustrating the distribution of stress/sleep disorder symptoms based on biological gender.

Similarly, the code explores the relationship between stress/sleep disorder symptoms and the risk of developing certain diseases. It follows a similar procedure as before, modifying the values in the relevant column, conducting a group-by analysis, and creating a stacked bar chart to visualize the results.

The code then examines the connection between disease experiences, family history, and specific diseases. It replaces values in the "Is there a history of these diseases in your family, or not?" column, grouping the data by disease experiences and family history, and counting the occur-

rences using the "count()" function. The results are stored in the "disease counts" variable.

To provide further insights, the code prints the counts of diseases for specific conditions, such as when the answer is "Yes" for disease experiences and "No" for family history, or when both answers are "No".

Additionally, the code creates two data tables: one for diseases and their respective counts and another for symptoms and their counts. It iterates through unique values in the relevant columns, counts their occurrences using the "sum()" function, and appends the results to the respective data tables. The tables are then printed to display the frequencies of diseases and symptoms.

4.3.2 The Data Analysis Code

```
1 import pandas as pd
2 import matplotlib.pyplot as plt
3
4 # Read data
5 df = pd.read_csv('https://docs.google.com/spreadsheets/d/1AkkxM_UP_whb-1
6     xe0NFEV7b7vrZP8y9peoKZNu-9ZvE/export?gid=310367587&format=csv')
7
8 # Relationships
9 mean_sleep = df.groupby(['biological gender'])['What is the average number of
10     sleep hours?'].mean()
11 mean_sleep.plot(kind='bar')
12
13 # Stacked bar chart for stress response
14 df['During your high school period, have you experienced symptoms of stress or
15     sleep disorder'] = df['During your high school period, have you experienced
16     symptoms of stress or sleep disorder'].replace(['Maybe'], 'Yes')
17 stress_resp = df.groupby(['biological gender'])['During your high school period,
18     have you experienced symptoms of stress or sleep disorder'].value_counts()
19 stress_resp.plot(kind='bar', stacked=True)
20
21 # Stacked bar chart for risk response
22 df['During your high school period, have you experienced symptoms of stress or
23     sleep disorder'] = df['During your high school period, have you experienced
24     symptoms of stress or sleep disorder'].replace(['Maybe'], 'Yes')
25 risk_resp = df.groupby(['During your high school period, have you experienced
26     symptoms of stress or sleep disorder'])['Did sleep disorders and stress during
27     that time affect your health and increase the risk of developing certain
28     diseases (such as hypertension, diabetes, shortsightedness, weight loss and
29     others), or were there any genetic disorders that may have contributed to your
30     health issues?'].value_counts()
31 risk_resp.plot(kind='bar', stacked=True)
```

```

21 # Disease experience vs family history
22 df['Is there a history of these diseases in your family, or not?'] = df['Is there
    a history of these diseases in your family, or not?'].replace(['I am not aware
    of that.'], 'No')
23 disease_counts = df.groupby(['During that time, what kinds of diseases did you
    experience as a result of sleep disorders and stress, and have you recovered
    from them? Could you provide medical analysis or a prescription?(optional)', '
    Is there a history of these diseases in your family, or not?'])['country'].
    count()
24 print(disease_counts.loc[('Yes', 'No')])
25 print(disease_counts.loc[('No', 'No')])
26
27 # Disease data table
28 disease_table = pd.DataFrame(columns=['Disease', 'Count'])
29 diseases = df['During that time, what kinds of diseases did you experience as a
    result of sleep disorders and stress, and have you recovered from them? Could
    you provide medical analysis or a prescription?(optional)'].unique()
30 for disease in diseases:
31     count = (df['During that time, what kinds of diseases did you experience as a
        result of sleep disorders and stress, and have you recovered from them?
        Could you provide medical analysis or a prescription?(optional)'] ==
        disease).sum()
32     disease_table = disease_table.append({'Disease': disease, 'Count': count},
        ignore_index=True)
33 print(disease_table)
34
35 # Symptom data table
36 symptom_table = pd.DataFrame(columns=['Symptom', 'Count'])
37 symptoms = df['Please provide any symptoms that you felt then (e.g., fever, loss
    of memory, inability to focus and comprehend, depression, anorexia, ect....)'].
    unique()
38 for symptom in symptoms:
39     count = (df['Please provide any symptoms that you felt then (e.g., fever, loss
        of memory, inability to focus and comprehend, depression, anorexia, ect
        ....)'] == symptom).sum()
40     symptom_table = symptom_table.append({'Symptom': symptom, 'Count': Here's the
        continuation of the code:
41
42     '''tex
43     count}, ignore_index=True)
44 symptom_table = symptom_table.sort_values('Count', ascending=False)
45 print(symptom_table)

```

4.4 Ethical Reflections and Study Constraints

4.4.1 Ethical Considerations:

Participants were given a detailed information sheet explaining the purpose of the study, the procedure, and potential risks. A voluntary participation form indicating informed consent was required to be signed. For example, participants were provided with an informed consent form explaining the purpose of the study to examine the relationship between hours of sleep, stress levels, and genetic variability .

Various procedures were used to protect the privacy of the participants and ensure the security of the data. All collected data were assigned a unique identifier and stored in a password-protected database accessible only to authorized researchers. For example, participants' names and personal information were replaced with unique letters and numbers to ensure anonymity

The study protocol and ethical considerations underwent rigorous review by the STEM October School's Research Review Board, which meticulously assessed the study design, participant recruitment methods, and data handling procedures to ensure strict adherence to ethical guidelines. Notably, the study obtained approval from the aforementioned board prior to the commencement of data collection, thus safeguarding the rights and welfare of the participants.

4.4.2 Study constraints

Owing to limited resources and time constraints, the designated period for data collection was confined to a mere two weeks. This predicament imposed inherent limitations regarding the recruitment of participants and the extent of data analysis that could be undertaken. For instance, the administration of the survey and subsequent follow-up responses were subject to time constraints within the designated timeframe.

Despite concerted efforts to secure participants from diverse countries, the ultimate sample size was restricted to a modest 150 individuals, falling within the age range of 15-26. To illustrate, the original intention was to recruit 500 participants; however, due to inherent design constraints, only 150 could be included in the study.

The research team encountered difficulties in enlisting participants from specific demographic groups, particularly individuals hailing from low-income regions. Despite proactive attempts to engage high school students via popular social media platforms like Discord and Telegram, the response rate from these specific groups fell below anticipated levels. For example, reaching out to participants from low-income areas proved arduous, mainly attributable to limited internet accessibility and a dearth of

technical proficiency among the target population.

Furthermore, financial constraints significantly impacted access to advanced tools and equipment. Consequently, researchers were unable to employ sophisticated sleep-monitoring devices or genetic testing methods that could have provided in-depth insights into the intricate connections between stress, sleep disorders, and genetic mutations.

5 Results

Our research endeavors were driven by the intention to gain insights from high school students. Our focus was exclusively on unraveling the intricate relationship between stress, sleep disorders, and genetic mutations. The methodology we employed for data collection revolved around a systematic deployment of a survey instrument, which is thoroughly outlined in the "Data Collection" subsection (4.2). After meticulously assembling the dataset, we conducted our analytical procedures using the Python programming language. The nuances of this process are elaborated upon in the "Data Analysis" subsection (4.3).

In the subsequent discourse, this section illuminates the outcomes of our research efforts. The exposition takes on a multifaceted nature, encompassing descriptive statistics (5.1), hypothesis testing, frequency distribution presentation, and correlation analysis explanation. These intricately interconnected aspects coalesce to present a comprehensive mosaic of findings that have emerged from this study.

5.1 Descriptive Statistics

Participant demographics exhibited variations across numerous countries. The majority consisted of students from Egypt, accounting for 60 percent, followed by 11 percent from Latin American countries. The remaining participants are represented in Figure 1.

The study's initial aim was to gather 500 participants across diverse age groups; however, the final sample consisted of only 150 individuals. Among them, 52 were 15 years old, and 47 were 16 years old. Figure 2 visually represents the distribution of the remaining age groups. The gender was

collected as well which are 50 percent males and 50 percent females

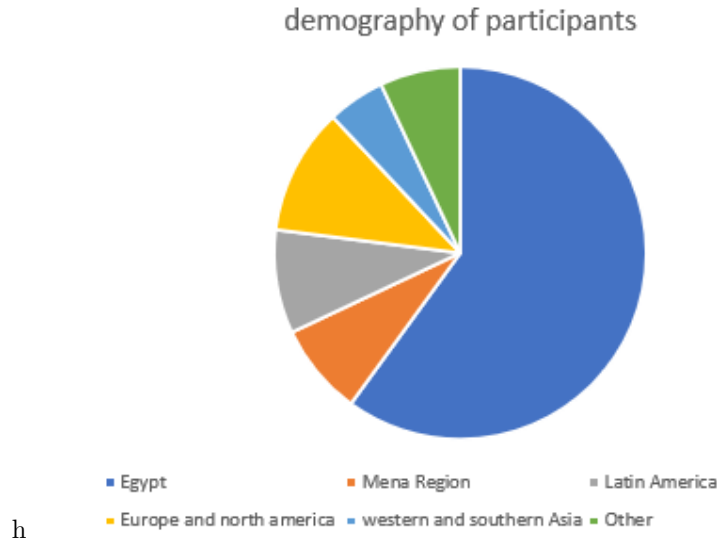


Figure 1: Demography of participants

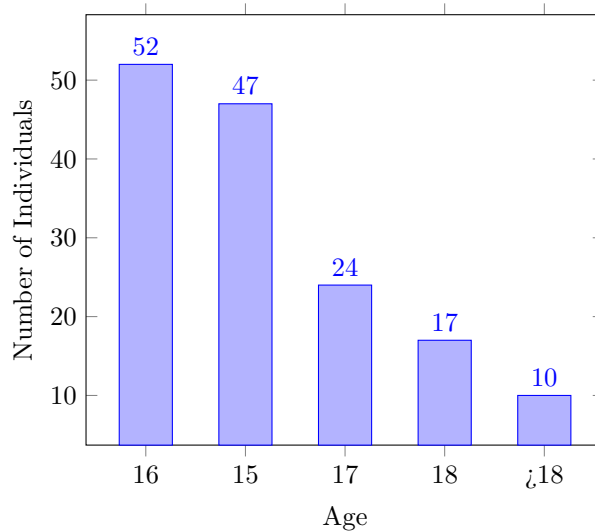


Figure 2: Age Distribution of 150 Individuals

Therefore, the total hours of sleep collected from all participants varied among them. However, the mean sleep duration was 7.2 hours, which is significantly lower compared to the recommended 9 to 9.25 hours for teenagers to maintain healthy sleep [45]. The standard deviation was 2.311.

Afterward, we asked the participants whether they had experienced any

stress or sleep disorders. The responses were as follows: 50 percent said 'yes,' 19 percent said 'maybe,' 14.5 percent said 'no,' and some expressed experiencing either stress or sleep disorders exclusively. Among those who reported experiencing stress, sleep disorders, or both, we posed questions about the potential impact on their health and the risk of developing certain diseases (such as hypertension, diabetes, shortsightedness, weight loss, and others). In response, 27 percent of the participants answered 'yes,' 11 said 'maybe,' and 17 said 'no.' Notably, those who responded 'no' had an average sleep duration ranging from 8.5 to 10 hours, which falls within the normal range for teenagers.

Furthermore, 45 percent of the participants had not visited a doctor, and thus, they might not have been aware of any underlying health issues. Subsequently, we inquired about any symptoms related to health issues, and again, the responses varied: 27 percent said 'yes,' 11 said 'maybe,' and 17 said 'no.' Interestingly, the group that responded 'no' had an average sleep duration ranging from 8.5 to 10 hours, aligning with the recommended sleep duration for teenagers.

Participants who suffered from either stress or sleep disorders, but not both, did not report suffering from any other diseases. Additionally, these individuals experienced symptoms such as inability to focus or weight disorders, but they recovered from these symptoms rapidly.

The findings related to this discussion are illustrated in Figure 3.

5.2 Surveying The Spectrum of Suffering

After collecting data from the group of participants who indicated awareness of their diseases through doctor visits and providing their family history, we have categorized it as follows. Please note that most of the participants have experienced multiple diseases.

In this group, 50 percent experienced depression, despite none of them having a family history of this common condition. Furthermore, only 50 percent of those individuals managed to recover from their depression. For participants suffering from either hypermetropia or myopia, the prevalence was 45 percent, with only 35 percent of them having a family history of these eye conditions. Notably, none of them achieved natural recovery

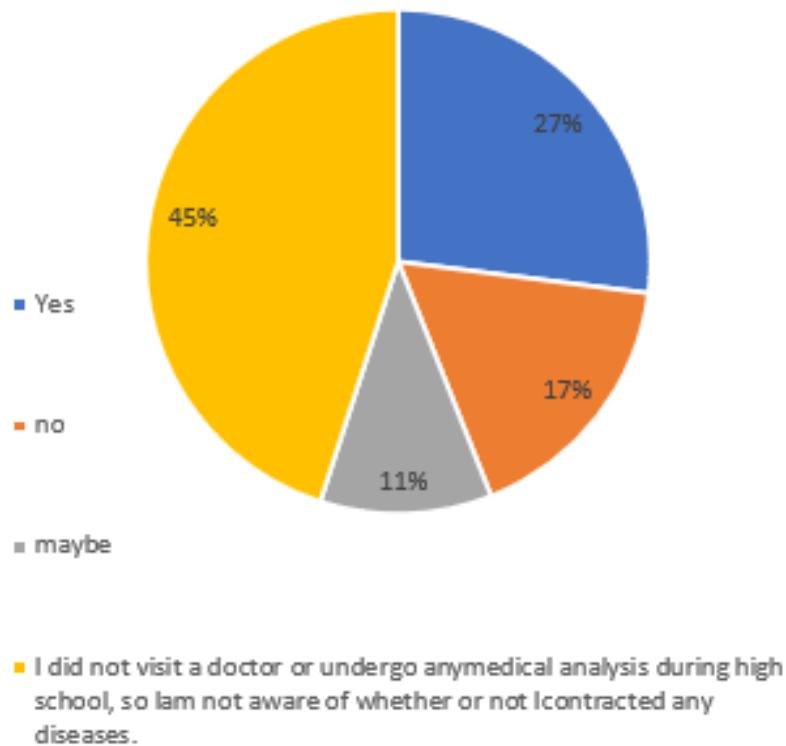


Figure 3: participants who experience sleep disorders or stress

or improvement through glasses alone; some required LASIK surgery.

Additionally, 40 percent of the participants reported mood swings, and 43 percent experienced a loss of their ability to focus. Out of those, only 60 percent eventually recovered, although it took a considerable amount of time and their recovery wasn't as effective as initially. None of these issues were part of their family medical history.

Furthermore, 48 percent of respondents faced weight or eating disorders, none of which had led to recovery, and none were part of their family history. Thirty percent of participants suffered from blood-related issues such as anemia and high blood pressure. Sadly, they didn't experience recovery from these conditions either. Only 5 percent had a family history of these ailments, and merely 3 percent were uncertain about their family's medical history.

Ten percent of the participants grappled with hair loss, and only 30 percent of them had a family history of this condition. Additionally, 20 percent of respondents faced genital diseases such as low sperm count, amenorrhea, dysfunction erectile, and infertility. Regrettably, they hadn't recovered from these conditions either.

Ten percent of participants reported nervous system disorders, and none had experienced recovery from them. Only 30 percent of those individuals had a family history of such disorders, while 10 percent were unsure of their family's medical history.

Lastly, 10 percent of participants dealt with other diseases such as foggy hands, hyperhidrosis, headaches, cardiovascular diseases, and diabetes. None of these conditions were present in their family history, and only 50 percent of those affected managed to recover from them.

5.3 Unveiling the Hidden Symptoms of Undiagnosed Illness

Among those participants who didn't visit a doctor and were unaware of suffering from any diseases, we collected data and categorized them as follows. It's important to note that some participants experienced two or more symptoms:

- Inability to Focus: 70 percent of these participants reported this issue.
- Depression: Fifty percent of them suffered from depression and had not recovered.
- Memory Loss: Thirty percent experienced memory loss.
- Inflammation in Genital Organs: Ten percent of both male and female participants suffered from inflammation in their genital organs.
- Social Anxiety: Thirty percent of these participants reported suffering from social anxiety.

They also experienced other symptoms such as:

- Difficulty gaining or losing weight.

- Loss of appetite.
- Respiratory problems like shortness of breath.
- Persistent fatigue.
- Anorexia.
- Fever-like symptoms.

6 Discussion

6.1 Genetic Influences Of The Results

Sleep disorders and stress can contribute to mutations in two ways:

Firstly, normally, the body has mechanisms to repair DNA damage. However, chronic stress and poor sleep can impair the body's ability to repair DNA lesions efficiently. In general, DNA repair processes may become less effective or slower in cases of prolonged stress and sleep disturbances, leading to a reduction in DNA repair, which, in turn, can cause mutations.

Secondly, sleep disorders and stress can have various negative effects on the body, including hormonal imbalances, increased inflammation, and compromised immune function. They can also increase the body's cortisol levels. Elevated stress hormones can increase oxidative stress, resulting in an imbalance between free radicals and antioxidants in the body. When this balance is disrupted, free radicals can interact with cellular components, including DNA. Oxidative stress can lead to various forms of DNA damage, such as single-strand breaks, double-strand breaks, and chemical modifications. This damage arises from factors like poor sleep patterns and stress, which can induce the production of reactive oxygen species (ROS) and free radicals. Consequently, DNA damage can cause mutations.

For instance, The case of depression is linked to damage to chromosome 3p25-26, affecting 25 percent of the participants. Hypertension results from a mutation in the PDE3A gene, which occurs due to reduced repair ability of that gene. Memory loss can be attributed to variant genes such

as APP, PSEN1, or PSEN2. Weight disorders are caused by the MC4R gene. Anemia can be traced back to mutations in the hemoglobin beta gene (HBB) located on chromosome 11p15.5. Hair loss is a common hereditary condition but can also occur due to a mutation in the androgen receptor gene (AR), affecting 10 percent of the participants. Additionally, the mutations have been found to potentially lead to deficiencies in eye sphericity or lens covering, resulting in either myopia or hyperopia. These conditions may be caused by damage to one of the following genes: CTSH, LEPREL1, SCO2, SLC39A5, ZNF644, 56, and WNT7B.”

When it comes to genital diseases, they can be dependent on genetic disorders. For example, erectile dysfunction is associated with the SIM1 gene. In cases of low sperm count, which can be an indicator of the CFTR gene mutation, a fertility specialist may recommend genetic testing for men. Additionally, a mutation in the Steroidogenic factor-1 (SF-1) gene is a frequent cause of primary amenorrhea in 46, XY female adolescents with low testosterone concentration.

As mentioned previously, some of the genetic conditions identified in the results are hereditary diseases that have occurred due to mutations in the mentioned genes. An unexpected finding in our study is the link between sleep disturbances and stress with the occurrence of genital diseases, especially in young adults in high school.

Another surprising discovery is that participants who suffer from either stress or sleep disorders, but not both, didn't experience significant diseases or even symptoms. They recovered rapidly. This phenomenon can be attributed to the fact that poor sleep quality without stress doesn't significantly affect the body's susceptibility to resulting oxidative stress. Furthermore, stress in combination with good sleep quality can enhance the repair of DNA.

This is due to the Senescence-Associated Secretory Phenotype (SASP), which is associated with senescent or aged cells. When cells enter a senescent state, they release a mixture of molecules, including proinflammatory cytokines, chemokines, growth factors, and proteases. Indeed, the

Senescence-Associated Secretory Phenotype (SASP) process appears to be more active during periods of good-quality sleep. Good sleep quality is associated with improved overall health and cellular repair mechanisms, including the regulation of SASP. During restorative sleep, the body can effectively engage in repair processes, which may include regulating SASP to support tissue repair and immune response.

6.2 Comparison With Previous Studies and Limitation

In comparison with previous studies, our research paper has been able to emphasize the relationship between sleep disorders and stress occurring together, resulting in various types of mutations, as previously mentioned. Additionally, our study has highlighted the processes through which stress and sleep disorders affect the genes of high school students. This contribution to the field of genetics adds new information to the existing literature..

Our study has several limitations. Firstly, the research was conducted over a period of only two months, which may not capture long-term effects accurately. Additionally, due to financial constraints, we were unable to conduct genetic analyses on the participants, limiting our ability to explore genetic factors further.

Furthermore, the logistical challenge of gathering all participants in one place hindered our ability to obtain comprehensive information about their diets and activities. This limitation impacted the depth of our data collection.

Another limitation pertains to the young adult participants, who may have faced challenges in providing accurate information about their health and lifestyle due to their age and relative inexperience.

Moreover, certain legal restrictions in many countries, such as Egypt, prohibit young adults under the age of 21 from undergoing certain analyses like semen analysis. As a result, some participants may have other diseases that they are not aware of, which could affect our findings.

These limitations should be taken into consideration when interpret-

ing the results of future research studies that may benefit from addressing these challenges for a more comprehensive understanding of the topic.

7 Conclusion

In conclusion, this research study aimed to investigate the relationship between sleep disorders, stress, and their impact on the genetic health of high school students. Our findings revealed several noteworthy insights into this complex interplay.

Firstly, we observed that participants who suffered from either stress or sleep disorders, but not both, did not report suffering from any other diseases. Furthermore, these individuals experienced symptoms such as the inability to focus or weight disorders but recovered from these symptoms rapidly. This phenomenon may be attributed to the different ways stress and sleep disorders influence the body's susceptibility to oxidative stress and DNA damage.

Secondly, we categorized a range of diseases among participants who were aware of their conditions through doctor visits and family history. These diseases encompass various genetic factors, including depression, eye conditions, mood swings, weight disorders, blood-related issues, hair loss, genital diseases, nervous system disorders, and other conditions. The presence of these diseases and their genetic underpinnings underscores the complex genetic influences on health.

Additionally, participants who were unaware of their diseases exhibited hidden symptoms, including the inability to focus, depression, memory loss, inflammation in genital organs, and social anxiety. These findings emphasize the importance of regular health check-ups and early disease detection.

Furthermore, our research suggests a relationship between the co-occurrence of sleep disorders and stress and an increased risk of mutations. Chronic stress and sleep disturbances can disrupt DNA repair mechanisms, leading

to a reduction in DNA repair efficiency and an elevated risk of mutations. Notably, the Senescence-Associated Secretory Phenotype (SASP) process may play a role in mitigating these effects, particularly during periods of good-quality sleep.

While this study provides valuable insights into the genetic influences of stress and sleep disorders on health, it has certain limitations, such as its relatively short duration, financial constraints, and challenges in data collection. Future research should aim to address these limitations for a more comprehensive understanding of the topic.

In summary, this research contributes to our understanding of the intricate relationship between stress, sleep disorders, genetics, and health outcomes. It highlights the importance of early disease detection, genetic factors, and the potential for genetic mutations in individuals experiencing these conditions. Further exploration of these factors can lead to improved healthcare strategies and interventions for affected individuals.

We recommend that further researchers strive to avoid challenges and imitations of our research and conduct genetic analysis. Furthermore, our findings are hoped to serve as a gateway for future potential cures or even preventive measures.

References

- [1] T. J. Berndt and D. Mekos, “Adolescents’ perceptions of the stressful and desirable aspects of the transition to junior high school,” *Journal of Research on Adolescence*, vol. 5, no. 1, pp. 123–142, 1995.
- [2] K. Lal, “Academic stress among adolescent in relation to intelligence and demographic factors,” *American International Journal of Research in Humanities, Arts and Social Sciences*, vol. 5, no. 1, pp. 123–129, 2014.
- [3] A. Alhujaili, W. Karwowski, T. T. Wan, and P. Hancock, “Affective and stress consequences of cyberbullying,” *Symmetry*, vol. 12, no. 9, p. 1536, 2020.
- [4] J. Cochran, *Education in Egypt (RLE Egypt)*. Routledge, 2012, vol. 1.
- [5] M. V. Vitiello, “Sleep disorders and aging: understanding the causes,” *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, vol. 52, no. 4, pp. M189–M191, 1997.
- [6] S.-F. Gau and W.-T. Soong, “Sleep problems of junior high school students in taipei,” *Sleep*, vol. 18, no. 8, pp. 667–673, 1995.
- [7] A. Germain, “Sleep disturbances as the hallmark of ptsd: where are we now?” *American Journal of Psychiatry*, vol. 170, no. 4, pp. 372–382, 2013.
- [8] E. A. Selby, “Chronic sleep disturbances and borderline personality disorder symptoms.” *Journal of consulting and clinical psychology*, vol. 81, no. 5, p. 941, 2013.
- [9] J. Vanek, J. Prasko, M. Ociskova, F. Hodny, M. Holubova, K. Minarikova, M. Slepecky, and V. Nesnidal, “Insomnia in patients with borderline personality disorder,” *Nature and Science of Sleep*, pp. 239–250, 2021.
- [10] L. J. Meltzer and J. A. Mindell, “Relationship between child sleep disturbances and maternal sleep, mood, and parenting stress: a pilot study.” *Journal of Family Psychology*, vol. 21, no. 1, p. 67, 2007.

- [11] S. Pabst, M. Brand, and O. T. Wolf, “Stress and decision making: A few minutes make all the difference,” *Behavioural brain research*, vol. 250, pp. 39–45, 2013.
- [12] G. Boyraz and D. N. Legros, “Coronavirus disease (covid-19) and traumatic stress: probable risk factors and correlates of posttraumatic stress disorder,” *Journal of Loss and Trauma*, vol. 25, no. 6-7, pp. 503–522, 2020.
- [13] R. J. Aitken, T. B. Smith, M. S. Jobling, M. A. Baker, and G. N. De Iuliis, “Oxidative stress and male reproductive health,” *Asian Journal of andrology*, vol. 16, no. 1, p. 31, 2014.
- [14] X. Xing, P. Xue, S. X. Li, J. Zhou, and X. Tang, “Sleep disturbance is associated with an increased risk of menstrual problems in female chinese university students,” *Sleep and Breathing*, vol. 24, pp. 1719–1727, 2020.
- [15] J. Baets, X. Duan, Y. Wu, G. Smith, W. W. Seeley, I. Mademan, N. M. McGrath, N. C. Beadell, J. Khoury, M.-V. Botuyan *et al.*, “Defects of mutant dnmt1 are linked to a spectrum of neurological disorders,” *Brain*, vol. 138, no. 4, pp. 845–861, 2015.
- [16] V. Kapoor, N. F. Watson, and L. Ball, “Chronic insomnia in the setting of mthfr polymorphism,” *Journal of Clinical Sleep Medicine*, vol. 18, no. 4, pp. 1215–1218, 2022.
- [17] D. Scotland-Coogan, “Anxiety symptoms and sleep disturbance in veterans with posttraumatic stress disorder: The impact of receiving and training a service dog,” *The Qualitative Report*, vol. 24, no. 10, pp. 2655–2674, 2019.
- [18] K. S. Han, L. Kim, and I. Shim, “Stress and sleep disorder,” *Experimental neurobiology*, vol. 21, no. 4, p. 141, 2012.
- [19] D. J. Brotman, S. H. Golden, and I. S. Wittstein, “The cardiovascular toll of stress,” *The Lancet*, vol. 370, no. 9592, pp. 1089–1100, 2007.
- [20] N. Brand, E. Hanson, and G. Godaert, “Chronic stress affects blood pressure and speed of short-term memory,” *Perceptual and Motor Skills*, vol. 91, no. 1, pp. 291–298, 2000.

- [21] B. A. Farber and L. J. Heifetz, “The satisfactions and stresses of psychotherapeutic work: A factor analytic study.” *Professional Psychology*, vol. 12, no. 5, p. 621, 1981.
- [22] N. K. L. Pope*, “The impact of stress in self-and peer assessment,” *Assessment & evaluation in higher education*, vol. 30, no. 1, pp. 51–63, 2005.
- [23]
- [24] H. Yaribeygi, Y. Panahi, H. Sahraei, T. P. Johnston, and A. Sahebkar, “The impact of stress on body function: A review,” *EXCLI journal*, vol. 16, p. 1057, 2017.
- [25] R. Chami, A. M. Monteleone, J. Treasure, and P. Monteleone, “Stress hormones and eating disorders,” *Molecular and cellular endocrinology*, vol. 497, p. 110349, 2019.
- [26] P. L. Foster, “Stress responses and genetic variation in bacteria,” *Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis*, vol. 569, no. 1-2, pp. 3–11, 2005.
- [27] J. Lim, M. Macluran, M. Price, B. Bennett, and P. Butow, “Short-and long-term impact of receiving genetic mutation results in women at increased risk for hereditary breast cancer,” *Journal of genetic counseling*, vol. 13, pp. 115–133, 2004.
- [28] L. Cisneros, K. J. Bussey, A. J. Orr, M. Miočević, C. H. Lineweaver, and P. Davies, “Ancient genes establish stress-induced mutation as a hallmark of cancer,” *PloS one*, vol. 12, no. 4, p. e0176258, 2017.
- [29] S. F. Lacerda-Pinheiro, R. F. F. P. Junior, M. A. P. de Lima, C. G. L. da Silva, M. d. S. V. dos Santos, A. G. T. Júnior, P. N. L. de Oliveira, K. D. B. Ribeiro, M. L. Rolim-Neto, and B. A. V. Bianco, “Are there depression and anxiety genetic markers and mutations? a systematic review,” *Journal of affective disorders*, vol. 168, pp. 387–398, 2014.
- [30] Q. Cui, H. Bi, Z. Lv, Q. Wu, J. Hua, B. Gu, C. Huo, M. Tang, Y. Chen, C. Chen *et al.*, “Diverse cmt2 neuropathies are linked to aberrant g3bp interactions in stress granules,” *Cell*, vol. 186, no. 4, pp. 803–820.e25, 2023. [Online]. Available: <https://doi.org/10.1016/j.cell.2022.12.046>

- [31] J. Ringwald, C. Wochnowski, K. Bosse, K. E. Giel, N. Schäffeler, S. Zipfel, and M. Teufel, “Psychological distress, anxiety, and depression of cancer-affected brca1/2 mutation carriers: a systematic review,” *Journal of genetic counseling*, vol. 25, no. 5, pp. 880–891, 2016.
- [32] M. G. Gottschalk and K. Domschke, “Novel developments in genetic and epigenetic mechanisms of anxiety,” *Current opinion in psychiatry*, vol. 29, no. 1, pp. 32–38, 2016.
- [33] M. Holland, M. Courtney, J. Vergara, D. McIntyre, S. Nix, A. Marion, and G. Shergill, “Homework and children in grades 3–6: Purpose, policy and non-academic impact,” in *Child & Youth Care Forum*, vol. 50. Springer, 2021, pp. 631–651.
- [34] Y. Wang, J. Lin, Y. Zeng, Y. Liu, Y. Li, K. Xia, J. Zhao, Y. Shen, and J. Ou, “Effects of sleep disturbances on behavioral problems in preschool children with autism spectrum disorder,” *Frontiers in Psychiatry*, vol. 11, p. 559694, 2021.
- [35] K. C. Allison, A. Spaeth, and C. M. Hopkins, “Sleep and eating disorders,” *Current psychiatry reports*, vol. 18, pp. 1–8, 2016.
- [36] C. Pont-Sunyer, A. Iranzo, C. Gaig, A. Fernández-Arcos, D. Vilas, F. Valldeoriola, Y. Compta, R. Fernández-Santiago, M. Fernández, A. Bayés *et al.*, “Sleep disorders in parkinsonian and nonparkinsonian lrrk2 mutation carriers,” *PloS one*, vol. 10, no. 7, p. e0132368, 2015.
- [37] L. Mazzone, V. Postorino, M. Siracusano, A. Riccioni, and P. Curatolo, “The relationship between sleep problems, neurobiological alterations, core symptoms of autism spectrum disorder, and psychiatric comorbidities,” *Journal of clinical medicine*, vol. 7, no. 5, p. 102, 2018.
- [38] L. Palagini, K. Biber, and D. Riemann, “The genetics of insomnia—evidence for epigenetic mechanisms?” *Sleep medicine reviews*, vol. 18, no. 3, pp. 225–235, 2014.
- [39] J. Husse, S. C. Hintze, G. Eichele, H. Lehnert, and H. Oster, “Circadian clock genes per1 and per2 regulate the response of metabolism-

- associated transcripts to sleep disruption,” *PLoS One*, vol. 7, no. 12, p. e52983, 2012.
- [40] R. J. Ramezani and P. W. Stacpoole, “Sleep disorders associated with primary mitochondrial diseases,” *Journal of Clinical Sleep Medicine*, vol. 10, no. 11, pp. 1233–1239, 2014.
- [41] A. M. Leroi, *Mutants: on genetic variety and the human body*. Penguin, 2005.
- [42] D. P. Snustad and M. J. Simmons, *Principles of genetics*. John Wiley & Sons, 2015.
- [43] R. M. Sapolsky, *Why zebras don’t get ulcers: The acclaimed guide to stress, stress-related diseases, and coping*. Holt paperbacks, 2004.
- [44] S. V. Faraone, M. T. Tsuang, and D. W. Tsuang, *Genetics of mental disorders: A guide for students, clinicians, and researchers*. Guilford Press, 1999.
- [45] M. Hirshkowitz, K. Whiton, S. M. Albert, C. Alessi, O. Bruni, L. DonCarlos, N. Hazen, J. Herman, E. S. Katz, L. Kheirandish-Gozal *et al.*, “National sleep foundation’s sleep time duration recommendations: methodology and results summary,” *Sleep health*, vol. 1, no. 1, pp. 40–43, 2015.