RISING RESEARCHERS by MOONPREP

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STUDENT RESEARCH JOURNAL

Introduction of Rising Researchers

This journal edition highlights students who participated in the Rising Researchers virtual research intensive. The course was designed to provide students with an introduction to the scientific method and an opportunity to sharpen their research skills. The core concepts learned include project design, manuscript writing, data analysis, and an introduction to public health and medicine.

Through lectures, hands-on experiments, public electronic health records databases, machine learning algorithms, statistical skills and tools, class and small group discussions, and oral presentations, students explored the computational world with emphasis on human diseases like cardiovascular disease and health equity issues.

The hands-on patient cohort selection and machine learning modeling emphasized the proper use of the scientific method to answer a research question, develop a hypothesis, carry out computational tasks, make observations, analyze, interpret and communicate results.

Research Goals

- Describe and demonstrate the ability to use the scientific method
- Independently construct a research question and design a novel research project
- Demonstrate competency in utilizing research tools and resources and interpreting data to support the research question
- Demonstrate competency to draw reasonable conclusions based on research findings
- Development of a research strategy, including the definition and justification of patient
  inclusion criteria, as well as study hypotheses
- Investigation of existing literature to build reference and resource lists
- Demonstrate the ability to communicate research and results through a manuscript and poster presentation
- Creation of research report for publication submission
- Final research poster presentation and publication in academic research journal

Students Have Mastered The Following Skills And Tasks

- Describe and demonstrate the ability to use the scientific method
- Demonstrate an understanding of research design, including patient selection criteria, generation and justification of hypotheses, and choosing appropriate hypothesis testing tools
- Demonstrate ability to record and interpret data from experiments
- Demonstrate ability to run multiple experiments to evaluate different hypotheses
- Demonstrate the ability to communicate research and results

Academic Team

Meet the Professor: Jedy C

- Jedy C is a professor of Genetics at the University of Massachusetts (UMass) Amherst.
- She received her doctoral degree (Ph.D.) in the Biochemistry and Molecular Biology Department and Plant Biology Program.
- Her research focuses include CRISPR/Cas9 Genome Editing, Genetics and Bioinformatics, Microbiology/bacteria-plant Interactions, and STEM education

Meet the Professor: Loren B

- Loren B is an MD/PhD candidate at the University of North Carolina at Chapel Hill
- Her research focus is on health behaviors and disparities.
- Prior to medical school, Loren was a research assistant at Duke University, where she wrote and published scientific manuscripts and led qualitative and quantitative research studies.

Journal Editor

Meet the Editor: Alexis M

- Alexis Mikhelis is a senior at Livingston High School in New Jersey
- Her research interests include women's health, genetics, prenatal care and hormones.
- Prior to Rising Researchers, Alexis published with the Waksman Institute of Microbiology at Rutgers University as a WSSP scholar. Alexis conducted an authentic research project in molecular biology and bioinformatics.

Rising Researchers Students

The following students successfully participated in the 2023/2024 Rising Researchers Course.

Divya Ariyur

Using Bioinformatics to Investigate Potential Molecular Biomarkers that link Alzheimer's Disease with Type 2 Diabetes

ABSTRACT

The prevalence of Alzheimer's disease (AD) and Type 2 Diabetes (T2D) is increasing worldwide. These diseases both present global health issues with serious impacts on human well-being and considerable effects on the costs of healthcare systems. Although traditionally considered as independent disease conditions, several studies have suggested a connection between AD and T2D. This is because AD and T2D have similarities of shared risk factors, impact on cognitive function or thinking and impact on brain function. A few recent studies have actually shown that having diabetes increases the chances of getting AD**.** However, the exact nature of the connection is still being investigated. Alzherimer's disease is a progressive and irreversible brain disorder that affects memory, thinking, and behavior. It is the most common cause of dementia in older adults, leading to decline in cognitive function and the ability to perform daily tasks. Among the many factors that contribute to the cause of AD, genetic or molecular factors is one of them. Mutation or changes in certain genes is known to increase the risk of developing AD. However, not everyone who has a mutation in any of these genes ends us with AD. It is possible that there are other genes, not yet discovered, that contribute to the cause of AD. On the other hand, Type 2 diabetes is a chronic metabolic disorder characterized by insulin resistance and high blood sugar levels. It often develops in adulthood and is associated with lifestyle factors, genetics, and obesity. Type 2 diabetes mellitus is also linked to problems with cognition or thinking and metabolic issues, which make cells more likely to be affected, thus increasing the chances of developing late-onset Alzheimer's disease as people age. In fact some research has proposed the term "type 3 diabetes" for Alzheimer's disease. While evidence suggests a link between Alzheimer's disease and Type 2

diabetes, the exact nature of the connection is still not established. One way to address this research gap is to identify genes that are shared between AD and T2D. Therefore, this study used several bioinformatics tools and databases, namely, NCBI, GEO, SR Plot, GO and KEGG, to identify genes that are potentially shared between AD and T2D. Results show that genes, SLC19A1, SLC2A2, SERPINA3, REG1A, and HGF from T2D have an association with Alzheimer's. While, genes TFAP2A, HAPLN2, and SCUBE1 from AD have an association with Type 2 diabetes. These eight genes are potential biomarkers that can contribute to the development of treatment methods that can simultaneously target and tackle both Alzherimer's disease and Type 2 Diabetes.

Keywords: Bioinformatics, NCBI, GEO, SR Plot, GO, KEGG, Alzheimer's disease (AD), Type 2 Diabetes (T2D), genes, biomarkers

INTRODUCTION

Incidence of Alzheimer's disease and Type 2 Diabetes

The occurrence of Alzheimer's disease (AD) and Type 2 Diabetes (T2D) is on the rise globally (Moheet et al, 2015; Chung and Lee, 2021). These conditions pose significant global health concerns, affecting human well-being and placing a strain on healthcare systems (Moheet et al, 2015). While historically viewed as separate illnesses, various studies have proposed a link between AD and T2D. They are both associated with aging, where higher rates of occurrences are seen as people grow older. It is estimated that millions of people worldwide are affected by Alzheimer's. In the United States, for example, in 2023, it was estimated that around 6.7 million Americans were living with Alzheimer's disease (Alzheimer's disease facts and figures, 2023). It's more prevalent among women than men. As for T2D, the global incidence is also on the rise, especially among adults. Type 2 Diabetes is closely linked to factors such as aging, unhealthy living habits, and obesity, as noted by Tuomi (2005) and Barbagallo and Dominguez (2014). An estimation made 22 years ago indicated that by 2025, the number of diabetic patients will reach 300 million (International Diabetes Federation, 2001; Adeghate, Schattner, Dunn, 2006).

What is Alzheimer's disease?

Alzheimer's disease is a progressive and irreversible brain disorder that affects memory, thinking, and behavior (Alzheimer's Association. 2023;

Breijyeh and Karaman, 2000; De-Paula et al, 2012). It is the most common cause of dementia in older adults, leading to decline in cognitive or thinking function and the ability to perform daily tasks. Alzheimer's diseases' prevalence increases with age (Breijyeh and Karaman, 2000; De-Paula et al, 2012). AD is characterized by the loss of synapses in many brain regions including the hippocampus and the deposition of abnormal proteins called amyloid beta (Aβ) and tau (Jack and Holtzman, 2013).

Many different factors are known that increase the risk of developing AD. These include genetics or biological molecules, lifestyle and the environment (Breijyeh and Karaman, 2000). Although many different factors have an impact on the development of AD, genes or molecular markers show a strong influence of the risk of developing the disease (Bertram et al, 2003; Bertram and Tanzi, 2004; Sauders et al, 1993). Mutations or changes in certain genes are known to increase the risk of Alzheimer's disease (Breijyeh and Karaman, 2000). For example, changes or mutations in the *UBQLN1* gene on chromosome 9q22 substantially increase the risk of Alzheimer's disease, possibly by influencing alternative splicing of this gene in the brain (Bertram et al, 2003).

Further, a gene called apolipoprotein E (APOE) is the most common gene linked to late-onset Alzheimer's disease. On the other hand, the risk of early-onset Alzheimer's disease is characterized by several genes including, Amyloid precursor protein (APP), Presenilin 1 (PSEN1) and Presenilin 2 (PSEN2) genes (Breijyeh and Karaman, 2000)**.**

However, not everyone who has a mutation in any of these genes ends us with AD. Therefore, the genetic basis of the disease is not yet established and this is a research gap this current study is addressing. Results from this study are important in contributing to the identification of genes or biological markers related to AD and T2D that can serve as potential targets for treatment.

<u>What is Type 2 Disease?</u>

Type 2 diabetes (T2D) is a chronic condition where the body doesn't respond effectively to insulin or doesn't produce enough insulin (Basu, 2017; Barbagallo and Dominguez, 2014; Ye et al, 2023). It's often related to obesity and lifestyle factors and can lead to high blood sugar levels. The

prevalence of Type 2 diabetes is increasing globally, particularly among adults, and is associated with factors like aging, unhealthy lifestyles, and obesity. (Tuomi, 2005; Barbagallo and Dominguez, 2014). Certain populations, including specific ethnic groups, have a higher risk (McQueen, 2023).

Several environmental and genetic factors are known to cause T2D. Environmental factors include being overweight, nutrition/diet, toxins, drugs and not being physically active. Genetic or inheritance factors include insulin resistance and changes in certain genes (Ali, 2013).

On the genetic level, some studies have identified genes that are associated with T2D. Examples include potassium inwardly-rectifying channel, subfamily J, member 11 (*KCNJ11*), Wolfram syndrome 1 (wolframin) (*WFS1*), peroxisome proliferator-activated receptor gamma (*PPARG*), insulin receptor substrate 1 (*IRS1*) and *IRS-2*, , HNF1 homeobox A (*HNF1A*), HNF1 homeobox B (*HNF1B*) and *HNF4A* (Ali, 2013). For most of these genes, their function or mechanism of action remain unclear. And it is possible that other genes, yet to be identified, are linked with T2D.

Potential link between Alzheimer's disease and Type 2 Diabetes

Evidence suggests a link between Alzheimer's disease and Type 2 diabetes due to shared risk factors, impact on cognitive function or thinking and impact on brain function (Moran et al, 2013; Moheet et al, 2015; Miles and Roof, 1922; Biessels, 2006; Rojas et al, 2021). But the exact nature of the connection is still under investigation. So far, studies have shown that diabetes has an impact on cognitive function and brain structure (Moheet et al, 2015). In fact, diabetes has been known to have an effect on the brain for more than one hundred years. For example, one study showed that people with diabetes performed poorly on cognitive tasks examining memory and attention (Miles and Roof, 1922). Another study used microarray data to identify genes that are differentially expressed (DEGs) to determine the potential pathophysiological mechanisms that are shared between AD and T2D (Ye et al, 2023).

Furthermore, recent studies show that having diabetes increases the chances of getting AD (Baglietto-Varga et al, 2016). In addition, diabetes and Alzheimer's share multiple neuropathological mechanisms and

anti-diabetic compounds are a promising therapeutic approach for AD patients (Baglietto-Varga et al, 2016). Therefore, it is important to understand how diabetes and Alzheimer's diseases are connected. It could help find new ways to slow down or stop the onset and progress of Alzheimer's disease (Baglietto-Varga et al, 2016) and diabetes.

One way to understand this connection is to identify genes or molecular biomarkers that are similar between AD and Diabetes. While some genes have been identified that potentially link AD to T2D, it is possible there are more genes yet to be discovered. Therefore, the goal of this current study is to identify novel genes or molecular biomarkers that potentially link AD and T2D. To achieve this, NCBI, GEO, SR Plot, GO and KEGG Bioinformatics tools and databases were used.

Using NCBI and GEO2R, we identified a total of 40 genes that are differentially expressed (DEGs), 20 in AD and 20 in T2D between the controls and the patients. These were further analyzed and validated using SR Plot, KEGG and GO bioinformatics tools to give a final total of 8 genes. Among the 8 genes, 3 from the AD dataset and 5 from the T2D dataset showed the potential to be common between AD and T2D. These eight genes represent potential genetic or molecular biomarkers that may aid in the potential development of treatment strategies capable of addressing both Alzheimer's disease and Type 2 Diabetes at the same time.

METHODS

To investigate potential genetic or molecular biomarkers that are shared between Alzheimer's disease (AD) and Type 2 Diabetes mellitus (T2D), this research used different Bioinformatics tools and databases. The method workflow is shown in Figure 1. First, I navigated through the extensive Gene Expression Omnibus (GEO) bioinformatics tool within NCBI, which contains genetic information contributed by different researchers worldwide (Edgar et al., 2002).

NCBI (National Center for Biotechnology Information) is a U.S. government bioinformatics platform that provides access to various biological and genomic databases and resources. It serves as a key hub for research in the life sciences. On the other hand, GEO is a tool within the NCBI's Gene Expression Omnibus (GEO) database (GEO, 2019) GEO uses algorithms to

analyze and compare gene expression data from RNA sequencing experiments, identifying genes that are differentially expressed between conditions, such as disease vs. control. Therefore, researchers use GEO to explore and extract valuable information from gene expression datasets (GEO Overview – GEO – NCBI, n.d.).

To identify genes or molecular biomarkers that overlap AD and DM, keyword searches were conducted focusing on Alzheimer's disease (AD) and Type 2 Diabetes mellitus (DM). This systematic search led to the discovery of two microarray datasets: GSE97760 and GSE20966. The GSE97760 dataset contains patients with AD ($n = 9$) and healthy controls ($n = 10$) (Naughton et al., 2015). GSE20966 consists of patients with DM ($n = 10$) and healthy controls ($n = 10$) from samples of β-cells obtained from normal pancreases (Zhong et al, 2019).

Identification of Differentially Expressed Genes (DEGs) with GEO2R

GEO2R (GEO2R – GEO – NCBI, n.d.) is an interactive online tool within NCBI used to compare and analyze two datasets collected under the same experimental conditions. It employs GEO query and limma to perform differential expression analysis. In this study, GEO2R was used to investigate differentially expressed genes (DEGs) between T2D and control samples. DEGs are genes that exhibit statistically significant changes in expression levels in response to different experimental conditions (Anjum et al, 2016). These conditions can include various factors like different tissues, treatments, disease states, developmental stages, or environmental stimuli (Anjum et al, 2016). DEGs were considered statistically significant when $|\text{logFC}| \geq 1$ and the P value < 0.05, serving as the cutoff criteria. The probe IDs were converted to gene symbols. Any single probe corresponding to multiple genes was removed. The top 10 upregulated DEGs and 10 downregulated genes were selected for further analysis. This procedure was repeated to investigate DEGs between AD and normal samples (Supplementary data).

SR Plot, Gene Ontology and Pathway Enrichment Analysis of DEGs

SR Plot is an online bioinformatics tool that helps plot and visualize biological data and analysis including genes, genomes, transcriptomics and epigenomics (SRplot- Science and Research Online Plot, n.d.). In addition,

SR Plot can be used to perform Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) pathway enrichment analysis.
GO is a common way to annotate genes, gene products, and sequences as potential biological phenomena, mainly including biological process (BP), cellular component (CC), and molecular function (MF). The biological process describes the major processes, or 'biological programs' that are accomplished by multiple molecular activities. Meanwhile, the cellular component refers to the location of the gene in questions, relative to cellular compartments and structures. The molecular function describes the activities that occur at the molecular level. Kyoto Encyclopedia of Genes and Genomes (KEGG) is a comprehensive database resource for the biological interpretation of genomic sequences and other high-throughput data (Zhu et al., 2020).

In this study, I used NCBI and GEO2R to identify DEGs in AD and T2D. Then GO and KEGG analyses for the top 20 DEGs in each dataset were performed using the SR plot online tool (SRplot- Science and Research Online Plot, n.d.) to analyze the DEGs at the functional level. Finally, I selected the genes that potentially connect AD and T2D for further analysis to determine the potential function of the identified genes.

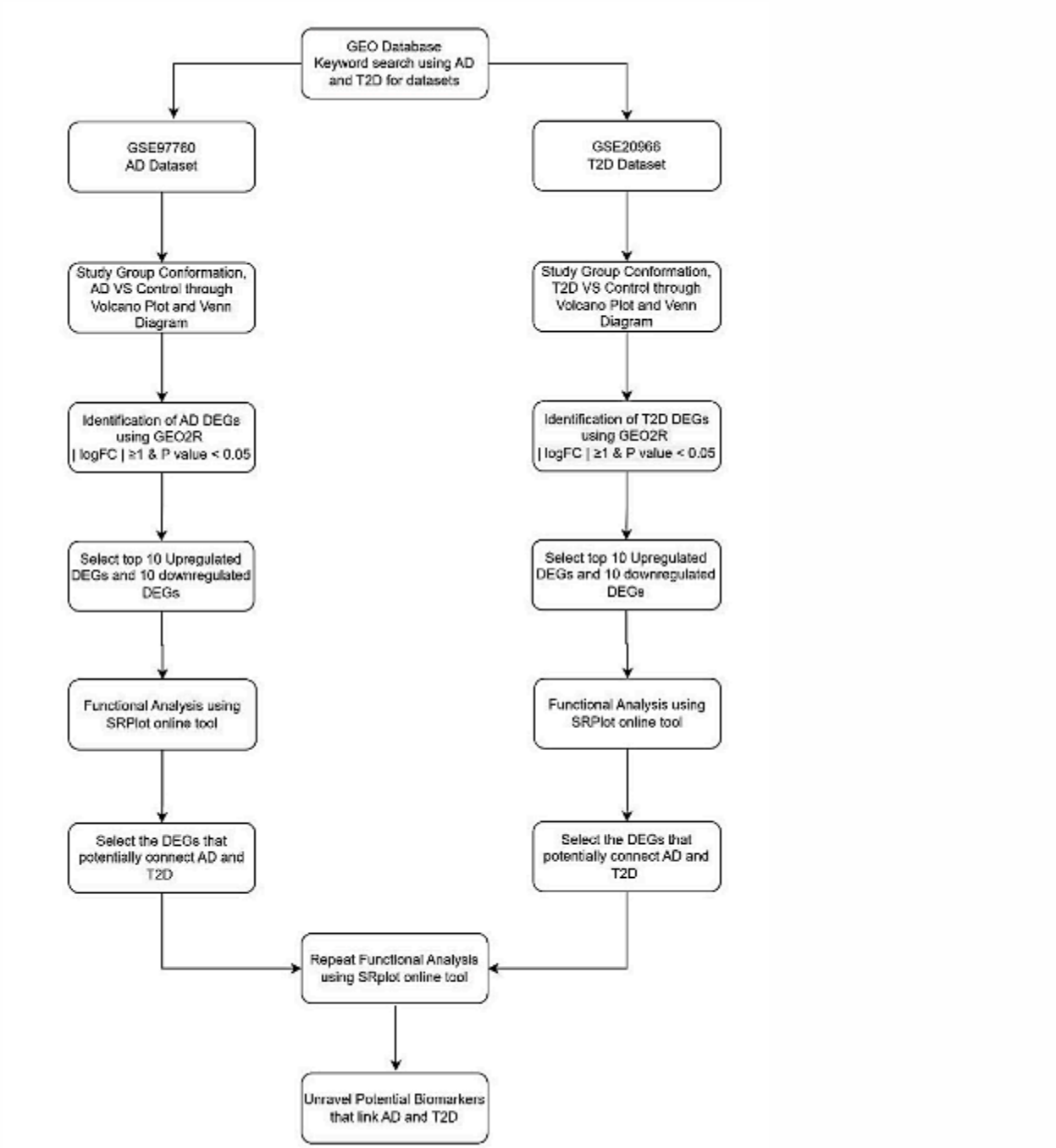

Figure 1: The workflow of the methodology used. NCBI, GEO, SR Plot, GO and KEGG Bioinformatics tools and databases were used . Using NCBI and GEO2R, A total of 40 genes that are differentially expressed (DEGs), 20 in AD and 20 in T2D between the controls and the patients. These were further analyzed and validated using SR Plot, KEGG and GO.

RESULTS

Identification of Differentially Expressed Genes (DEGs)

Differentially expressed genes (DEGs) are genes whose expression levels, typically measured in terms of mRNA or protein abundance using microarray or RNA sequencing experiments, show significant variations between two or more experimental conditions, such as different cell types, tissues, treatments, disease states, or time points (Anjum et al, 2016). DEGs can be either up-regulated (showing increased expression) or down-regulated (showing decreased expression) in one condition compared to another. The identification of DEGs is a fundamental aspect of gene expression analysis and is crucial for understanding the molecular basis of various biological processes and conditions (Anjum et al, 2016).

In this study, the DEGs analyzed were retrieved from microarray experiments conducted by Naughton et al (2015). Specifically, Bioinformatics tool and database GEO2R, housed within NCBI, was used to analyze all the data in the GSE97760 dataset. This dataset contains microarray profiles of blood genome-wide gene expression in female individuals with advanced Alzheimer's Disease and in healthy controls. The top differentially expressed genes (DEGs) in AD patients and healthy control groups were identified (Figure 2). Figure 2a and 2b shows a volcano plot and venn diagram of the differentially expressed genes (DEGs) between AD and control samples. In the volcano plot, up-regulated genes are represented in red, down-regulated genes in blue, and non significant genes in black. These DEGs were further analyzed by selecting the most negative or most positive values of the $|\text{logFC}| \geq 1$ and selecting the genes with statistically significant values ($P < 0.05$). Between AD patients and healthy controls, a total of 20 DEGs, including 10 up-regulated DEGs and 10 down-regulated DEGs, were filtered (Supplementary data).

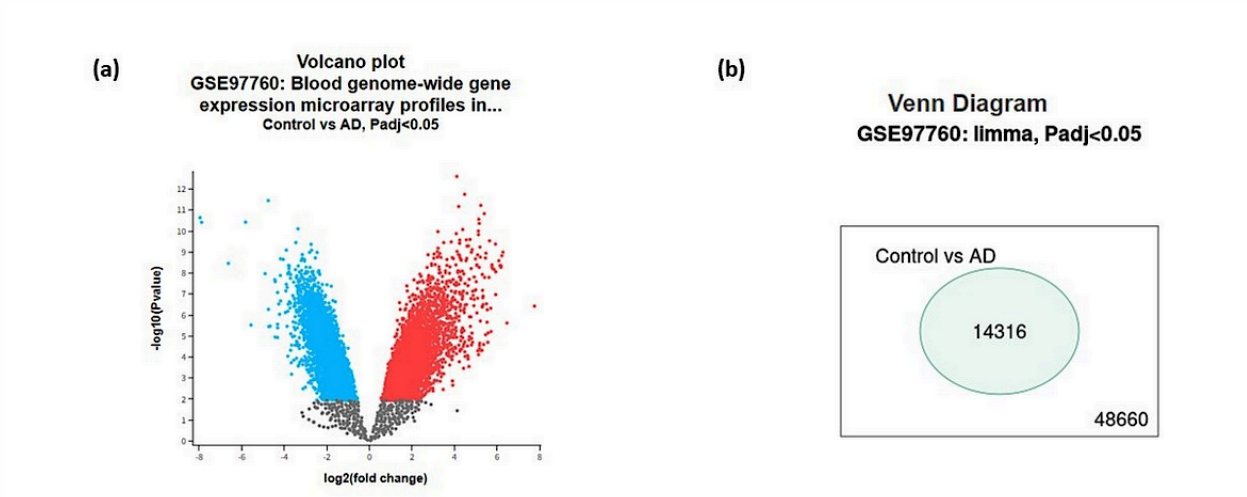

Figure 2: Microarray differential gene analysis in the AD group (a) The Volcano Plot of the genes between AD and controls: red and blue indicate upregulation and downregulation, respectively (**b**) Venn Diagram of AD and control group.

The same process was repeated for the T2D dataset GSE20966, where DEGs were identified (Figure 3). This dataset contained gene expression profiles derived from beta-cell enriched tissue obtained through Laser Capture Microdissection from individuals with type 2 diabetes (Marselli et al, 2010). Using GEO2R Bioinformatics tool, we identified the DEGs from this dataset (Figure 3) and statistical analyses were used to select a total of 20 DEGs, including 10 up-regulated DEGs and 10 down-regulated DEGs, were chosen between T2D and healthy controls (Supplementary data). Figure 3a and 3b shows the volcano plot and venn diagram of the genes between T2D and control samples. The upregulated genes are indicated in red color in the volcano plot, while the down-regulated genes are shown in blue color. Black color in the volcano plot indicates non-significant genes.

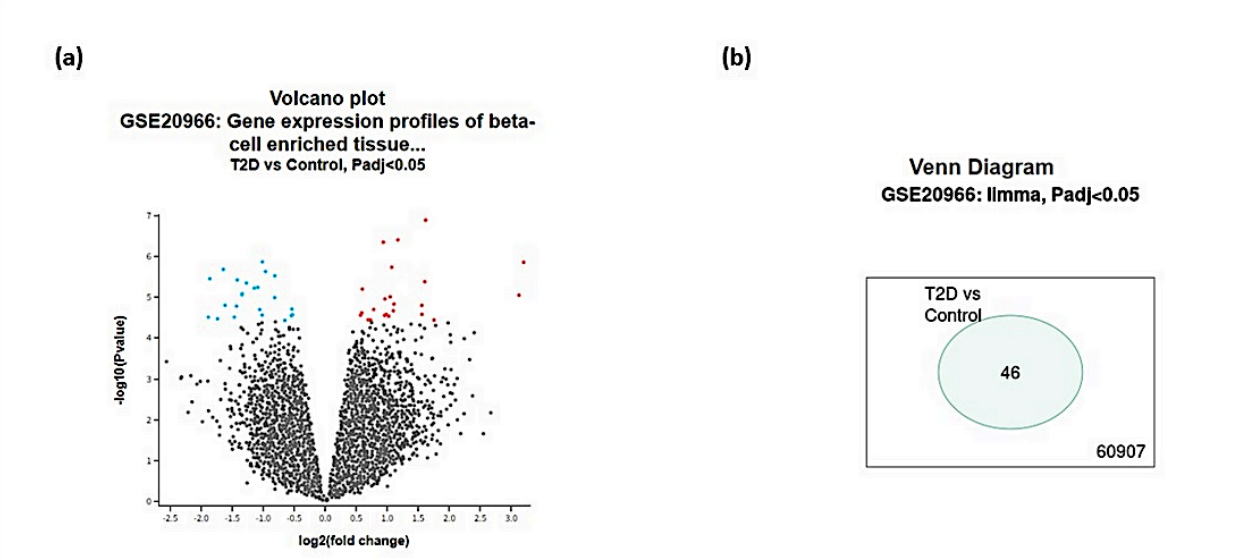

Figure 3: Microarray differential gene analysis in the T2D group (a) The Volcano Plot of the genes between T2D and controls: red and blue indicate upregulation and downregulation, respectively **(b)** Venn Diagram of T2D and control group.

Analysis of the functional features of common DEGs

GO functions and KEGG Pathway enrichment analyses were performed to analyze the biological functions and pathways involved in the shortlisted DEGs from AD and T2D. AD GO analysis results show the three ontologies bar graph (Figure 4a), biological process (BP) (Figure 4b), which contains extracellular matrix organization, extracellular structure organization, inner ear morphogenesis; cellular components (CC) (Figure S1a), which involved laminin complex, filopodium tip, sterocilium tip; molecular functions (MF) (Figure S1b), such as hyaluronic acid binding, UDP-galactosyltransferase activity and actin-dependent ATPase activity. KEGG Pathways (Figure 4c and Figure S1c) included Galactose metabolism, Viral life cycle – HIV-1, ECM-receptor interaction.

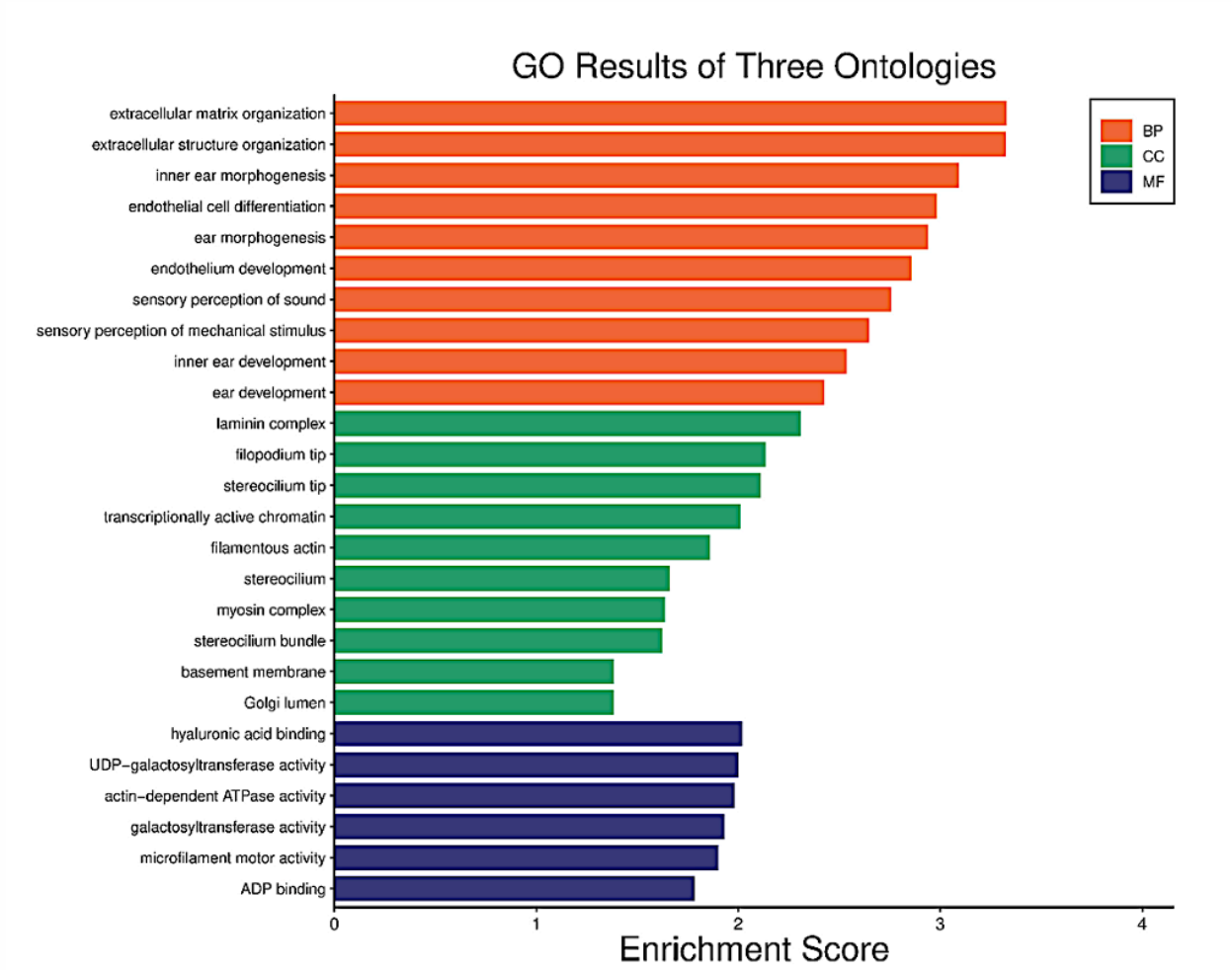

Figure 4a. Functional characteristics analysis for the AD-related DEGs: GO enrichment results of three ontologies. The orange indicates biological processes while the green indicates cellular components and the purple represents molecular functions.

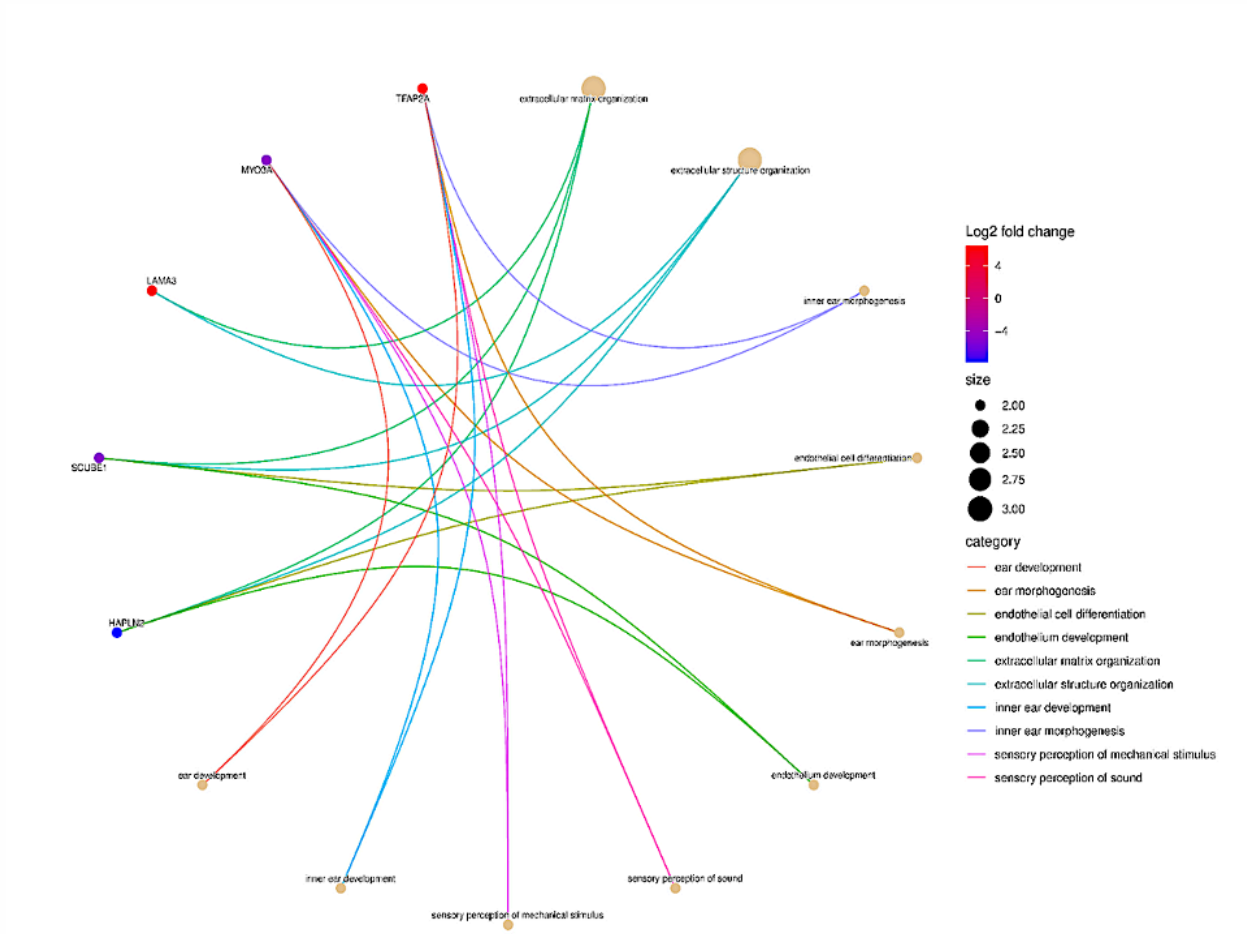

Figure 4b. Functional characteristics analysis for the AD-related DEGs: The Biological Process (BP) category of GO enrichment analysis. The nodes (dots) at the tip of the lines indicate genes. Genes with high log2 fold change are red while those with the lowest log2 fold change are blue in color.

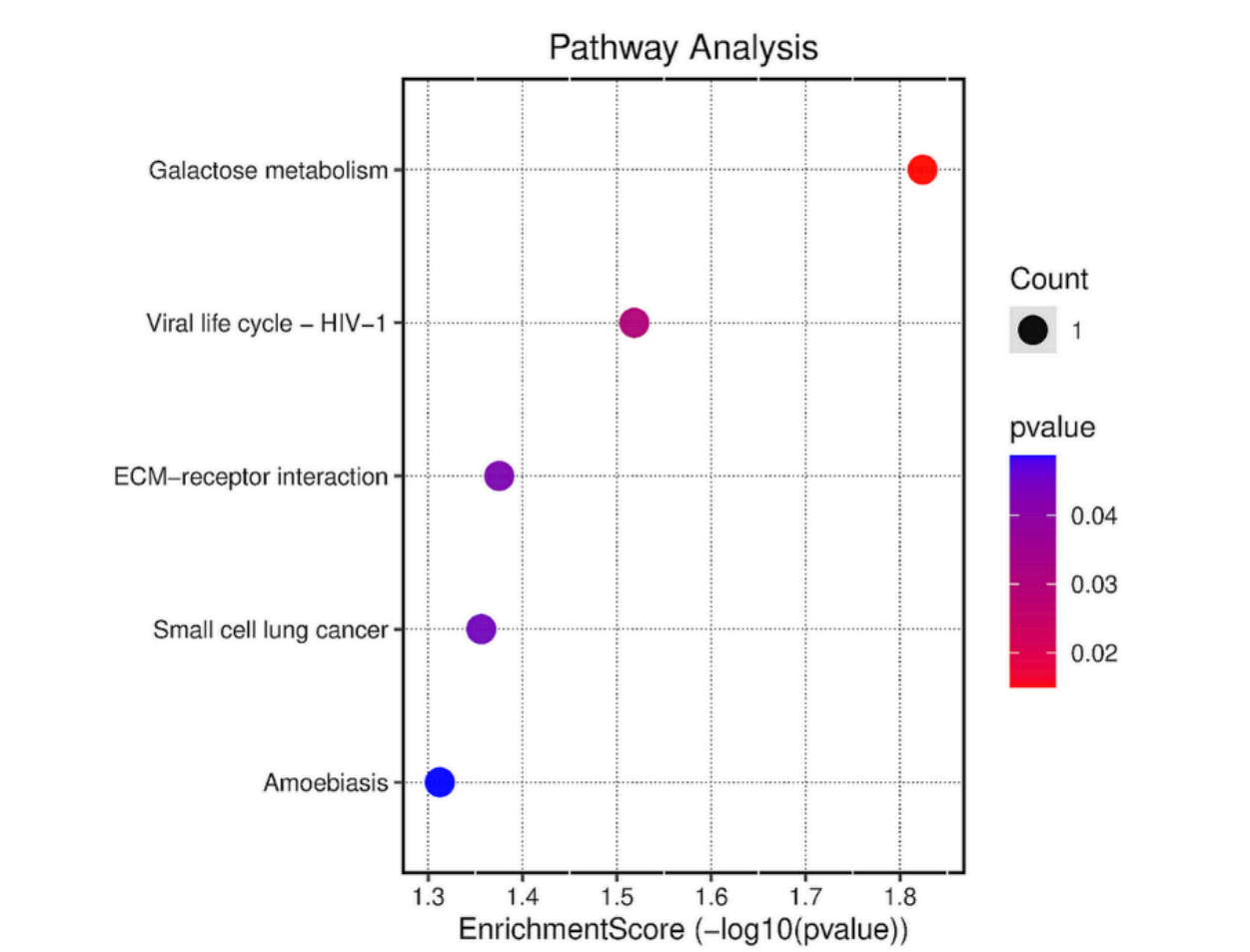

Figure 4c. The dotplot of KEGG pathway enrichment results for AD-related DEGs. The dot size represents the number of genes in each KEGG pathway; p.adjust (adjusted P-value): Red < purple < blue.
T2D GO analysis results show the three ontologies (Figure 5a), biological process (BP) (Figure 5b), which contains hepatocyte growth factor receptor signaling pathway, liver development, hepatobiliary system development; cellular components (CC) (Figure S2a), which involved platelet alpha granule, brush border, apical part of cell; molecular functions (MF) (Figure S2b), such as vitamin transmembrane transporter activity, peptidase regulator activity and growth factor activity. KEGG Pathway (Figure 5c, and Figure S2c) included Glucagon Signaling Pathway, Insulin resistance and Focal adhesion.

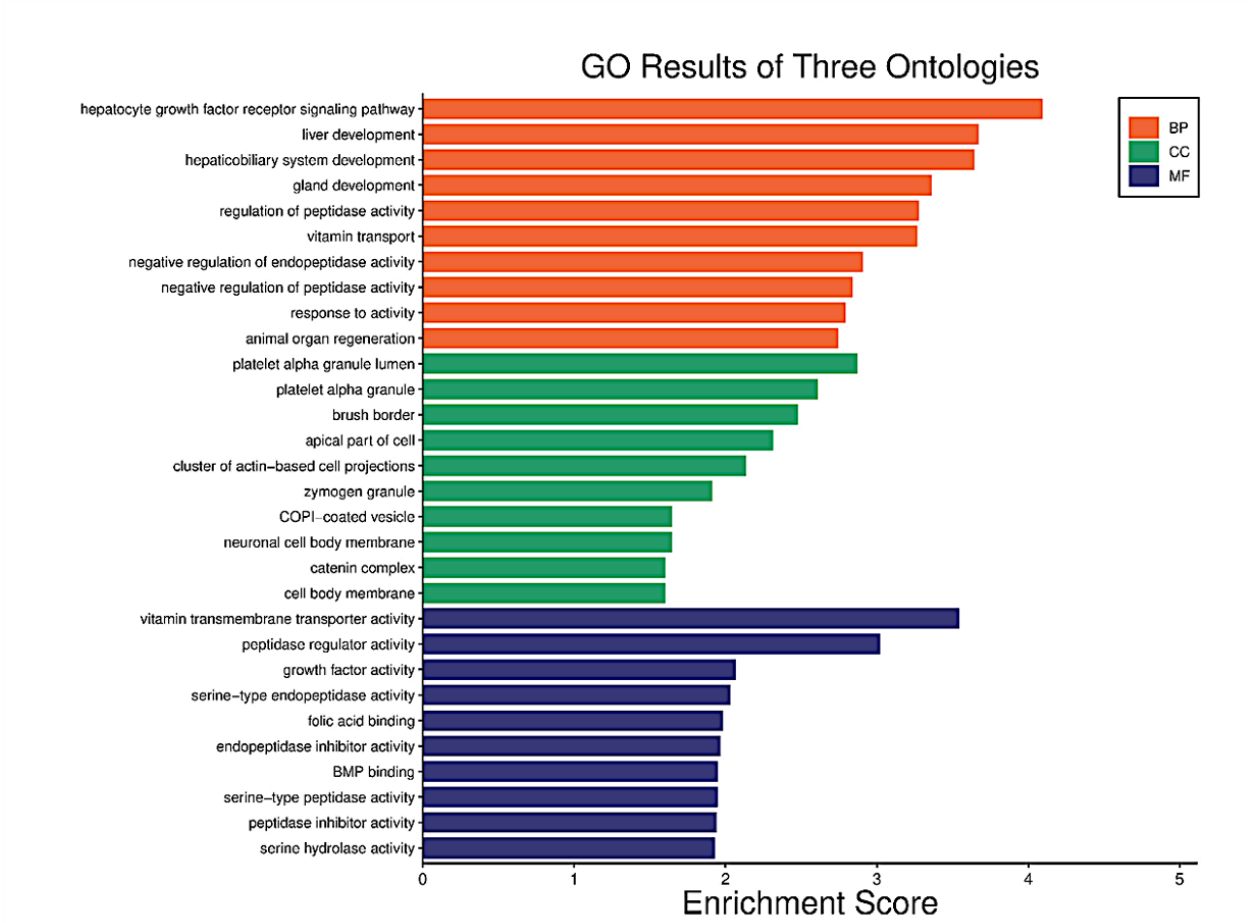

Figure 5a. Functional characteristics analysis for the T2D-related DEGs: GO enrichment results of three ontologies. The orange indicates biological processes while the green indicates cellular components and the purple represents molecular functions.

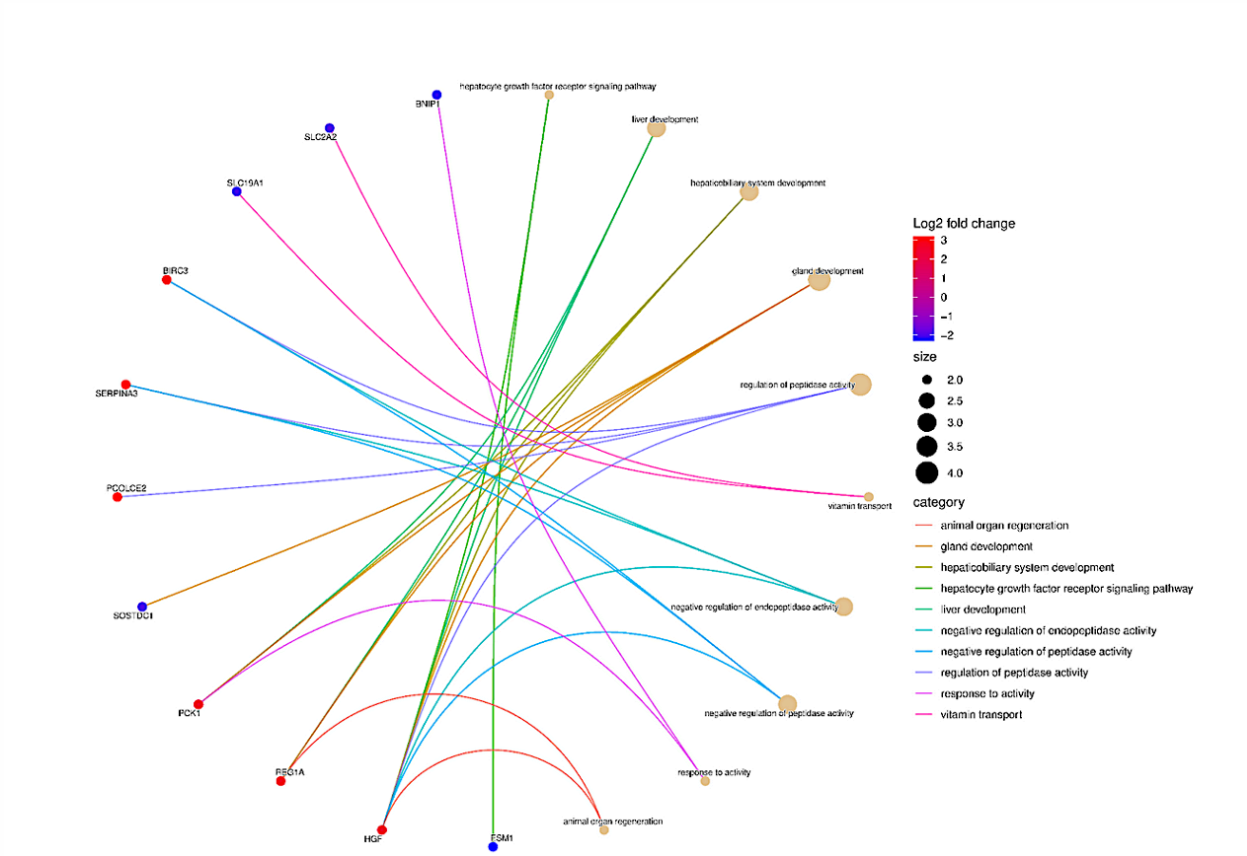

Figure 5b. Functional characteristics analysis for the T2D-related DEGs: The Biological Process (BP) category of GO enrichment analysis. The nodes (dots) at the tip of the lines indicate genes. Genes with high log2 fold change are red while those with the lowest log2 fold change are blue in color.

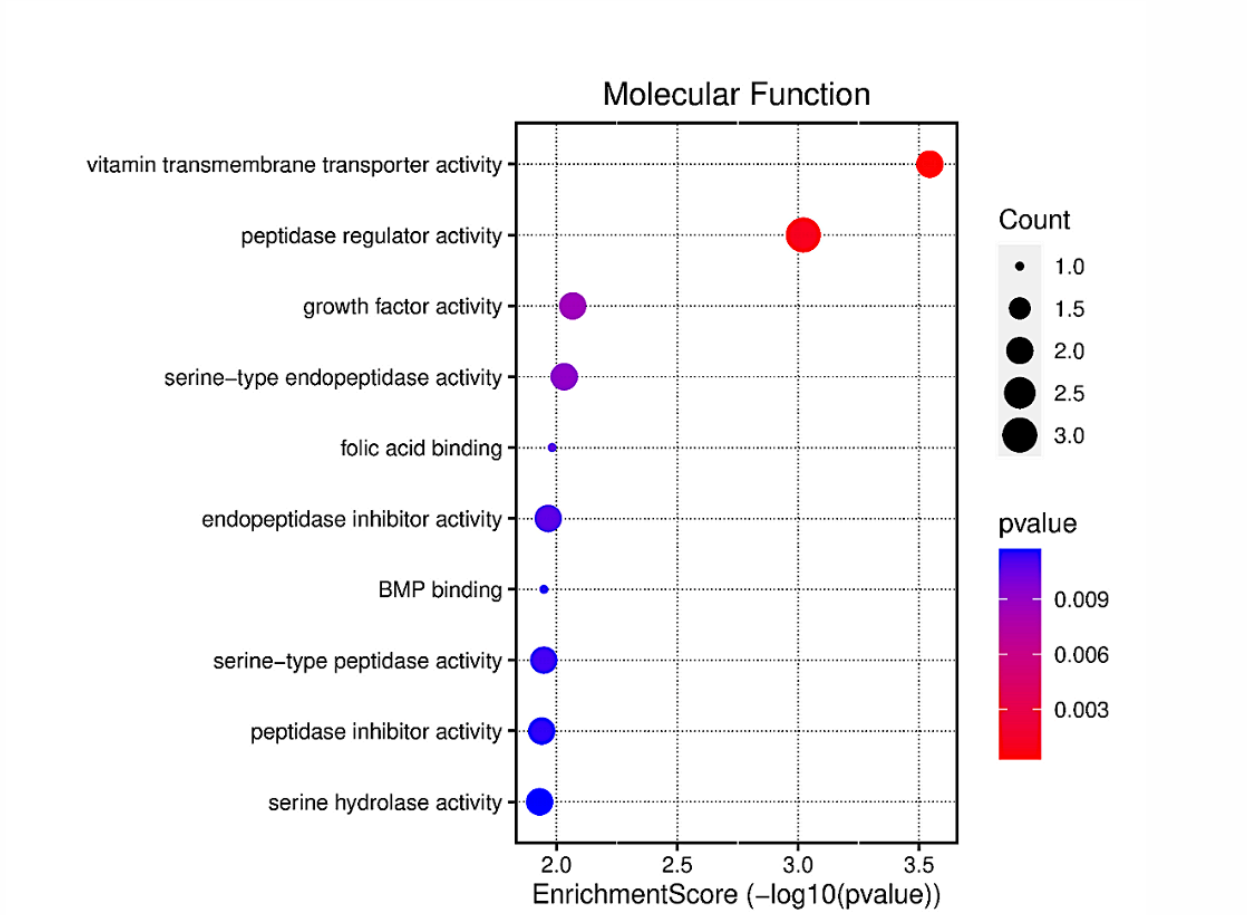

Figure 5c. The dotplot of KEGG pathway enrichment results for T2D-related DEGs. The dot size represents the number of genes in each KEGG pathway; p.adjust (adjusted P-value): Red < purple < blue.
A repeat GO and KEGG Analysis were performed for the selected AD and T2D DEGs. AD GO analysis results (Figure 6) show the three ontologies (Figure 6a), biological process (BP) (Figure 6b), which contains extracellular matrix organization, endothelial cell differentiation, endothelium development; cellular components (CC) (Figure S3a), which involved extrinsic component of plasma membrane, Extrinsic component of membrane; molecular functions (MF) (Figure S3b), such as hyaluronic acid binding, carboxylic acid binding and organic acid binding. There were no KEGG Pathway results for these DEGs.

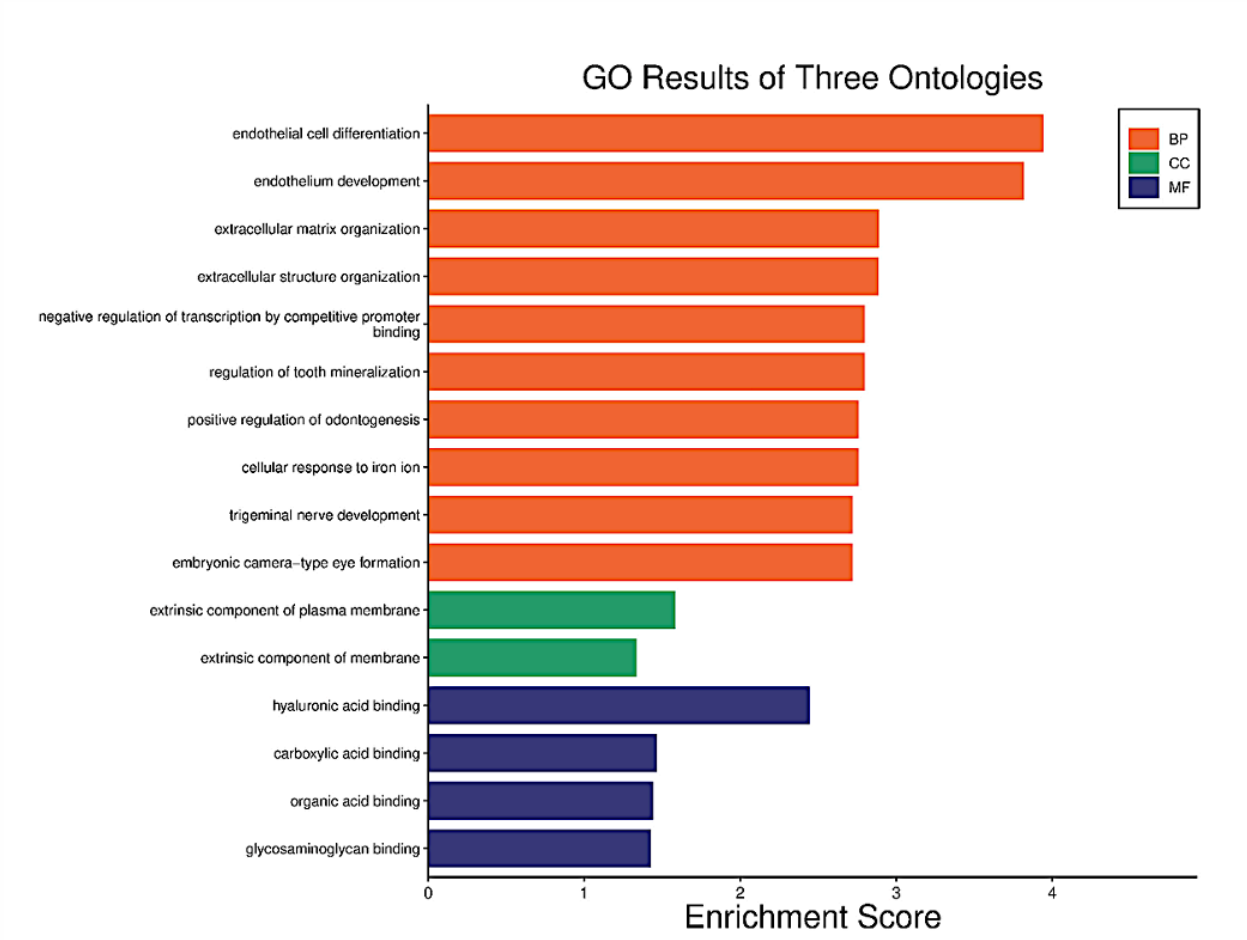

Figure 6a. Functional characteristics analysis for the AD-related DEGs: GO enrichment results of three ontologies. The orange indicates biological processes while the green indicates cellular components and the purple represents molecular functions

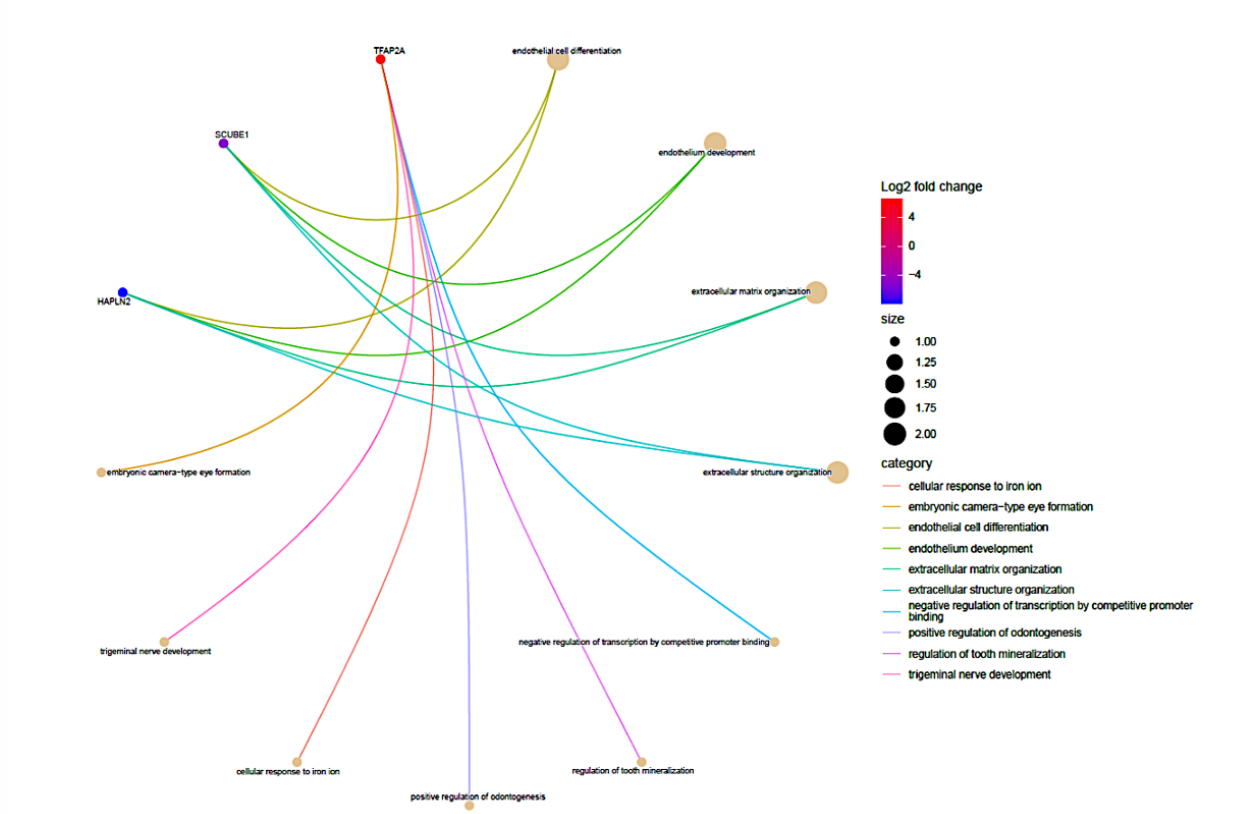

Figure 6b. Functional characteristics analysis for the AD-related DEGs: The Biological Process (BP) category of GO enrichment analysis. The nodes (dots) at the tip of the lines indicate genes. The genes with high log2 fold change are red while those with the lowest log2 fold change are blue in color. These genes were further analyzed to determine their association with T2D.

T2D GO analysis results show the three ontologies (Figure 7a), biological process (BP) (Figure 7b), which contains vitamin transport, animal organ regeneration, digestive system process; cellular components (CC) (Figure S4a), which involved platelet alpha granule, platelet alpha granule lumen, brush border; molecular functions (MF) (Figure S4b), such as vitamin transmembrane transporter activity, growth factor activity and organic anion transmembrane transporter activity. KEGG Pathway (Figure S4c and S4d) included vitamin digestion and absorption, maturity onset diabetes of the young and Antifolate resistance. Among the significant enrichment pathways were the renal cell carcinoma and prolactin signaling pathways which indicate the potential connection between T2D and AD.

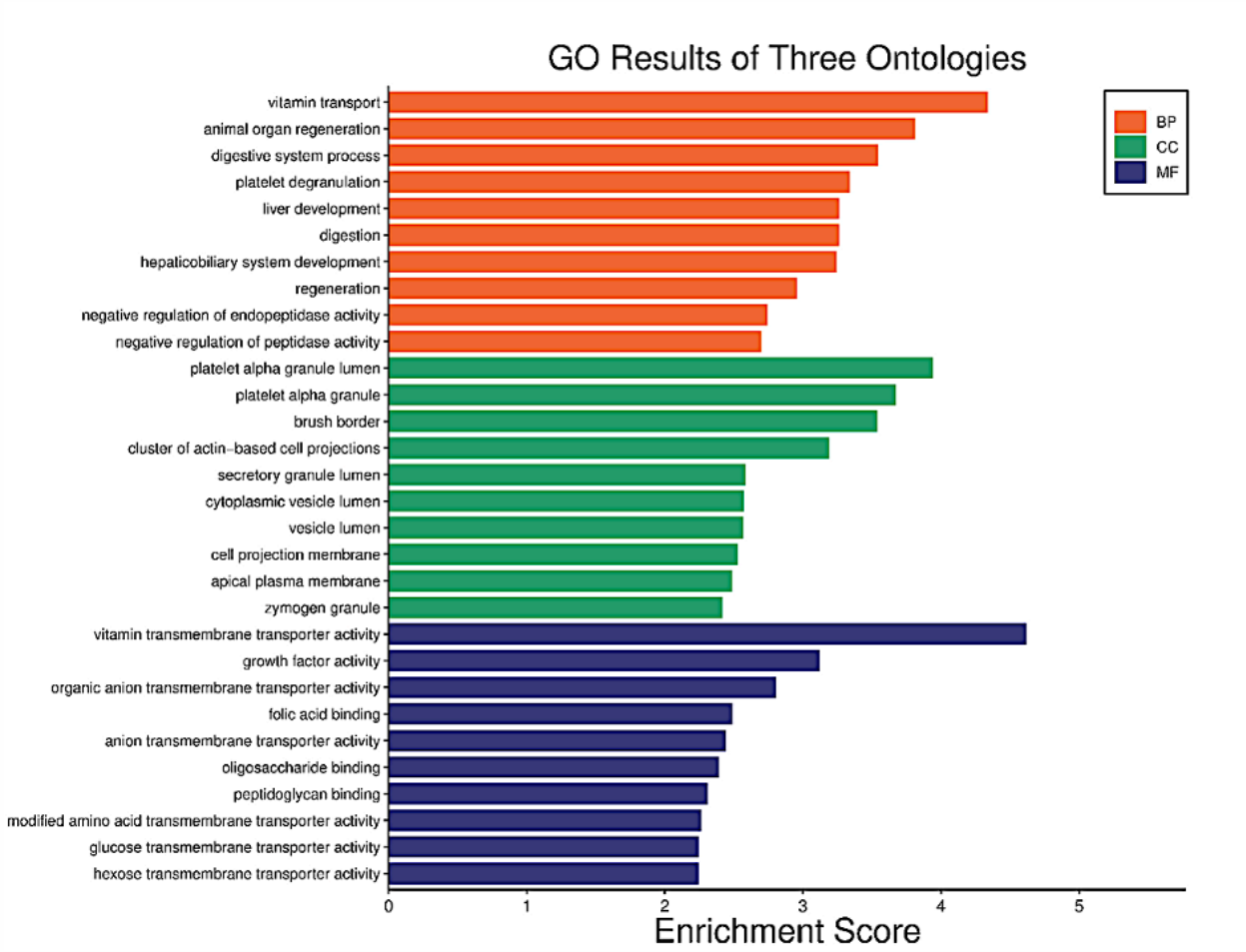

Figure 7a. Functional characteristics analysis for the T2D-related DEGs: GO enrichment results of three ontologies. The orange indicates biological processes while the green indicates cellular components and the purple represents molecular functions.

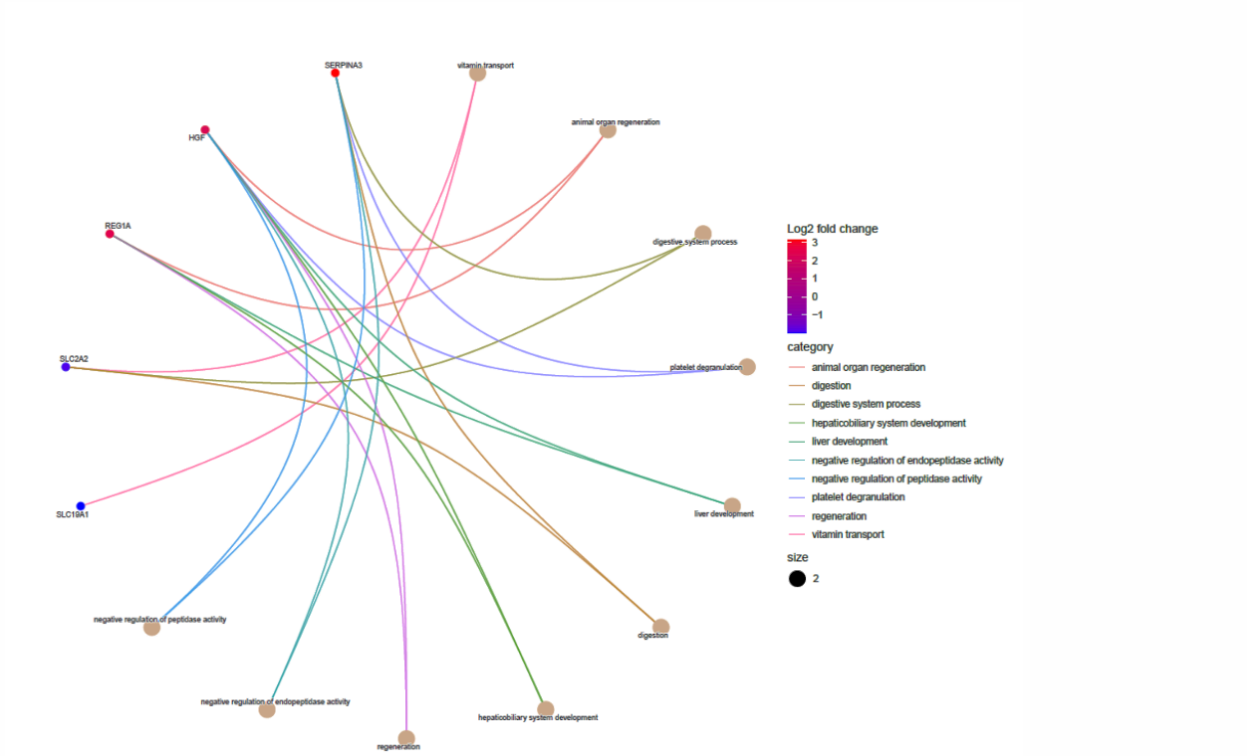

Figure 7b. Functional characteristics analysis for the T2D-related DEGs: The Biological Process (BP) category of GO enrichment analysis. The nodes (dots) at the tip of the lines indicate genes. The genes with high log2 fold change are red while those with the lowest log2 fold change are blue in color. These genes were further analyzed to determine their association with AD.

Finally, after examining all the outcomes of Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) analyses for both Alzheimer's disease (AD) and type 2 diabetes (T2D) (Figures 4-7), a total of eight genes exhibiting potential associations with both conditions were identified and subsequently shortlisted (Table 1). Specifically, the genes SLC19A1, SLC2A2, SERPINA3, REG1A, and HGF from T2D showed to have an association with Alzheimer's. While, genes TFAP2A, HAPLN2, and SCUBE1 from AD showed an association with Type 2 diabetes (Table 1).

Table 1: Genes from AD and T2D that have an association with each other.

Genes from AD that have an association with T2D	Genes from T2D that have an association with AD
TFAP2A	SERPINA3
HAPLN2	SLC19A1
SCUBE1	SLC2A2
	REG1A
	HGF

SUPPLEMENTARY DATA

Supplementary 1: R Script used to generate DEG results using GEO2R bioinformatics tool

R script for T2D Dataset 20966

R script for AD Dataset 97760

Supplementary 2: Excel sheet showing results of the chosen top 40 DEGs for both AD and T2D that are statistically significant based on $|\text{logFC}| \geq 1$ and $P < 0.05$.

(a) Top 20 AD DEGs

(b)Top 20 T2D DEGs

(c) Top 8 AD and T2D Genes that have association with each other

SUPPLEMENTARY FIGURES

Supplementary Figure 1 AD First GO and KEGG

Supplementary Figure 2 T2D First GO and KEGG

Supplementary Figure 3 AD Second GO and KEGG

Supplementary Figure 4 T2D First GO and KEGG

DISCUSSION

The increasing worldwide prevalence of Alzheimer's disease (AD) and Type 2 Diabetes (T2D) has caused significant health challenges and impacted healthcare costs (Moheet et al, 2015; Chung and Lee, 2021). Despite being seen as separate conditions, numerous studies have indicated a potential link between AD and T2D due to shared risk factors and effects on cognitive or thinking function, and brain structure (Moran et al, 2013; Moheet et al, 2015; Miles and Roof, 1922; Biessels, 2006; Rojas et al, 2021). In fact some research has proposed the term "type 3 diabetes" for Alzheimer's disease (Kandimalla, Thirumala & Reddy (2017; Mittal, Mani & Katare (2016). However, the exact genetic or molecular relationship between AD and T2D is still under exploration. While some studies have identified certain genes that

potentially link these two conditions, it is possible that there are other genes, not yet discovered. Therefore, the goal of this study was to determine novel genes or molecular markers that potentially connect AD with type 2 diabetes.

To achieve this, we used various bioinformatics tools and databases namely, NCBI, GEO, SR Plot, GO, and KEGG to identify potential genes shared between AD and T2D. Our findings suggest that specific genes and pathways are potentially commonly implicated in biological processes and molecular functions of both AD and T2D.

Using NCBI and GEO2R, we identified differentially expressed genes (DEGs) in AD and T2D (Figures 2-3). I then used statistics to shortlist the DEGs to 10 up-regulated genes and 10 down-regulated genes for each condition (Supplementary 2a). The GO and KEGG analysis of the functional features of the common DEGs revealed significant biological processes and molecular functions shared between AD and T2D (Figures 4-7). For example, the GO analysis for AD highlighted extracellular matrix organization, endothelial cell differentiation, and hyaluronic acid binding, among others (Figure 4). On the other hand, the GO analysis for T2D pointed to significant processes such as vitamin transport, digestive system function, and growth factor activity (Figure 5). Additionally, the KEGG pathway analysis for both AD and T2D provided insights into the potential connection between these diseases, indicating pathways such as vitamin digestion and absorption, maturity onset diabetes of the young, and antifolate resistance (Figure 4-7).

The overlap of several pathways, including renal cell carcinoma and prolactin signaling (Figure 4-5), further supports the potential interplay between AD and T2D. These pathway results suggest that there are potential shared mechanisms that contribute to the development and progression of both diseases.

Finally, based on the GO and KEGG analysis results for AD and T2D (Figures 4 – 7) , the genes that had potential connection to both AD and T2D were shortlisted (Table 1). Specifically, TFAP2A, SCUBE1, HAPLN2 from AD have an association with T2D, while the genes SERPINA3, REG1A, HGF, SLC19A1, and SLC2A2 from T2D have an association with AD (Table 1). These identified genetic markers provide a starting point for future research and

potential clinical interventions targeting the complex relationship between AD and T2D.

Limitations: However the lack of availability of many studies that show significant DEGs for AD and T2D limited our study to only two GEO datasets (Figure 2 – 3). Further, because this study uses bioinformatics data (tools and databases) derived from microarray laboratory experiments performed by other researchers, further validation of these genes is needed in a laboratory or clinical setting. Likewise, further study is needed to determine whether these genes are beneficial for clinical assessment.

Conclusion:Several bioinformatics tools and databases showed potential shared genes, biological processes, molecular functions and pathways that contribute to the development and progression of AD and T2D. We identified 3 genes; TFAP2A, SCUBE1, HAPLN2 from AD that have an association with T2D, as well the 5 genes SERPINA3, REG1A, HGF, SLC19A1, and SLC2A2 from T2D that have an association with AD. These eight genes serve as potential genetic biomarkers that can potentially contribute to the development of treatment strategies capable of addressing both AD and T2D at the same time.

The findings of this study lay the groundwork for further research in a laboratory and clinical setting to investigate the molecular interactions and pathways that contribute to the complex relationship between AD and T2D. The insights provided by this study have the potential to drive the development of new treatment interventions that could significantly improve patient outcomes and quality of life for individuals affected by both AD and T2D.

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Vedaant Bhatia

The Impact Of Socioeconomic Circumstances On Cardiovascular Health

ABSTRACT

Background: As a growing international issue, cardiovascular disease (CVD) is notorious for its mortality rates and five key risk factors: diabetes, substance abuse, poor dieting, poor exercise, and stress. However, a social determinant of health, socioeconomic status (SES) is also hypothesized to play a significant role in cardiovascular health. This literature review aims to explore the significance of SES in cardiovascular health globally using five research articles.

Methods: The search engines PubMed and Google Scholar were utilized in this literature review, using key search terms, [Cardiology OR Cardiovascular Disease OR CVD] AND [Socioeconomic]. The inclusion criteria were factors related to income, occupation, living standards, or class. The exclusion criteria were factors based on demographics like culture or ethnicity, non-original research, and sample groups under 5,000 participants.

Results: All five studies concluded that there is a proportional correlation to cardiovascular health and SES. Notably, 25% of all US adults with early coronary heart disease (CHD) burden between ages 35 and 64 years of age were found to have low SES.

Discussion: The correlation between SES and CVD is significant and on a global scale, in a variety of ways including worsening risk factors, preventing sustainable access to healthcare resources, and increasing mortality rates.

INTRODUCTION

Significance of the Problem

Cardiovascular disease (CVD) is the most widespread major disease in the United States, so much so that ⅛ of all deaths in the United States are due to CVD [1]. The risk factors associated with CVD are comorbidity of diabetes, substance abuse, poor dieting, a lack of exercise, and stress [1]. However, there are also social determinants of health that are known to correlate to these risk factors and the trends of cardiovascular health all over the planet, including education, systemic prejudice, healthcare access, community, and socioeconomic status (SES) [2]. By investigating the trends of social

determinants, we can address modern-day issues when it comes to cardiology and the risk factors associated with it.

Socioeconomic status (SES) for example, may contribute to higher risk for CVD due to poorer health insurance in occupations, less flexible work schedules, and low income that may prevent people who are symptomatic for CVD refrain from getting often expensive and demanding healthcare resources [2]. Someone's SES is tied to risk factors like stress or poor exercise due to a lack of flexibility in work hours, or poor dieting due to less income providing less options on the table. Hence, addressing SES is important to improving the cardiovascular health of individuals all over the globe.

Objectives

This literature review will analyze studies of cardiovascular health among populations of varying demographics to find out whether or not SES is a significant factor in CVD. By highlighting the social links to cardiovascular health, this paper aims to advocate for the further financial aid of lower SES-bound individuals to make a positive impact on cardiovascular health.

METHODS

Search Strategy

PubMed and Google Scholar were used to conduct this scoping literature review on CVD and how it correlates to SES. The key search terms were [Cardiology OR Cardiovascular disease OR CVD] AND [Socioeconomic].

Inclusion and Exclusion Criteria

The inclusion criteria were factors related to income, living standards, class, or occupation that correlated to cardiovascular health, not exclusive to the United States. In general, I attempted to search for studies based in different regions and levels of development to widen the scope of our results. The exclusion criteria were factors based on demographics such as ethnicity and culture, or non-original research. Research that surveyed a low population (<5,000) of individuals were also excluded.

RESULTS

Tabulated Results Summary

Title	Author	Procedure	Conclusion
Relationship between the shift of socioeconomic status and cardiovascular mortality [3].	Jidong Sung, Yun-Mi Song, Kyung Pyo Hong	-172,812 participants -South Korea -Mortality rates based on SES were assessed for all patients to find a link between SES and CVD	Mortality rates were found to be significantly higher for people of low and middle SES
Modifiable risk factors, cardiovascular disease, and mortality in 155 722 individuals from 21 high-income, middle-income, and low-income countries (PURE): a prospective cohort study [4].	Salim Yusuf, Philip Joseph, Sumathy Rangarajan, et al.	-155,722 participants -21 countries among varying SES -The goal was to analyze modifiable risk factors for CVD and mortality rates	Out of 14 modifiable risk factors found, a significant amount of mortality rates were based on SES, concluding that SES was a significant factor.

Effects of Socioeconomic Status on Access to Invasive Cardiac Procedures and on Mortality after Acute Myocardial Infarction [5].	David A. Alter, M.D., C. David Naylor, M.D., D.Phil., Peter Austin, Ph.D., and Jack V. Tu, M.D., Ph.D.	-51,951 patients -Canada -Income averages assessed based on access to cardiac procedures a year after myocardial infarction	Results concluded decreased waiting times for treatments were sharply connected to an increase in median neighborhood salary
Association of Low Socioeconomic Status With Premature Coronary Heart Disease in US Adults [6].	Joanne Penko, Rita Hamad, Dhruv S. Kazi, et al.	-31.2 million participants -United States -The observed risk factors were compared with SES to determine the correlation between SES and cardiovascular health	This study concluded that SES is a significant factor in the cardiovascular health of American adults
Impact of Socioeconomic Status on Cardiovascular Disease and Mortality in 24,947 Individuals With Type 1 Diabetes [7].	Araz Rawshani, Anne-Marie Svensson, Annika Rosengren, Björn Eliasson, Soffia Gudbjörnsdottir	-24,947 patients -Sweden -The study examined SES to determine if it was a risk factor of CVD in type 1 diabetes patients	The study concluded that SES was significant in the outcome of CVD in the patients

Study Procedures

Sung et al. selected 178,812 deceased participants who were 20+ years of age, had available medical records, and varying SES in a study to determine the correlation between socioeconomic status (SES) and cardiovascular disease (CVD) [3]. Similarly, The Prospective Urban Rural Epidemiology (PURE) study conducted by Yusuf et al. had 155,722 people, although they acquired data from 21 countries among varying economic statuses in order to obtain a wider scope. Yusuf et al. aimed to provide a thorough analysis of modifiable risk factors for CVD all over the globe using mortality rates among different ethnic and socioeconomic groups [4]. In contrast, a vital study composed by Alter et al. solely in the Canadian province of Ontario examined the performance of the Canadian healthcare system by assessing the impact of neighborhood incomes on access to cardiac procedures and mortality a year following myocardial infarction. Alter et al. assessed 51,591 patients and former patients who experienced acute myocardial infarction and used the median income of their respective neighborhoods to assess their SES [5]. Being a notable maverick, Penko et al. conducted a computer simulation study using the Cardiovascular Disease Policy Model and trends of coronary heart disease (CHD) in the United States. Penko et al. conducted a computer simulation and utilized mortality rates and the trends of risk factors to be compared with SES among all American adults between 35 and 64 years old to detect whether or not an underlying correlation exists between SES and risk factors for CHD in the United States [6]. Finally, Rawshani et al. performed a study in Sweden to investigate the linkage between SES and CVD in adults with type-1 diabetes[7]. Rawshani et al. studied 24,947 Swedish adults with type-1 diabetes and measured socioeconomic factors including income, occupational benefits, living standards, and marital status [7].

Study Outcomes

Sung et al. determined that cardiovascular mortality rates were significantly higher for people of low and even middle SES compared to higher SES [3]. The used regression model used a hazard ratio of 0.46, 95% confidence interval 0.4-0.52 which demonstrated that an upward shift of SES was associated with a lower risk of cardiovascular mortality [3]. Additionally, Yusuf et al. observed that a concerning amount of mortality rates were based on poverty, a lack of exercise, and other factors related to lower-income circumstances affecting people’s cardiovascular health [4]. The

population-attributable fractions or PAF were composed of many unfortunate shortcomings when it came to the wealth gaps in different countries, causing a concerning amount of substance abuse that made up 26.3% of PAF [4]. Moreover, Alter et al. noted that the waiting times for treatments for patients in the study decreased by 45% and that fatality was reduced by 10% for every $10,000 increase in neighborhood median income [5]. Likewise, Penko et al. recorded that 25% of all adults aged between 35 and 64 years old in the United States who demonstrated early CHD burden were of significantly low SES based on projections through the simulation [6]. Furthermore, using the Cox Regression Model with a socioeconomic variable, Rawshani et al. produced results indicating that factors associated with SES including marital status and higher income demonstrated a 50% lower risk of CVD in all potential-risk patients. It was noted that lower SES was observed to have increased the fatality rate of CVD by 2-3 times in type 1 diabetes patients [7].

DISCUSSION

Key Findings

Based on the findings of these five studies, it's abundantly clear that SES has an impact on CVD of an individual of almost any age globally, whether it comes to causing early burden for CHD, increasing mortality rates, or worsening due to associated risk factors [2-7]. To reiterate, in the United States alone, CVD is an overwhelming fifth of deaths and the fact that a fourth of early burden of cardiovascular issues are correlated to a low SES demonstrates the large role SES plays in cardiovascular health nationwide [1, 6]. It's also transparent that this is an international issue, as shown by increased mortality rates based on SES in 21+ countries all over the globe, and that mainstream risk factors including diabetes and stress plays a role in CVD in accordance with SES [2, 4, 7].

Addressing Drawbacks

Although the evidence gathered from analyzing the five studies does heavily align with the idea that cardiovascular health correlates to SES, there are potential drawbacks to the analysis that should be addressed before proceeding [2-7]. While these studies did take large numbers of people into account, they may not represent entire populations. It's also worth noting four of the studies are entirely focused on a developed yet diverse set of nations including the United States, Republic of Korea, Sweden, and Canada

which may not be representative of cardiovascular health on an international scale, particularly in low-income countries [3-7]. While most of the studies were based in developed countries, Yusuf et al. took into account 21 diverse nations of varying development and standards of living which strengthens our ability to generalize the findings globally [4].

Moving Forward

The purpose of this literature review was to bring light to the global issue of SES upon cardiovascular health in order to promote further research and application of strategies to combat economic disparity and classism as modifiable risk factors of CVD.

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Vedaant Bhatia

Plasmid Gene Therapy To Treat Type-1 And Type-2 Diabetes

ABSTRACT

Current treatments for Type-1 and Type-2 diabetes suffer from their demand for frequent injections to their drug interaction issues. Plasmid gene therapy for diabetes has recently gained popularity, where a gene of interest (GOI) is inserted into a bacterial plasmid and injected into the body to go through biological processes. The gene is then translated to increase insulin levels and regulate blood glucose (BG) levels. Using the search engines Google Scholar and PubMed, I found experiments that met my inclusion and exclusion criteria on the basis of plasmid gene therapeutic experiments on animals genetically similar to humans. Experiments with the genes of interest adiponectin (ADN), glucagon-like peptide 1 (GLP-1), and IL-4/IL-10 had consistent results lasting multiple weeks. A treatment using electrotransfer (ET) administration methods also had consistent results. These treatments have demonstrated that they may be better than current ones, like intensive insulin therapy (IIT), Metformin, and Ozempic. Further testing may show that plasmid gene therapy has a strong potential to treat both forms of diabetes in humans.

INTRODUCTION

Diabetes is a chronic disease affecting how the body regulates blood glucose (BG) and is caused by a lack of insulin production in the body [1]. Diabetes comes in two forms: Type-1 and Type-2 [1]. Type-1 tends to develop at a younger age as it is a genetic disorder, while Type-2 is more prevalent in adults as it is a result of a lack of physical activity and a healthy diet [1]. Type-1 diabetes occurs when the human immune system incorrectly attacks the pancreatic β-cells that produce insulin [1]. Without insulin to facilitate glucose, BG levels rise, leading to hyperglycemia [1]. Hyperglycemia causes dehydration and frequent urges to urinate. Eventually, these complications will cause harm to vital organs including the heart [1]. In comparison, Type-2 diabetes is not caused by a flawed attack on pancreatic β-cells influenced by genes, but rather behavioral factors such as poor diet and lack of exercise [2]. BG levels reach a peak if the pancreatic β-cells are simply unable to produce enough insulin to keep them under control [2]. When BG levels are excessively high as a result, diabetes patients are at risk of

hyperglycemia [1]. Diabetes exists in a variety of ways and has a variety of current treatments [3].

Popular treatments for Type-1 diabetes include intensive insulin therapy (IIT) where insulin is administered to the body to substitute the insulin that was never produced by the failing pancreatic β-cells [4]. IIT requires 4-5 self-checks for BG levels and 3-4 injections of insulin daily [4]. Although IIT has been proven to assist in lowering BG levels and reducing the risks of organ damage, 1 in 2 patients fail to improve under IIT [4]. The time, money, and dedication a Type-1 diabetic is expected to inject insulin throughout the day are difficult to maintain, and failure to monitor their decreased BG levels carefully puts patients at risk for hypoglycemia, the reciprocation of hyperglycemia. Hypoglycemia causes nausea, headaches, fatigue, and if left untreated for long enough, organ damage just like its counterpart, hyperglycemia [1].

Ozempic, an injection that produces sufficient amounts of insulin and regulates BG levels is a known treatment for Type-2 diabetes [5]. However, injections must be administered weekly, which may make adherence difficult for patients [5]. On the other hand, one of the most popular treatments for Type-2 diabetes worldwide is Metformin, a medication taken daily that reduces the amount of glucose produced by the liver leading to decreased BG levels [6]. However, Metformin lacks effectiveness when in contact with other drugs prescribed for treating Type-2 diabetes, which is a significant problem for people with more severe Type-2 diabetes who may need more than just Metformin [6].

A different form of diabetic treatment has recently grown in popularity: using genes of interest (GOIs) in plasmid vectors. A plasmid is a circular molecule of DNA found in bacteria that is commonly used in biomedicine as a vector for the replication of genes of interest due to its versatility and reproduction capabilities [7]. This form of gene therapy has been tested commonly on rodents who share 97.5% of their working DNA with human beings [8]. By adding a GOI to a bacterial plasmid vector, the plasmid, if administered carefully, will ensure quick and effective replication of the GOI. This applies to both Type-1 and Type-2 diabetes because hormones that assist in insulin production such as adiponectin (ADN) will be replicated on their own inside the body. With fewer injections, the risks of hyperglycemia

and hypoglycemia are reduced [9]. Similarly, glucagon-like peptide-1 (GLP-1) has been known for its potential in diabetes treatment for decades, but the dipeptidyl peptidase 4 (DPP4) enzyme denatures GLP-1 in the body [10]. Plasmids protect this GOI, ensuring that GLP-1 levels increase in the body, effectively treating diabetes [10]. Unlike Metformin, there are no clear drug interactions with this treatment [6, 10]. Although Ozempic is an effective GLP-1 treatment as well, it must be taken every week whereas plasmid treatments appear to only require administration every 2 [11] or even 3 weeks [10]. Scientists may even be able to utilize gene therapy to prevent diabetes, as they counter the autoreactive T-cells in Type-1 diabetes with helpful T-cells IL-4 and IL-10 as genes of interest [12]. Additionally, new methods of administration are being developed, such as electrotransfer (ET) [13]. With plasmids, we can see a new future in the realm of Type-1 and Type-2 diabetes treatments.

The objective of this study is to review the effectiveness of a variety of genes of interest to be carried in the plasmid vector and assert their utilities based on how they were tested on animal models.

METHODS

The search terms I used were “Type-1 diabetes”, “Type-2 diabetes”, “plasmids”, “gene therapy”, and “gene of interest” in the search databases PubMed and Google Scholar.

My inclusion criteria were research that used animal models with similar genetic make-up to humans, involved bacterial plasmids as the form of gene therapy with any gene of interest, and specifically used plasmids for the purposes of researching how plasmids offer protection and sustainability in the body. I also allowed experiments that aimed to treat any form of diabetes.

My exclusion criteria were research that focused on advertising a gene of interest or strategy, used animal models with less genetic similarity to humans, or used gene therapy technology without bacterial plasmid vectors, such as CRISPR technology. I also excluded research that met the above criteria for diseases that are not for treating diabetes or increasing insulin in the body.

RESULTS

GOIs and Methods of Administration

Nan et al. and Fukushima et al. administered ADN using the plasmid vector pVAX1 for the former, but unspecified for the latter [9, 14]. They also both administered streptozotocin (STZ) into the mice in order to give them Type-2 diabetes, however, Nan et al. administered the plasmid into multiple cell lines of the tail vein including HeLa, HEK293, HepG2, HT22, and SK-Hep 1 while Fukushima et al. focused on administration to HepG2 cells only [9, 14]. Jean et al. and Kumar et al. both administered GLP-1, but Kumar et al. used a GLP-1/Fc fusion protein, including a fragment crystallizable receptor (Fc) antibody [10, 11]. In contrast, Jean et al. used therapeutic nanocomplexes for plasmid material (TNC) [10]. Kumar et al. aimed to extend the shortened lifespan of GLP-1 in the body by administering the plasmid into the mice's tibialis anterior muscle while Jean et al. administered the plasmid into the skeletal muscle [11]. In contrast, Martinenghi et al. and Ko et al. utilized plasmids to treat Type-1 diabetic mice models [13, 12]. Instead of focusing on a specific GOI, Martinenghi et al. engineered special DNA sequences that directly code for insulin and focused on the procedure of an ET [13]. Ko et al. implemented beneficial T-cells, IL-4 and IL-10 as GOIs to prevent Type-1 diabetes in susceptible mice [12].

Outcomes

Nan et al. observed that when the expression levels of mRNA were checked, the cell lines that were exposed to the ADN-encoded plasmid treated the Type-2 diabetes effectively by facilitating BG levels and increasing insulin [9]. The duration of these results remained consistent for around 2 weeks [9]. In comparison, Fukushima et al. observed that the untreated mice exhibited lower ADN levels than the treated mice, who demonstrated mRNA levels of ADN 10-15 times more than before in HepG2 cell lines, also persisting for about 2 weeks after treatment [14]. Moreover, Kumar et al. noted that the GLP-1/Fc plasmid lifespan increased to 2 weeks after radioimmunoassay (RIA) confirmed its presence in transfected cells [11]. Jean et al. observed that treated diabetic mice models demonstrated roughly quadruple the amounts of GLP-1 compared to the control mice [10]. Raised GLP-1 levels persisted for 24 days following the final injection [10]. Furthermore, Martinenghi et al. recorded that 17 of the 20 mice observed had detectable levels of mature insulin in their bloodstream for around 6 weeks and that the procedure kept the treated mice alive for about 10

weeks [13]. Additionally, Ko et al. identified that the pancreatic □-cells in the untreated mice were severely damaged, while 75% of treated mice had perfectly healthy cells for about 6 weeks [12].

Source	GOI	Outcome	Duration of results
Nan M., Park J., Myung C.	ADN	Diabetic mice that originally had very low ADN levels demonstrated 10-15 times higher ADN levels after treatment.	2 weeks
Kumar M., Hunag Y., Glinka Y., Prud'Homme G., Wang Q.	GLP-1/Fc	Researchers successfully treated mice with Type-2 diabetes with a plasmid vector to protect the GLP-1/Fc fusion protein from denaturing.	2 weeks
Jean M., Alameh M., Buschmann M., Merzouki A.	GLP-1	The treated diabetic mice had 5 times more GLP-1 in their bloodstream than untreated mice and insulin levels peaked at 3 times more than before.	3 weeks
Ko K., Lee M., Joon J., Wan S.	IL-4 and IL-10	Untreated mice had severely damaged pancreatic β-cells, while diabetes was prevented in about 75% of treated mice.	6 weeks

Martinenghi S., Cusella De Angelis G., Biressi S., Amadio S., Bifari F., Roncarolo M., Bordignon C., Falqui L.	Self-made GOI, ET method	Seventeen out of 20 diabetic mice expressed consistent levels of mature insulin after administration into the skeletal muscle.	6 weeks
Fukushima M., Hattori Y., Tsukada H., Koga K., Kajiwara E., Kawano K., Kobayashi T., Kamata K., Maitani Y.	ADN	At the end of the study, treated diabetic mice had significantly higher ADN levels than the untreated mice.	2 weeks

DISCUSSION

Benefits of Longer Length

Based on observations from these gene therapeutic studies, the treatment effects have demonstrated remarkable consistency for 2, 3, and even up to 6 weeks in both types of diabetes [9, 11, 14, 10, 12, 13]. This suggests that individuals affected by Type-1 and Type-2 diabetes could anticipate significantly reduced frequency of injections compared to treatments of IIT requiring multiple daily injections or weekly injections for Ozempic. Gene therapy presents a promising solution for diabetic patients experiencing cognitive, aging, or physical issues who struggle to cope with frequent injections.

Further Benefits of Plasmid Gene Therapy

The efficiency of plasmid gene therapy is likely attributed to the protective capabilities of plasmid vectors, particularly in the case of GLP-1 [10, 11]. Moreover, the security of plasmid vectors may indicate that concerns related to cross-interactions with other drugs, which can be problematic for treatments like Metformin, are not a hindrance during plasmid gene therapy [5].

Addressing Drawbacks

Plasmid gene therapy for diabetes is still at an experimental stage and further examinations of its safety should be addressed. Still, the promising results that this unique treatment has to offer show the potential of it to eclipse current treatments. The ability to treat both forms of diabetes simply by changing the GOI has huge potential in cost-effective research, which is why advancement to human test subjects is necessary. The many similarities were salient to the synthesis of the key points of these experiments, but their differences in the type of diabetes, the GOIs, etc are important to help understand the versatility and effectiveness that plasmids ultimately hold in biomedicine.

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Naomi Burakovsky

What are the Effects of Educational Level on Cardiovascular Disease?

ABSTRACT

Introduction: Cardiovascular disease (CVD), including heart attacks and strokes, is the leading cause of death globally. Education may be a social determinant that plays a role as a protective factor in CVD. This research aims to answer the question: does one's level of educational attainment play a role influencing their risk of a lifetime CVD?

Methods: In order to investigate the relationship between CVD and educational levels, a scoping literature review was conducted through search engines PubMed and Google Scholar. The key search terms used were CVD, education, social determinants of health and impact. The articles used included studies that reported on level of education as a variable and reported on CVD risk in their results. Systematic reviews and other literature reviews were excluded.

Results: It was found that men are more likely to be smokers and that smokers more often had professions as hairdressers, hotel workers, butchers, and painters. Individuals with a less than high school education had a 13.54% risk (females) and an 8.92% risk (males) of being heavy smokers. In men, the CVD risk decreased from 59% to 42.2% while the educational level increased. Another study revealed that more than half of participants with less than a high school education experienced a lifetime CVD event.

Discussion:The level of education an individual receives was found to be correlated to their risk of a lifetime CVD. However, this study had limitations in that it could not fully isolate the direct impact of CVD on education, as there were mediation factors in between. Scientists and researchers should conduct further research on this topic, maybe even in different areas of the globe (to examine different educational systems), to find a more concrete answer on this topic.

INTRODUCTION

About 800,000 people die yearly of cardiovascular disease (CVD) in the US alone, which is 1 in every 3 deaths [1]. CVD is the leading cause of death not only in the US, but in the entire world. CVD demands urgent attention in order to mitigate its impact.

Smoking is known to damage the lungs and heart, and statistics show that smoking even one cigarette per day can cause a 40-50% increased CVD risk [2]. Obesity is also proven to be another factor in the development of CVD [3]. However, education is a social determinant of health that does not receive much attention.

Whether the level of education that one receives relates to their risk of developing CVD needs to be studied more due to the lack of attention it receives as a social determinant of CVD. Especially considering that CVD is such a big cause of death, any study that could help attain more information on the topic should demand attention. This study will review the relationship between the educational level of a person (eg. less than high school, high school graduates, more than high school) and their risk of developing CVD.

METHODS

This scoping literature search reviewed studies examining CVD's relation to educational attainment. The search engines used were PubMed and Google Scholar. The key search terms used included CVD, education, social determinants of health, and impact. The articles were included if they reported the level of education as a variable, studied mediation factors, and if they provided helpful information on how levels of education relate to CVD risk in general. The articles were excluded if they were not original research from credible, peer reviewed sources.

RESULTS

Additionally, people of all genders are more inclined to smoke if they work in certain professions, such as hairdressing, butchery, painting, or hotel work and men in general are more likely to be heavy smokers [5]. In addition, poor education is a significant factor in the intensity of cigarette smoking, with a 13.54% risk for females and an 8.92% risk for males being heavy smokers compared to individuals with higher education [5]. Another finding by the study indicated that less than 40% of smokers received a higher education, while more than 70% had a lower education [5].

It was observed that for men, the lifetime risk of CVD decreased from 59% to 42.2% as their educational level increased [6]. Another conclusion presented in the article stated that more than half of participants with less

than a high school education experienced CVD in their life [6]. Another study summarized that higher education was proven to improve high-density lipoprotein, good cholesterol, which subsequently decreases CVD risk [7]. Regarding racial differences, another study found similar results. For both black and white middle aged men and women with a less than a high school education, an increased risk of a stroke was found compared to those with a college completion [6].

Article	Purpose	Independent Variable	Dependent Variable
Educational Attainment and Lifetime Risk of CVD	To investigate the number of years that individuals lived with and without CVD through their level of education.	Level of education Sex	Lifetime CVD risk
Impact of school and vocational education on smoking behavior	To research how school impacts smoking behavior of adolescents and young adults.	Level of education Occupation	Smoking status
Association of Educational Attainment with Lifetime Risk of CVD	To find what the lifetime risks of CVD are according to educational attainment and to emphasize the need to reduce CVD inequalities.	Level of education Cholesterol levels	Lifetime CVD risk
Educational attainment and CVD in the United States		Level of education Race	Lifetime CVD risk

DISCUSSION

The key takeaway from the reviewed studies is that a lower educational attainment is found to increase the individual's risk of a lifetime CVD.

Smoking is also found to be a significant factor as increased smoking can be caused from lower educational levels and smoking is a attribute to CVD [5]. Now that a contributing cause to CVD is known, more immediate action can be taken to improve this situation globally.

The main research question of this study was whether one's level of educational attainment plays a role influencing their risk of a lifetime CVD, and after reviewing the studies and examining their results, the answer is found to be yes. Individuals with higher educational attainment demonstrate a correspondingly reduced risk of developing CVD, and vice versa. This finding demonstrates the profound impact of education on health issues. If educational attainment is linked to CVD maybe improving educational opportunities globally will serve as a preventive measure against CVD. However, it is beyond the scope of this study to determine whether education is a correlation or a causation of CVD, if it directly causes CVD or not. In addition, there are many other contributing variables to this topic, so the existing data may be incorrect as it does not fully isolate just education and CVD. This research is also not able to distinctly separate different educational systems, as the educational systems in the world differ and are very diverse from one another.

Further research is needed to establish a concrete position on whether educational levels are a variable to CVD risk or if it is directly associated. I also believe that many studies should be done in different areas across the globe, to review if different educational systems make an impact.

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Armaan Dewal

The Impact of Healthcare on Cardiovascular Disease

ABSTRACT

Background: In 2005, cardiovascular disease was the leading cause of death around the world, causing over 17.5 million deaths worldwide. Not only harming people, CVD has affected economies. In the UK, CVD cost the government 29.1 billion dollars. The major cost for this was utilization of healthcare services, which was 60% of the cost.

Methods: Google Scholar and PubMed were searched in this scoping literature review using the key search terms Healthcare, Impact, Cardiovascular, Disease.

Results: The more vascular beds (or, areas of blood vessels providing blood flow to a region) affected, the higher the healthcare cost. Cardiovascular disease and stroke cost the UK economy £29.1 billion in 2004. The two diseases accounted for more than half of the total cost, with heart disease costing £8.5 billion and stroke costing £8.0 billion. Healthcare made up 60% of the total cost, while productivity losses due to death and illness accounted for 23% of the cost, and informal care-related costs made up the remaining 17%.

Discussion: Regular check-ups can help catch heart problems early and reduce the risk of death or heart problems. This means that going for check-ups can save money in the long run and keep people healthier. Our findings match what others have said: it's important to find heart issues early to save money and keep people healthier. We need to make check-ups easier to get, especially for those at higher risk. This study tells us we can save money and lives by making sure more people get checked regularly. In the UK, heart disease and strokes incur significant healthcare expenses.

INTRODUCTION

Significance of the Problem

According to Rohina et al., in 2005 cardiovascular disease was the leading cause of death around the world, causing over 17.5 million deaths worldwide and 80% of these deaths occurred in low-income countries [1]. People who receive health screenings have significantly lower risks of getting affected by cardiovascular diseases (CVD). Not only harming people, CVD has affected economies. For instance, according to Luengo-Fernández, in the UK, CVD

cost the government 29.1 billion dollars [2]. The major cost for this was utilization of healthcare services, which was 60% of the cost [2].

Objectives

The aim of this paper is to investigate how much cardiovascular disease contributes to healthcare spending and how screening can prevent cardiovascular disease and decrease healthcare spending.

METHODS

Search Strategy

I used Google Scholar and Pubmed to conduct a scoping literature review. The key searched terms used wereHealthcare, Impact, Cardiovascular, Disease.

Inclusion and Exclusion Criteria

Studies that included patients over the age of 50 and studies that included healthcare utilization as a variable were included. Systematic reviews and meta-analyses were excluded.

RESULTS

According to Weng et al., 47.0% of 539,089 individuals with type 2 diabetes mellitus (DM) and atherosclerotic cardiovascular disease (ASCVD) had ASCVD affecting more than one region of blood vessels, also known as vascular beds [3]. The most common ASCVD diagnoses were acute coronary syndrome (26.6%), peripheral arterial disease (24.5%), and stroke (18.6%) [3]. The mean annual total healthcare costs per person increased with the number of affected vascular beds, from $17,741 for one bed to $25,877 for two beds to $33,412 for three beds [3]. A similar pattern of increased healthcare utilization was observed with an increasing number of vascular beds. Among individuals with one affected vascular bed, mean total healthcare costs per person were comparable across age subgroups [3]. However, if more than one vascular bed was affected, the mean total healthcare costs were highest in the youngest age cohort [3].

According to Luengo-Fernández et al., a study by the University of Oxford found that heart disease and stroke cost the UK economy £29.1 billion in 2004 [2]. The two diseases accounted for more than half of the total cost, with heart disease costing £8.5 billion and stroke costing £8.0 billion [2].

The biggest part of the cost was healthcare, which made up 60% of the total cost [2]. Productivity losses due to death and illness accounted for 23% of the cost, while informal care-related costs made up the remaining 17% [2].

According to Lee et al., out of 443,337 people, 160,607 people (that's 36.2% of the total) underwent a health screening in 2003-2004, and 110,278 people underwent another screening in 2005-2006 [1]. The study found that people who went for a health screening in 2003-2004 had a lower risk of dying from any cause and developing cardiovascular diseases between 2005-2010 compared to those who didn't go for a screening [1]. The incidence rate per 1000 people was also lower for those who went for a screening [1].

DISCUSSION

Our study showed that heart problems cost a lot for healthcare and hurt people's health. People with diabetes and heart issues had higher healthcare costs, especially if they had problems in more than one area. In the UK, heart disease and strokes incur significant healthcare expenses.

Getting regular check-ups made a big difference. People who got checked had lower risks of dying or getting heart problems. This means going for check-ups helps catch problems early and saves money in the long run. Our findings match what others have said: it's important to find heart issues early to save money and keep people healthier. We need to make check-ups easier to get, especially for those at higher risk. This study tells us we can save money and lives by making sure more people get checked regularly.

Overall, I was not surprised with my findings. I expected these results, since I understand just how important healthcare is for the health and wellbeing of people. So, difficult access to good healthcare can prevent good health and wellbeing. In the future, we can use this data to try and increase the amount of healthcare access in other areas, especially in underprivileged areas. This way, more people can gain access to healthcare all around the world.

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Michael Fritz

The Impact of Maternal Environmental Factors on the Development of Autism Spectrum Disorder

ABSTRACT

Autism spectrum disorder (ASD) is characterized by difficulties in social interaction, communication, and behavior, with both genetic and environmental factors playing a role. The ongoing research focuses on understanding the complex interplay between genes and the environment, including maternal factors, to unravel ASD's origins. This exploration into specific triggers and protective elements is essential for informing preventive strategies and enhancing the overall well-being of individuals with ASD. I hypothesize that the environmental factors that the maternal parent has experienced cause a higher chance for the child to develop autism. Utilizing a PRISMA flow chart, articles from PubMed were screened for relevance to maternal parent and environmental factors during pregnancy. Out of 8 considered articles, 5 were selected based on alignment with the hypothesis, clarity on the maternal aspect, and reliable data. The chosen articles form a foundation for the subsequent analysis of maternal environmental factors and their impact. The findings underscore the multifaceted nature of environmental factors influencing ASD risk and the need for ongoing research to unravel the complexities involved. While the hypothesis was partially confirmed, the consensus across articles emphasizes the necessity for further exploration and concrete evidence to establish more definitive connections between maternal factors and ASD outcomes.

INTRODUCTION

Autism spectrum disorder (ASD) is a neurodevelopmental disorder defined by difficulties with social interaction, communication, and behavior. It affects people differently, resulting in a wide range of symptoms and skills. ASD is characterized by difficulties recognizing and interpreting social cues, difficulties creating and maintaining relationships, repetitive behaviors or restricted interests, and, in certain cases, sensory sensitivity. ASD affects a person's ability to communicate effectively, participate in normal social activities, and adapt to changes in routines or situations [1]. Individuals with ASD who get early diagnosis and intervention, as well as tailored support,

can optimize their strengths and overcome the challenges associated with the disease to lead productive lives [2].

The current standard understanding of ASD recognizes its complex and multifactorial nature, involving a combination of genetic and environmental factors [1]. While research has made significant strides in identifying genetic markers associated with ASD and understanding certain prenatal risk factors, much remains to be explored. One critical area is the intricate interplay between genes and the environment, as well as epigenetic influences, during crucial developmental stages. Further investigation is needed to uncover the specific environmental triggers and protective factors that may contribute to or mitigate ASD risk [1,2,5]. Additionally, enhancing early detection and intervention strategies, improving access to support services, and promoting inclusive communities are ongoing priorities in the effort to provide better outcomes and quality of life for individuals with ASD. The field of ASD research continues to evolve, emphasizing the importance of a holistic, multidisciplinary approach to unraveling the complexities of this condition and addressing the unmet needs of those affected.
I hypothesize that the environmental factors that the maternal parent has experienced cause a higher chance for the child to develop autism. The information gap in ASD revolves around understanding its exact causes, including the complex interaction between genetics and the environment [3]. While genetics plays a significant role, identifying specific genetic markers and mechanisms remains a challenge. Additionally, we need improved early detection methods, individualized treatments, and research on co-occurring conditions. Addressing the unique needs of adults with ASD, fostering societal acceptance, and tracking long-term outcomes are also vital areas for further exploration to enhance our knowledge and support for individuals with ASD [4].

METHODS

I used PubMed to identify the original 37,527 articles for this scoping literature review. The inclusion criteria for this literature review focused on articles that covered the maternal parent and the environmental risk and protective factors that affect the maternal parent in pregnancy when giving birth. The exclusion criteria was a main focus on genetics.

The PRISMA flow chart shows that I excluded two articles that focused mainly on genetic factors, which was outside of the scope of the literature review and one outside the year range I was researching for (2013-2023). I included articles that were directly related to my hypothesis, which were clear when talking about the maternal parent and environmental factors.

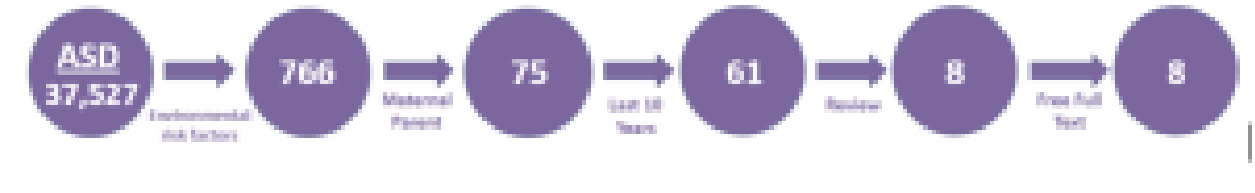

Figure 2. A PRISMA flow chart/diagram summarizes the screening process. It records the number of articles initially found, then records the number of articles per stage of the selection process.

RESULTS

The collective findings from the five articles converge on the understanding that ASD is a multifaceted condition arising from the intricate interplay between genetic predisposition and various environmental factors. A consistent theme across the research is the recognition of advanced parental age, both maternal and paternal, as a potential risk factor associated with an increased vulnerability to ASD in children [2,3]. Prenatal environmental factors take center stage in these discussions, encompassing aspects such as maternal immune activation, stress, drug exposure during pregnancy, and complications related to birth [1,2]. The articles also delve into the intricate relationship between nutrition and ASD, exploring the potential impact of nutritional elements like folic acid, vitamin D, iron, zinc, and copper, despite acknowledging some inconsistencies in the existing evidence [1,5].

Controversial associations, such as those between ASD and vaccination, maternal smoking, thimerosal exposure, and assisted reproductive technologies, are collectively dismissed based on evidence from systematic reviews and meta-analyses [1]. The researchers stress the importance of evidence-based approaches and advocate for continued research using advanced molecular biology and big data methods to unravel the complexities of gene-environment interactions in ASD development [3]. The articles highlight the need for a nuanced and holistic understanding of ASD, acknowledging the dynamic interplay between genetic and environmental components. They call for ongoing efforts in personalized risk assessments,

preventive measures, and potential therapeutic interventions based on a comprehensive comprehension of the intricate influences shaping neurodevelopmental disorders like ASD [2,3].

Articles from Boelte et al. and Modabbernia et al. provide a comprehensive overview of the association between nutrition and ASD, and while they share common themes, there are some differences in their emphasis and findings [2,3]. Modabbernia et al. reviews various nutritional factors and their potential links to ASD, including folic acid, vitamin D, protein, calcium, zinc, and omega-3 fatty acids [3]. It highlights the inconclusive nature of many studies, pointing out limitations such as assessing nutritional elements after ASD development [3]. The article suggests that while there are indications of deficiencies in certain nutrients, causal interpretations should be approached cautiously [3].

On the other hand, Boelte's article delves into specific nutrients like vitamin D, iron, zinc, and copper, emphasizing the role of maternal nutrition during pregnancy [2]. It discusses the potential impact of short interpregnancy intervals on autism risk and explores the association between vitamin D deficiency and ASD, with evidence suggesting that deficiencies in early development may contribute to the etiology of autism [2].

While both articles agree on the need for caution in interpreting findings, they slightly differ in their emphasis. Modabbernia et al. provides a broader overview of various nutritional elements, highlighting the inconclusive nature of many studies [2]. Boelte et al. on the other hand, focuses more specifically on individual nutrients and their potential roles, with a notable emphasis on protective factors [3]. Ultimately, the articles collectively suggest that there is ongoing research and debate in understanding the nuanced relationship between nutrition and ASD, emphasizing the need for further investigation, well-designed studies, and a holistic approach to unraveling these complex associations [2,3].

Doi et al. explores the Developmental Origins of Health and Disease (DOHaD) theory, focusing on its application to ASD [4]. The DOHaD theory suggests that environmental factors during prenatal and postnatal development can induce predictive adaptive responses, impacting future health outcomes [4]. Specifically, the review delves into the prenatal

environment, highlighting factors such as maternal immune activation (MIA), stress, and drug exposure as potential risk factors for neurodevelopmental disorders (NDDs) including ASD [5]. It discusses how inflammatory responses triggered by infections or autoimmune diseases in pregnant mothers can affect fetal brain development and increase the risk of ASD. Additionally, the article explores the impact of drug exposure, such as thalidomide and valproic acid, on neural development and the potential link to ASD. The discussion extends to the effects of preterm birth, low birth weight, and intrauterine growth restriction on the onset of ASD and other NDDs [4]. Overall, the review emphasizes the importance of understanding the prenatal environment and its influence on fetal development to inform preventive medicine and therapeutic interventions for individuals at risk of ASD and related disorders.

DISCUSSION

In summary, these articles collectively emphasize the intricate interplay of genetic and environmental factors in ASD. Advanced parental age, prenatal environmental factors, and nutritional elements are recognized as key contributors, calling for a holistic understanding of ASD etiology. The dismissal of controversial associations underscores the importance of evidence-based approaches, while ongoing research, utilizing advanced methodologies, is encouraged. The nuanced perspectives from Modabbernia et al. and Boelte et al. highlight the ongoing debate around the nutritional dimensions of ASD, emphasizing the need for well-designed studies [2,3]. The focus of Doi et al. on the DOHaD theory adds depth, emphasizing the critical influence of the prenatal environment and the potential for tailored preventive and therapeutic interventions [4]. In navigating the complex realm of ASD, these insights underscore the ongoing pursuit of knowledge and precision in unraveling the mysteries of gene-environment interactions in neurodevelopmental disorders.

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Rohan Gazula

The Effect of Urbanization-Related Built Environment on Global Cardiovascular Health

ABSTRACT

Background: This review focuses on the global significance of cardiovascular diseases (CVDs) and the determinants of a higher CVD risk. Due to the lack of existing reviews that have explored these determinants on an international scale, this review aims to examine the effect of urbanization, including neighborhood characteristics, air pollution, and fast food accessibility worldwide.

Methods: This literature review searched Google Scholar and Pubmed for academic articles published since 2015. This search was focused on original research that evaluated the correlation between neighborhood features, air pollution, and fast food on CVD risk, and the search ultimately concluded with five relevant articles which met specific inclusion and exclusion criteria.

Results: Violent crimes, noise pollution, and proximity to major roads were all associated with increased risk of CVD, with noise pollution and proximity to roads resulting in 225% and 286% increases in risk of CVD and myocardial infarction respectively. Increased air pollution was also associated with increased odds of CVD, as increasing NO2 content by 10 parts per billion resulted in a diabetes odds increase of 16%. The correlation between fast-food restaurant proximity and CVD risk was inconclusive.

Discussion: Correlations between neighborhood features and air pollution with CVD risk are evident from the results, but this review is limited by its scope, considering two urban areas per correlation. It is in lawmakers' best interest to attempt to pass laws to lessen the effect of noise pollution and improve proximity to public transit, as these factors will help lessen CVD risk. Further research is required to explore these correlations on a larger, more global scale, and there is a need for more longitudinal studies concerning fast food and CVD risk.

INTRODUCTION

Non-communicable diseases (NCDs) remain the leading cause of death across the world, and their global mortality rates are steadily increasing [1]. In this global health threat, cardiovascular diseases (CVDs) consistently play the biggest role, with overall cases doubling in the last 20 years [2]. The

rising prevalence of CVDs not only results in more danger to public health, but it also has negative financial implications; according to the World Heart Federation's estimates, "the global cost of CVD will rise from roughly $863 billion in 2010 to $1044 billion in 2030" [2]. As the impact of CVDs remains a significant issue in our ever-changing world, it is important to better understand how health determinants may affect the risk of CVDs and how we may improve our strategies to address these diseases.

In recent scientific literature, several reviews have been conducted to help understand the determinants for CVDs in isolated groups, often citing lifestyle behaviors, access to healthcare, and financial situation as common areas for improvement [2, 3]. Therefore, these factors are important in assessing the risk for CVD in any population. However, few reviews have analyzed the determinants of CVDs on an multinational scale, comparing different populations in their respective environments. When looking on an international level, across nations and cultures worldwide, one trend is clear: the world continues to urbanize. As a result, the effects of urbanization require consideration and review in the realm of CVD risk.

Increased urbanization ultimately has several important implications concerning the risk of CVDs, these being notably increasing air pollution, noise pollution, and fast food accessibility and consumption. Due to these imminent trends, it is paramount to understand how these determinants affect CVD risk on an international basis. For instance, the rise in global urbanization is clear and predictable. According to the United Nations 2018 World Urbanization Prospects, 55% of the world's population already lives in urban areas, and by the year 2050, this number is estimated to rise to 68% of the world's population [4]. Increased urbanization follows suit with an increase in fast food restaurant density and consumption, as the proportion of fast-food related calories consumed by children and adolescents increased from 10.6% in 2009–2010 to 14.4% in 2017–2018 [5]. Increased urbanization also generally involves an increase in air and noise pollution [4]. Considering these effects will occur on a worldwide scale, the aim of this literature review is to fully assess the effect of the built environment on cardiovascular health on an international level; specifically, this review covers the worldwide effect of air pollution, infrastructure, and fast food on the risk of CVD.

METHODS

In this literature review, Google Scholar and Pubmed were both searched for recent academic articles published in 2015 or later. Academic publications were deemed viable for review under the condition that they involved original research; inclusion criteria required a study design of investigation of quantitative correlation between exposures and outcomes. Inclusion criteria also included exposures of measured or identified facets of built environment, including neighborhood factors, fast food accessibility, and air pollutant measures, along with outcomes of measured cardiovascular health. Exclusion criteria for this literature review excluded any design of comprehensive literature review papers associated with built environment and cardiovascular health. Key search terms were also used in each search engine's query—specifically, the terms built environment, fast food restaurant density, air pollution, cardiovascular disease, cardiovascular health, and CVD. Data was then obtained from a series of five relevant articles which matched the previous criteria, with a focus on the correlation between built environment and cardiovascular health on a global scale. Finally, the review considered other reputable sources to provide necessary background information about global trends and cardiovascular health.

RESULTS

Neighborhood Characteristics and Infrastructure

One study carried out by Chum and O'Campo attempted to comprehensively investigate the link between neighborhood factors and cardiovascular diseases in Toronto, Canada discovering that—when adjusted for neighborhood income and environmental characteristics—their data suggest exposure to violent crimes, environmental noise, and proximity to major roads were associated with increased odds of CVDs [6]. Specifically, researchers were able to discover that individuals living in the quartile with the lowest rate of violent crime were on average 13% less at risk for myocardial infarction (MI) [6]. The researchers also discovered that respondents who reported "strongly agree" to being disturbed by noise at home were on average 225% more at risk of CVD while those who responded "disagree" were on average at 21% lower risk of CVD [6]. Furthermore, individuals living within 100m of a major road are on average 286% more at risk for MI [6]. In a different study, which investigated the effect of neighborhood walkability on hypertension and diabetes risk in Ontario, Canada, Howell et al. discovered that individuals living in the lowest

walkability areas were 28% more at risk for hypertension and have 25% greater odds of having diabetes compared to those living in the highest walkability areas [7]. Moreover, in another study conducted by Lee et al. in Korea, a farther distance from public transit was associated with increased odds of heart attack [8], as the second farthest and farthest thirds from public transit were at a 41% and 22% greater risk of MI when compared to the group closest to public transit [8].

Air Pollution

Alongside walkability, Howell et al. also attempted to investigate the effect of traffic-related air pollution on hypertension and diabetes risk. The study discovered that traffic-related air pollution can predict the risk of hypertension and diabetes, associating higher traffic-related air pollution with higher odds of both diseases' risk; for every 10 parts per billion (p.p.b) unit increase in nitrogen oxide (NO_2), the odds of having hypertension increase by 2%, and the odds of diabetes increase by 16% [7]. In another study conducted in the United Kingdom, Wang et al. discovered a 67% higher frequency of incident heart failure (HF) in the highest air pollution quintile when compared to the lowest air pollution quintile [9].

Fast Food

Poelman et al. attempted to analyze the correlation between fast-food restaurant density and risk of CVDs in the Netherlands [10]. In street network-based buffers of 500m, 1000m, and 3000m around residential addresses, the study discovered that the frequency of stroke, coronary heart disease (CHD) and HF was higher within 500m buffers with at least one fast-food restaurant, as well as within 1000m buffers [10]. In urban areas, individuals living in 500m buffers with 2 fast food restaurants are 6% more at risk of stroke, 13% more at risk of CHD, and 15% more at risk of HF [10]. However, in Korea, Lee et al. discovered that living in areas with high concentration of fast-food restaurants was associated with a decreased risk of stroke, as the second highest and highest fast food density thirds were 42% and 36% less at risk of stroke, even when accounting for multivariable analysis [8].

Table 1: Article Descriptions

Article	Purpose	Independent Variable	Dependent Variable
Cross-sectional associations between residential environmental exposures and cardiovascular diseases	Attempted to comprehensively investigate the link between various neighborhood factors and CVDs in Canada.	Violent crimes Housing requiring major repairs Traffic exposure Noise level perception Neighborhood socioeconomic status	Self-reported history of physician diagnosis of myocardial infarction (MI), angina, coronary heart disease (CHD), stroke, and congestive heart failure (CHF)
Interaction between neighborhood walkability and traffic-related air pollution on hypertension and diabetes: The CANHEART cohort	Attempted to investigate the effect of neighborhood walkability and traffic-related air pollution on hypertension and diabetes risk in Canada.	Walkability exposure Traffic-related air pollution	Hypertension Diagnosis Diabetes Diagnosis

Objectively Measured Built Environments and Cardiovascular Diseases in Middle-Aged and Older Korean Adults	Attempted to assess the association between the built environment and CVDs.	Population density Fast-Food restaurant density Proximity to Public Facilities, Parks, and Public Transit	A self-reported history of a physician's diagnosis of conditions such as hypertension, diabetes, dyslipidemia, stroke, MI, or angina
Joint exposure to various ambient air pollutants and incident heart failure: a prospective analysis in UK Biobank	Aimed to investigate the relationship between long-term exposure to ambient air pollutants and incidence of heart failure in the United Kingdom.	Air Pollution Exposures	Self-reported information and hospital inpatient records of prevalent heart failure and incident heart failure
Relations between the residential fast-food environment and the individual risk of cardiovascular diseases in the Netherlands: A nationwide follow-up study	To analyze the correlation between fast-food restaurant density and risk of CVDs in the Netherlands.	Fast-Food Outlet Density (FFD)	Incidence of CVD, CHD, stroke, and CHF

DISCUSSION

Findings and Interpretations

After analyzing the data, it is apparent that the built environment does correlate with CVD risk. A primary aspect of an individual's built environment

is the residential neighborhood and its characteristics or infrastructure. In a study based in the urban environment of Toronto, Canada, researchers found that less exposure to crime was linked with a lower risk for MI [6]. In the same study, researchers also discovered that increased noise pollution and proximity to a major road was correlated with a significantly increased risk of CVD [6]. Another study based in Ontario, Canada also discovered that lower walkability was associated with higher odds of hypertension and diabetes [7]. In a different study, it was discovered that individuals living farthest away from public transit had a greater risk of MI in the urban Gyeonggi Province in South Korea [8]. These results all generally meet the expectations of current scientific literature regarding built environment and CVD risk, and they reaffirm the notion that built environment—across the world—is in fact a factor which has significant implications on cardiovascular health [11].

Air pollution was further found to be a driving force behind the risk of CVD in urban areas, with correlations confirmed on a worldwide basis by both studies in Ontario, Canada and the United Kingdom. In the Ontario-based study, even a miniscule increase in the ppb content of NO2 was associated with a direct increase in odds of hypertension and the odds of diabetes, and in the United Kingdom, higher air pollution was linked with a significantly higher frequency of HF [7, 9]. These results confirm the existing literature that suggests that air pollution exposure is associated with cardiovascular disease risk [9].

The correlation between fast-food restaurant density and CVD risk was not nearly as straightforward. In the Netherlands, a study discovered that the proximity of one's residence to a fast-food restaurant was correlated with marginally increased risk of stroke, but a significantly higher risk of CHD and HF [10]. However, a study in Korea determined that living in an area with high concentration of fast-food restaurants was correlated at significantly lower risk of stroke, even when accounting for several other factors [8]. These conflicting results are surprising, as current scientific literature suggests that fast food's high saturated fat content can increase diabetes and obesity risk while its high salt content can raise blood pressure, thereby negatively impacting cardiovascular health [12]. While this trend holds true in the Dutch study, it may not hold entirely in the Korean study because

fast-food density may not necessarily be associated with fast-food consumption among Koreans [8].

Implications and Limitations

As evidenced by the results, there is clearly a correlation between certain neighborhood factors and CVD and between air pollution and CVD. These correlations are evident among different populations on different continents, ranging from Canada, the United Kingdom, and Korea. It is safe to assume, therefore, that these correlations may be present on a global scale and require the attention of policymakers. When factoring the rapid rate of urban sprawl, many of these neighborhood factors will become more exaggerated over time [4]. Overall, these correlations may appear evident among two populations, but the claim that these correlations appear on a global scale is limited by the scope of the study, having only considered two urban populations per correlation. Furthermore, when considering fast food, it appears that fast-food restaurant density alone is not a substantial prerequisite for increased CVD risk among all populations, and again these claims are limited by the scope of the study. However, the findings of this review do not call into question the original belief that increased fast-food consumption is related to increased CVD risk [12].

One potential explanation for these correlations could be exercise as a mediating role. For example, in neighborhoods with lower crime rate and higher walkability, individuals would likely feel a greater propensity to exercise, which has been linked with improved cardiovascular health [13]. Pointing out such trends of which specific factors lead to a risk of CVD is nonetheless important, as it increases policymakers' and individuals' awareness of the risk factors behind CVD and allows for more efficient resource allocation, which can improve healthcare costs and facilitate research in specific areas.

Applications and Future Research

Considering the correlations between neighborhood factors and air pollution with heightened CVD risk, it is in legislators' best interest to attempt to curb the effect of these factors. For example, lawmakers can opt to bring about stricter laws regarding noise pollution and improving accessibility to public transit, which have a significant effect on citizens' cardiovascular health. Policymakers may also find it in their best interest to invest in stricter

environmental protection laws, especially concerning air pollution as our world continues to urbanize. Curtailing the effect of air pollution will significantly improve citizens' cardiovascular health, as long-term air pollutant exposure is a significant threat to public health worldwide [11].

Given the scope of this review, exploring the effect of neighborhood factors and air pollution in two urban areas each, further research is required to confirm claims that these correlations with CVD risk are in fact global trends. Potential grounds for future research would include a larger sample size of urban areas to consider, to better determine the worldwide presence of such trends. In addition, due to the unexpected finding regarding the correlation between fast-food and CVD, there is a need for further longitudinal studies attempting to explore the relationship between these two variables in the future.

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Monica Gupta

Association Between Residential Noise Pollution and Cardiovascular Disease

ABSTRACT

Background: Residential noise pollution is identified as a critical environmental factor with potential implications for cardiovascular health, driven by global urbanization and increased transportation activities. This review aims to address critical gaps in understanding the correlation between residential noise pollution and cardiovascular health by investigating existing evidence and highlighting the implications for public health. The objectives are outlined, focusing on the research question guiding the inquiry: how does chronic exposure to residential noise contribute to the development and progression of cardiovascular diseases?

Methods: PubMed and Google Scholar with specific search terms. Inclusion and exclusion criteria are established to ensure the relevance of the selected articles, focusing on clinical studies within the last five years that specifically address residential noise pollution and cardiovascular disease.

PubMed and Google Scholar were searched using the key terms: "Residential Noise Pollution", "Cardiovascular Disease Risk", "Traffic Noise", and "CVD." Inclusion criteria focused on clinical studies within the last five years that specifically address residential noise pollution and cardiovascular disease. Exclusion criteria was a study design of a systematic review or meta-analysis.

Results: Excessive noise is identified as a significant threat to mental, behavioral, and neurological well-being, contributing to global fatalities and the disease burden. The impact on heart health is highlighted, particularly among women facing high noise levels during both day and night. Prolonged exposure, especially in areas with louder road traffic noise, escalates cardiovascular risks, with aircraft noise above 45 dB emerging as an additional contributor.

Discussion: The key findings underscore a significant association between residential noise pollution and cardiovascular disease, emphasizing its multifaceted impact on overall health. The differential impact on men and women is highlighted, aligning with current literature, and emphasizing the global significance of noise as a health hazard. The unexpected decrease in risk among men during the day prompts exploration into potential protective

factors or coping mechanisms. Future applications may involve targeted interventions and public health campaigns to mitigate noise exposure, especially in vulnerable populations.

INTRODUCTION

Significance of the Problem

Residential noise pollution has emerged as a critical environmental factor with potential implications for cardiovascular health. The growing urbanization and increased transportation activities worldwide have contributed to elevated levels of noise exposure in residential areas, prompting concerns about its impact on cardiovascular diseases (CVD) risk. Cardiovascular diseases, including stroke, myocardial infarction, and ischemic heart disease, represent significant contributors to global morbidity and mortality. The World Health Organization (WHO) states that CVD is the leading cause of mortality worldwide, accounting for approximately 17.9 million deaths annually [1]. As urbanization continues, understanding the intricate links between residential noise exposure and cardiovascular outcomes becomes crucial for developing effective preventive measures. Poor health outcomes associated with CVD, such as diminished quality of life, increased healthcare costs, and premature mortality emphasize the urgency of addressing the impact of residential noise pollution on public health. Despite growing awareness, existing data and scientific methodologies have proven insufficient in providing a clear correlation between residential noise and CVD outcomes.

Objectives

This literature review aims to investigate and synthesize existing evidence on the association between residential noise pollution and CVD, addressing critical gaps in understanding and highlighting the implications for public health. The overarching research question guiding this inquiry is: how does chronic exposure to residential noise contribute to the development and progression of cardiovascular diseases? It also aims to enhance the collective understanding of the implications of residential noise exposure to public health.

METHODS

Search Strategy: The search engines used were PubMed and Google Scholar. The key search terms used were “Residential Noise Pollution”, “Cardiovascular Disease Risk”, “Traffic Noise”, and “CVD”.

Inclusion and Exclusion Criteria: The inclusion factors were if the article was a clinical study, written in the last 5 years (2018 – 2023), was about residential noise pollution and cardiovascular disease. All systematic reviews and meta-analyses were excluded.

RESULTS

Excessive noise poses a significant threat to mental, behavioral, and neurological well-being, contributing to 3% of worldwide fatalities and comprising 10% of the global disease burden [2]. Particularly noteworthy is its impact on heart health, especially among women facing high noise levels during both day and night, showing an 8% higher risk of hospitalization for heart problems [2]. Further analysis reveals that women exposed to nighttime noise experienced a 0.5% higher risk, while daytime noise exhibited a 0.4% increase [2]. Intriguingly, during the day, men showed a non-statistically significant 6% decrease in risk [2].

Table 1: Risk Associations between Vehicular Traffic Noise Exposure and Cardiovascular Diseases

Noise Exposure Level	Mortality Risks	Hospitalization Risks
Medium-high and high levels	Excess risks for CVD (general pop.)	Increased risks for women (cerebrovascular, ischemic diseases)
High levels (women)	Excess risks for CVD	Increased risks for cerebrovascular, acute MI
High levels (men)	Decreased risks for certain conditions	Not specified

Drawing insights from the UK Biobank's extensive data involving over 370,000 participants, it was identified that residential road traffic noise exceeding 65 dB[A] was associated with subtle changes in blood pressure [3]. Notably, individuals not taking hypertension medication displayed a positive association between road traffic noise and self-reported hypertension, particularly in the 60-65 dB[A] range [3].

Table 2: Road Traffic Noise and Cardiovascular Disease Risk Factors in UK Biobank

Exposure Level (dB[A])	Change in Blood Pressure
>65	Slight changes
>65 (adjusted)	Significant associations
60-65	Positive association with self-reported hypertension (non-medicated individuals)

In Montreal, Canada, health data analysis from adults aged 45 and above unveiled a significant link between increased environmental noise levels and a heightened risk of ischemic stroke, with an 8% increase for every 10-decibel rise in the 24-hour equivalent noise level [4]. Additionally, there was an association found between higher levels of total environmental noise and increased myocardial infarction (MI) incidence, affecting 3.8% of the 1,065,414 individuals studied examining the entire adult population of Denmark from 1995 to 2017 [5,6].

Living in places with louder road traffic noise on one side of the building is associated with a higher risk of heart problems, including ischemic heart disease, heart attack, chest pain, and heart failure [6]. The risks escalate with a 10 dB higher noise level over a 10-year period [6]. Additionally,

exposure to aircraft noise above 45 dB appears to increase the chances of heart attack and heart failure [6].

DISCUSSION

The key findings of this research reveal a significant association between residential noise pollution and cardiovascular disease, underscoring the multifaceted impact of excessive noise on overall health. Notably, women exposed to high noise levels during both day and night exhibited an 8% higher risk of hospitalization for heart problems, with nighttime noise contributing to a 0.5% higher risk and daytime noise to a 0.4% increase [2]. Interestingly, men showed a non-statistically significant 6% decrease in risk during the day [2]. Moreover, the study, drawing insights from the UK Biobank and health data analysis in Montreal, Canada, supports the correlation between elevated noise levels and cardiovascular risks, including changes in blood pressure, self-reported hypertension, and an increased risk of ischemic stroke and myocardial infarction [5]. The risks further escalate with prolonged exposure, particularly in locations with louder road traffic noise, and aircraft noise above 45 dB emerges as an additional contributor [6].

It is evident that residential noise pollution constitutes a substantial risk factor for cardiovascular diseases, with the differential impact on men and women warranting further investigation. The results align with current literature, emphasizing the global significance of noise as a health hazard. The unexpected decrease in risk among men during the day prompts exploration into potential protective factors or coping mechanisms. Future applications of this data may involve targeted interventions to mitigate noise exposure, especially in vulnerable populations. Understanding the nuances of noise-related health risks can inform public health policies aimed at minimizing these adverse effects and promoting cardiovascular well-being.

However, it is crucial to acknowledge certain limitations in this study. The observed gender differences and non-statistically significant findings among men during the day necessitate additional research to clarify the underlying mechanisms. Moreover, the study primarily relies on observational data, limiting the ability to establish causation definitively. Future studies could incorporate more extensive datasets, diverse populations, and experimental designs to enhance the robustness of our conclusions.

In light of these findings, implementing noise reduction strategies in residential areas, particularly those with high traffic density, and incorporating noise mitigation measures in urban planning is recommended. Public health campaigns to raise awareness about the cardiovascular risks associated with residential noise pollution, especially among women, could further contribute to preventive efforts. Additionally, targeted interventions addressing specific sources of noise, such as vehicular traffic and aircraft, may prove effective in reducing the burden of cardiovascular diseases linked to environmental noise exposure.

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Shriya Havangi

Effects of Inhaling Secondhand Smoke on Cardiovascular Health

ABSTRACT

Introduction: Secondhand smoke is hazardous to an individual's health and has caused more than 41,000 deaths in a year. However, the relationship between secondhand smoke and its prognosis to heart failure is still not answered. The aim of this paper is to investigate the effects of inhaling secondhand smoke on cardiovascular health.

Methods: Relevant research articles were found and reviewed through PubMed and Google Scholar, which were written from 2003 to 2023. Key search terms used were "secondhand smoke", "cardiovascular health", "inhalation", "United States", and "environment".

Results: The results revealed that inhaling secondhand smoke results in increased rates of mortality, a negative effect on heart rate variability, and an increased chance of cardiovascular disease. The effects of exposure to secondhand tobacco smoke on cardiovascular health leads to myocardial infarction, peripheral artery disease, and transient ischemic attack.

Discussion: These findings emphasize the dangerous effects of inhaling secondhand smoke, and how it is a hazardous public health issue. The effects of inhaling secondhand smoke on mortality risk can be as dangerous and likely as first hand smoke exposure.

INTRODUCTION

Secondhand smoke (SHS) is constantly in the air and inescapable in any environment like houses, parks, and offices. Based on surveys done between 2010 and 2018, the global prevalence of SHS exposure in 142 countries was 62.9% (95% confidence interval 61.7& – 64.1%) on one or more days [1]. The number of people who do not smoke but are exposed to SHS in the US has declined, however, the gaps in SHS exposure still remain [2]. SHS contains many toxic chemicals including formaldehyde, benzene, vinyl chloride, arsenic ammonia and hydrogen cyanide [3]. Regardless of short-term or long-term exposure to SHS, there is no risk-free level of exposure to SHS, which inturn can increase the chances of an individual's health being affected when it's inhaled [3]. SHS is an extreme health hazard and has caused more than 41,000 deaths per year [3]. SHS impacts many parts of the body and also can cause coronary heart disease (CHD). SHS

causes about 34,000 early deaths among nonsmokers, due to CHD, each year in the United States. [4] Thus, SHS has become a prevalent factor in causes of deaths among many people in the United States. Additionally, secondhand tobacco smoke (SHTS) is a type of SHS which leads to morbidity and premature mortality among people worldwide [5]. The people who's CVH is affected by any SHS also can include other social determinants like their race, ethnic minorities, and lower socioeconomic status [5].

The impact of SHS on the cardiovascular system was recognized about 20 years ago [6]. Based on a previous study which surveyed US residents, SHS has a clinically important effect on sensitivity to cardiovascular disease [8]. CVDs can be related to atherosclerosis, myocardial infarction, arrhythmia, and arterial stiffness [8]. These CVD can be caused by minutes or hours of SHS exposure is almost 80% to 90% greater than first hand smoke [8]. In addition, patients who already suffer with underlying health conditions also can have a great impact when SHS is inhaled.Thus, it is important to know the effects of the inhalation of SHS from the environment on cardiovascular health (CVH). However, the association between SHS and prognosis of heart failure is still unclear [7]. To decrease the risks to CVH, the impacts of SHS on the cardiovascular system need to be identified. Thereafter, effective interventions can be created to decrease mortality rates and risk for developing Cardiovascular Disease (CVD). Therefore it is important to examine the relationship between inhaling SHS and the CVH risks that become associated with it.

The present study is aimed to determine how the inhalation of SHS that is in the environment affects CVH. The study aims to identify the various CVDs that are caused through SHS and examine how prevalent it is to obtain those diseases through SHS. Additionally, the study aims to examine the trends of mortality rates due to these CVDs.

METHODS

PubMed and Google Scholar were searched using the key search terms "secondhand smoke", "cardiovascular health", "inhalation", "United States", and "environment" to conduct a scoping literature review.

The inclusion criteria were articles written in 2003-2023 and conducted in the United States. Exclusion criteria included papers that gathered data from

countries outside of the U.S., individuals under 17 years, studies done on pregnant or severely sick individuals, and literature reviews.

RESULTS

A study was conducted where 88 patients were exposed to household SHS and 484 weren't, after a mean follow up 9.2 years later, there were 72 deaths in the exposed group and 403 deaths in the unexposed group [7]. After adjusting other social determinant factors like demographic variables and socioeconomic variables, household SHS exposure was associated with a 43% increase in mortality risk (Hazard Ratio: 1.43, 95% Confidence Interval: 1.10 – 1.86, $p = 0.007$) [7]. The associations between household SHS and mortality risks didn't significantly differ between people who have never smoked and ex-smokers ($p > 0.1$) [7].

Another study was conducted with flight attendants to analyze the effects when they were exposed to secondhand tobacco smoke [5]. Secondhand tobacco smoke (SHTS) is a type of SHS which leads to morbidity and premature mortality among people worldwide [5]. Other social determinants such as: their race, ethnic minorities, and lower socioeconomic status, can also impact the level to which SHTS affects people [5]. The effects of SHTS exposure on CVH led to myocardial infarction (MI) (Odds Ratio (OR) = 1.40, 95% CI: 1.04, 3.27), peripheral artery disease (PAD) (OR = 1.27, 95% CI: 1.00, 1.97), and transient ischemic attack (TIA) (OR = 1.11, 95% CI: 0.84, 1.68) [5]. Workers, like the flight attendants, exhibited low morbidity when exposed to SHTS because being healthy is required to maintain employment [5].

A study panel conducted among residents in the United States analyzed the association between 15 – 240 minute SHS fine particulate matter (PM2.5) movement and signs of heart rate variability (HRV) [9]. The associations between PM2.5 moving average exposure decreases and increases HRV regardless of short term or long term SHS exposure [9]. When an individual was exposed to 15 minutes (short-term) of moving average PM2.5 in SHS and 240 minutes (long-term) of moving average PM2.5 in SHS their HRV decreased [9]. However, when an individual was exposed to 120 minutes of moving average PM2.5 in SHS their HRV increased [9]. When people are exposed to SHS for any amount of time, it in turn has a negative association with CVH [9].

Finally, a survey which was conducted analyzed the biomarkers which were affected by SHS that leads to CVD in never smoking adults [10]. After analyzing blood samples, people who were exposed to SHTS had increased amounts of cotinine, found in tobacco leaves, in their blood [10]. People who had: no cotinine (a metabolite of nicotine) in their blood increased by 5%, low cotinine in their blood increased by 18%, high cotinine in their blood increased by 56% [10]. Regardless of the high and low groups of cotinine, both groups had higher levels of fibrinogen and homocysteine, biomarkers of CVD [10]. Having a low-level exposure to SHS has a clinically significant effect on the vulnerability to CVD since the levels of cotinine won't be as high meaning that fibrinogen and homocysteine levels won't be as significantly affected to increase the chances of CVD [10]. Exposure to SHS is suitable to be a crucial avoidable cause of CVD in the United States population [10].

DISCUSSION

There has been extensive research on how inhaling SHS affects cardiovascular health. The results indicate that household SHS exposure was associated with a higher risk of death among heart failure patients [7]. Additionally, when other social determinant factors were considered as variables, the mortality risk increased by 43% [7]. SHTS also leads to MI, PAD, and TIA, which increases the chance of morbidity and mortality [5]. Additionally, when an individual is exposed to SHS it has a negative impact on their heart variability and can cause the rhythms to increase or decrease [9]. The CVD's that occur from SHS inhalation result from cotinine levels in blood increasing, which in turn increases the levels of fibrinogen and homocysteine [10].

It is suggested that the effects of household SHS on mortality risk can be as great as first hand smoke exposure [7]. These results should be taken into account when considering how SHS can be just as harmful as first-hand smoke. This data also suggests that inhaling SHS is a dangerous public health issue. Additionally, these results provide an insight into the relationship between how elevated CVD biomarkers, fibrinogen and homocysteine, can lead to CVD such as MI, PAD, and TIA [7,10]. These results build off the existing knowledge of how SHS can affect certain organs in the body, specifically the heart.

The limitation of this paper is that due to this being a scoping review, not all research papers were able to be analyzed in this paper. Additionally, the effects of inhaling e-cigarette smoke on CVH was not discussed in this paper. Therefore comparing this to the effects of SHTS can't fully apply to e-cigarette smoke.

Further research should focus on how SHS exacerbates health disparities in individuals who already have CVD or other existing health conditions. Through this research the mortality rates can be observed to see if there is a significant impact. Additionally future research should analyze the effectiveness of smoke free laws, and if it positively or negatively affects CVH.

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Nitya Kolli

Impact of the Environment on Cardiovascular Disease

ABSTRACT

An average of 17.9 million deaths occur yearly due to cardiovascular disease, making it the leading cause of death worldwide. The purpose of this scoping literature review is to determine the impact of healthy food location and access to green spaces on cardiovascular disease. The search terms environment, heart health, cardiovascular disease, and diets were used to search PubMed and Google Scholar to find articles that met the inclusion and exclusion criteria. Cardiovascular disease (CVD) risks were low in areas where there was high access to healthy food options and walkable areas. Access to physical activity areas positively influenced women by 22% and men by 16%. Green spaces are associated with reducing heart disease such as a 7% decrease in acute myocardial infarction, a 6% decrease in heart failure, and a 10% decrease in cardiovascular mortality. Access to healthy food and green spaces for physical activity can influence CVD rates. Policymakers should advocate for healthier food options and support businesses that promote active lifestyles.

INTRODUCTION

Cardiovascular disease (CVD) continues to be the main cause of mortality in the world, resulting in an average of 17.9 million deaths per year [1]. The buildup of fatty plaque, also known as atherosclerosis, in the arteries causes CVD. It leads to decreased blood flow and oxygen to the heart or brain and is caused by factors such as high blood pressure and high cholesterol. This disease can engender a variety of poor health problems such as heart attack, stroke, and heart failure. Understanding the impact of an individual's surroundings and build, which is the human-made conditions, the environment has been neglected by many researchers when finding the causes of cardiovascular disease. Due to cardiovascular disease's commonality in many individuals, it is essential to address the disease and its prevention because of its significant impact on health care and the lifestyle of many. It is important to note the main influences of the disease to recognize its primary causes and risks. Recent studies have shown that the environment has a large impact on the management of cardiovascular health and CVD risks [2].

A built environment and neighborhood can cause some locations and individuals to be more affected by CVD than others. The environment has a large influence on cardiovascular health through access to healthy foods and the availability of green spaces. Improving the built environment and implementing certain policies related to increasing the accessibility of healthy foods and physical activity facilities can improve cardiovascular health. Having easier access to green spaces can also allow an individual to have greater access to facilities to prevent health issues. It was found that people living in areas with a high density of greenery had 37% fewer chances of being hospitalized for CVD [1]. Residing in areas with a greater availability of green spaces has effects on one's health. In a longitudinal study conducted in Ontario, Canada, greater degrees of greenness were linked to a lower risk of cardiovascular disease [2]. Access to healthy food locations also has an immense impact on an individual's health. Poor diets are a major cause of cardiovascular disease in people [4]. A person's diet can have negative or positive effects on their health. A person's diet has a great influence on cardiovascular diseases such as blood pressure and obesity [3]. Not having access to healthy foods and places to eat can cause an individual to lean towards unhealthy foods, ultimately leading to a high risk for cardiovascular health. Changing the availability and access to foods within an environment would increase the health of a person's diet. The presence of grocery stores, fast food restaurants, and healthy restaurants influences the design and diets of people in the built environment [4]. The density of essential resources in built environments has a large impact on the influence of cardiovascular disease among individuals.

The purpose of this study is to determine how the density of areas with healthy food locations and access to parks and green spaces affect cardiovascular disease. By analyzing the information gathered we can understand which types of environments result in lower or higher rates of cardiovascular disease among its population of individuals. This research allows healthcare professionals to create policies to promote healthier environments and ways of living.

METHODS

PubMed and Google Scholar were searched using the search terms environment, cardiovascular disease, heart health, and diets to conduct a

scoping literature review. Inclusion criteria were articles with participants between the ages of 20 and 80 years, published in English, and written from 2000 to 2023. Exclusion criteria were articles written in languages other than English, written before 2000, and systematic reviews.

RESULTS

In a study analyzing the built environment relationship to blood pressure changes, individuals living in high-walkable neighborhoods were tied with decreased systolic and diastolic blood pressure, and environments with a high density of fast-food restaurants and low walkability were related to increases in blood pressure [5]. Zheng et al. examined the relationship between access to healthy food to CVD risks and concluded that CVD risks were low in areas where there was a high mass of healthy food options [6]. Areas where the density of fast food places is high tend to make more people lean towards unhealthy diets, increasing their risk of CVD [6].

Unger et al. demonstrated that men and women have different cardiovascular health (CVH) based on environmental factors [7]. They found that having healthy food available and access to physical activity allowed women to have a greater chance of having better CVH than men [7]. They emphasized that the environment where an individual resides has a great effect on their chance of having an ideal CVH [7]. In a given environment where there is greater access to physical activity resources, men’s health is positively impacted by 16% while it is 22% for women [7]. Having residential greenness can have a greater impact on the chances of developing heart disease [8]. Living in areas with green spaces is linked to a 7% decrease in acute myocardial infarction (AMI), a 6% decrease in heart failure (HF), and a 10% decrease in cardiovascular mortality among the adult population [8]. The study also alludes to the fact that positive impact is observed only in the cardiovascular health of people without preexisting conditions of HF and AMI [8].

Table 1: Article Summaries

Article	**Purpose**	**Independent Variable**	**Dependent variable**
Built environment and changes in blood pressure in middle aged and older adults	Analyzes the built environment and resident lifestyle choices in relation to blood pressure changes	– Neighborhood walkability – Density of fast food restaurants	– Changes in systolic and diastolic blood pressure
Scientometric Analysis of The Relationship between a Built Environment and Cardiovascular Disease	To understand the relationship between the built environment and cardiovascular disease and the preventative care needed for CVD.	– Accessibility to healthy foods – Accessibility of green spaces	– CVH Cardiovascular risk factors
Association of Neighborhood Characteristics with Cardiovascular Health in the Multi-Ethnic Study of Atherosclerosis (MESA)	To examine the influence of neighborhood environments on cardiovascular disease and the disease's risk factors.	– favorable food stores – Healthy food availability – Walking environment	– CVH score

Residential Greenness and Cardiovascular Disease Incidence, Readmission, and Mortality	Examines the relationships between green spaces and AMI, HF, and CVH.	– Levels of residential green spaces	– AMI -HF -CVH

DISCUSSION

The study conducted by Zheng et al. demonstrates a correlation between access to healthy food options and cardiovascular disease risks [6]. They determined that areas with a large density of healthy food options had low rates of cardiovascular disease whereas areas with a high concentration of fast food restaurants had high rates of cardiovascular disease due to fast food chains promoting unhealthy diets [6]. The analysis by Unger et al. supports the theory that giving access to fitness amenities can have positive effects on the health of both women and men [7]. The data suggests that access to exercise facilities positively impacts women's health by 22% and 16% for men [7]. Furthermore, the results made by Chen et al. indicate that access to green spaces is associated with a reduced likelihood of developing heart diseases [8]. The adult population residing near urban greenery is coupled with a 10% decrease in mortality from cardiovascular disease, a 6% decrease in HF, and a 7% decrease in AMI [8].

Based on the findings, we can conclude that an environment has a significant impact on cardiovascular health. In alignment with my research question, the availability of healthy food resources and green spaces for physical activities within an environment has the potential to influence CVD rates. The results of the study by Dariush Mozaffarian suggest that having a well-balanced, diverse, and heart-healthy diet is important in preventing CVD [4]. However, based on the findings of a similar study by Li et al., without access to and the willingness to consume healthy foods, individuals won't be able to take on a healthy diet and ultimately will have a difficult time preventing CVD [5]. These two studies are similar yet different due to one article highlighting the importance of a healthy diet in preventing heart disease and the other emphasizing the influence of access to nutritious foods has on people's capability to adopt a healthy diet. These results should be taken into account by policymakers at all levels around the world to

advocate policies that support healthier food options and look into supporting businesses that promote active lifestyles to help their citizens live healthy and happier lifestyles.

Limitations

A limitation of this literature review is that we solely focused on gathering and analyzing information from articles with people between the ages of 20 to 80 years old. Due to the lack of data collected on infants and younger people, the effect of the environment on the CVD rates of these individuals could not be determined. As a result, our findings fail to adequately represent the impact of the environment on CVD rates across all ages. However as the majority of CVD is seen in adults over age 40, the results pertain to the most relevant population.

Recommendations

Further research should be conducted across all age groups from infants to the elderly, to gain a more extensive and cohesive understanding of the influence of the environment on cardiovascular disease rates over the life course. Lawmakers should mandate and facilitate the establishment of recreation centers and healthy food choices within cities influencing citizens to make healthy choices and lead healthier lifestyles. Sidewalks, pedestrian-friendly walking areas, and parks should be more prevalent in the environment to promote physical activity among individuals. Lastly, future studies should consider the effect of the environment on individuals with pre-existing health conditions. People with pre-existing conditions can be affected differently by their built environment and it is important to recognize the difference between people with and without underlying health conditions.

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Sheryl Lawrence

Treatments for Breast Cancer in Postmenopausal Women

ABSTRACT

Breast Cancer is a disease that affects 264,000 women annually. Postmenopausal women over the age of fifty are at a higher risk of developing breast cancer because they are exposed to high levels of estrogen. Endocrine Therapy is supposed to inhibit hormone production and prevent hormones like estrogen from making breast cancer cells grow and divide. However, CDK 4/6 (proteins in the cell cycle) has a resistance to Endocrine Therapy therefore it allows breast cancer cell production to continue. This paper explores the various drugs (cdk4/6 inhibitor drugs, estrogen receptor degrader drugs, PI3k inhibitor drugs, mTOR inhibitor drugs, and SRC kinase inhibitor drugs) being assessed in clinical trials in the last 5 years to inhibit breast cancer cell production in postmenopausal women. The inclusion factors were if the article was a clinical study, written in the last 5 years (since 2018), was related to breast cancer in postmenopausal women. The exclusion criteria was if it did not include both"breast cancer", and "postmenopausal women". CDK 4/6 inhibitor drugs, estrogen receptor degrader drugs, PI3k inhibitor drugs, and mTor inhibitor drugs presented many benefits like increased life expectancy and reduction in breast cancer cells. SRC kinase drugs had no effect on breast cancer in postmenopausal women despite their many side effects. Certain drugs are better suited with certain types of breast cancer.

INTRODUCTION

Breast Cancer is a disease that affects 264,000 women annually [1]. Breast Cancer is when breast cancer tissue cells grow faster than normal which is caused by high levels of estrogen and progestin (hormones). Postmenopausal women over the age of fifty are at a higher risk of developing breast cancer because they are exposed to high levels of estrogen [2]. The cell cycle allows cells to replicate including cancer cells. Endocrine Therapy is supposed to inhibit hormone production and prevent hormones like estrogen from making breast cancer cells grow and divide [3]. However, CDK 4/6 (proteins in the cell cycle) has a resistance to Endocrine Therapy therefore it allows breast cancer cell production to continue [4]. Some drugs are inhibiting proteins in clinical studies: Ki67 is a protein

associated with tumor aggressiveness, mTOR is associated with cell growth, and SRC kinase regulates cell growth.

The research question is, “what alternative drugs are being evaluated that can help inhibit Breast Cancer cell production?” This paper explores the various drugs (cdk4/6 inhibitor drugs, estrogen receptor degrader drugs, PI3k inhibitor drugs, mTOR inhibitor drugs, and SRC kinase inhibitor drugs) being assessed in clinical trials in the last 5 years to inhibit breast cancer cell production in postmenopausal women. All these drugs target specific proteins (CDK 4/6, Pi3K, mTOR, and SRC kinase) and protein receptors (estrogen receptors) which are being tested to determine if breast cancer cell production will diminish. Both the efficacy and side effects of each new treatment are discussed in the paper.

METHODS

Search Terms

The search terms used were “Breast Cancer”, “Postmenopausal Women” and “Treatments.

Search Databases

I used the PUBMED database to gather all my information based on the search terms.

Inclusion and Exclusion Criteria

The inclusion factors were if the article was a clinical study, written in the last 5 years (since 2018), was related to breast cancer in postmenopausal women. The exclusion criteria was if it did not include both“breast cancer”, and “postmenopausal women”.

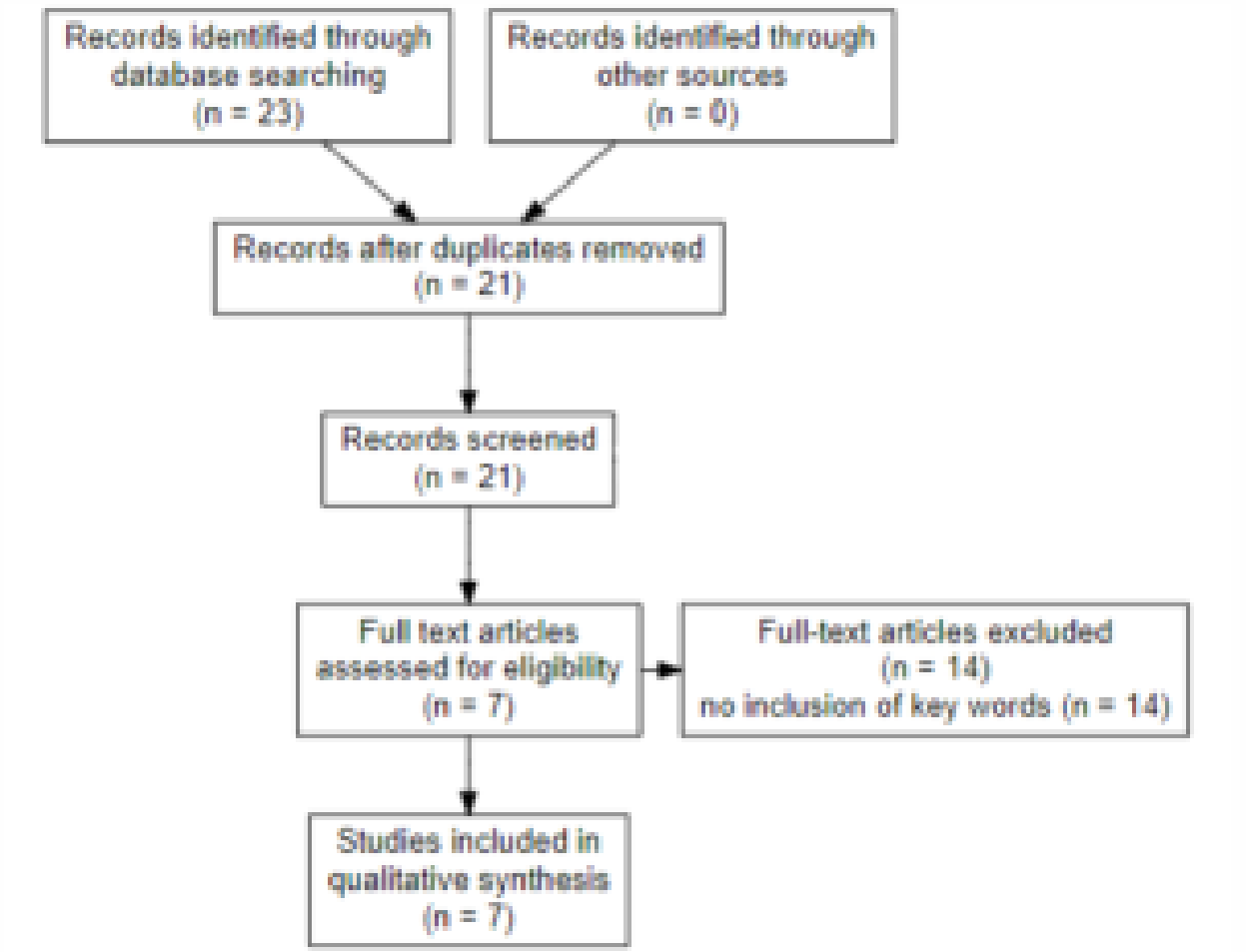

Figure 1. PRISMA Flowchart

RESULTS

CDK 4/6 Inhibitor Drugs

A clinical study evaluated the change in Ki67 after treatment of abemaciclib, anastrozole, or abemaciclib plus anastrozole in Postmenopausal women with stage I-IIIB HR(+)/HER2(-) breast cancer. The change in Ki67 expression by demonstrating a greater decrease in expression of the tumor Ki67 levels after 2 weeks of abemaciclib alone (−91%) or in combination with anastrozole (−93%) compared with anastrozole alone (−63%) [5]. They also found that complete cell-cycle arrest was achieved in most patients after 2 weeks of treatment with abemaciclib either alone or in combination with anastrozole as compared with anastrozole alone [5]. Dr. Prat and Saura evaluated the impact of CDK4/6 inhibition with endocrine therapy in comparison to chemotherapy. The result is that, 23 (46·9%; 95% CI

32·5–61·7) of 49 patients in the ribociclib plus letrozole group and 24 (46·1%; 32·9–61·5) of 52 patients in the chemotherapy group were low-ROR (molecular degrading) [6]. CDK4/6 inhibitors combined with endocrine therapy had higher rates of neutropenia, leukopenia, thrombocytopenia, anemia, fatigue, diarrhea, febrile neutropenia, nausea and increased alanine aminotransferase (ALT) [7].

mTOR Inhibitor Drugs

Dr. Bardia and Modi evaluated a triple therapy of CDK4/6 inhibitor, mTOR inhibitor, and endocrine therapy. They had two study groups where one group received triple therapy while the other group did not. When examining tumor biopsies, they found that 14/33 patients treated with triplet therapy demonstrated a trend toward higher overall baseline expression of cell-cycle control genes and genes involved in the MAPK pathway in patients with PD compared with those with SD [8]. Dr. Angelo Leo and Dr. Stephen Johnston evaluated the effectiveness and safety of buparlisib plus fulvestrant in patients with advanced breast cancer who were pretreated with endocrine therapy and mTOR inhibitors. Between Jan 15, 2013, and March 31, 2016, 432 patients were randomly assigned to the buparlisib (n=289) or placebo (n=143) groups. Only 1- 2% of people experienced adverse side effects. Median progression-free survival was significantly longer in the buparlisib versus placebo group (3·9 months [95% CI 2·8–4·2] *vs* 1·8 months [1·5–2·8]; hazard ratio [HR] 0·67, 95% CI 0·53–0·84, one-sided p=0·00030) [9]. The side effects are hyperglycaemia, hypertension, and fatigue.

Estrogen receptor degrader

Dr. Bardia and Chandarlapaty evaluated once-daily amcenestrant, an oral drug that is a estrogen receptor (ER) degrader, in postmenopausal women with ER+/HER2- advanced breast cancer. The overall clinical benefit rate was 28.3% [10]. The drug revealed ER inhibition and degradation through paired tumor biopsies and cell-free DNA which showed detectable ERS1 mutations and Y537S mutations. The most frequently reported adverse events were as follows: hot flush ($n = 5$; 31.3%), diarrhea and nausea ($n = 4$ each; 25.0%), as well as decreased appetite, constipation, night sweats, and asthenia ($n = 3$ each; 18.8%) [11].

PI3K inhibitor

Dejan Juric and Janku evaluated PI3Kα-specific inhibitor (oral drug) plus fulvestrant in patients with ER+ advanced breast cancer (ABC). Alpelisib plus fulvestrant had a manageable safety profile with the alpelisib maximum tolerated dose of 400 mg and a recommended phase 2 dose of 300 mg once daily and objective response rate was higher (29% vs 0%) in patients with. Skin may be sensitive to combined PI3K inhibition with endocrine therapy resulting in rashes. However, they can be treated with the use of antihistamines, topical, or systemic corticosteroids [13].

SRC kinase inhibitor

Dr. Oswald and Symeonides evaluated the effect of saracatinib addition to aromatase inhibitors (AI) in patients with hormone receptor-positive metastatic breast cancer. Aracatinib was not associated with an improved PFS (3.7 months v. 5.6 months placebo/AI) and did not reduce likelihood of bony progression. Saracatinib was well tolerated with dose reductions in 16% and the main side effects were gastrointestinal, hypophosphatemia and rash. There was no observed beneficial effect on bone metastases [13].

DISCUSSION

While estrogen therapy was not effective for postmenopausal women with breast cancer, CDK 4/6 inhibitor drugs, estrogen receptor degrader drugs, pi3k inhibitor drugs, and mTor inhibitor drugs presented many benefits like increased life expectancy and reduction in breast cancer cells. SRC kinase drugs had no effect on breast cancer in postmenopausal women [13]. Triple therapy with endocrine therapy and mTOR and CDK4/6 inhibition is beneficial for postmenopausal women with HR+, HER2− ABC [11]. Overall, there are multiple newly tested drugs that are specifically beneficial for reducing breast cancer in postmenopausal women. Future research regarding breast cancer therapy in postmenopausal women should focus on creating a drug that reduces the amount of side effects such as anemia and nausea.

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Praneel Mallaiah

How Quality of Education Impacts Cardiovascular Disease

ABSTRACT

Cardiovascular diseases (CVD) remains at the top of the list for causing the most deaths among people. While medical procedures and pharmacological treatments exist to treat CVD, prevention of upstream influences could make a major impact on decreasing rates of CVD. This review highlights the potential impact of educational factors on lifestyle choices, health behaviors, and access to resources that contribute to cardiovascular health. By examining relevant literature and conducting empirical research, the study aims to provide insights into the correlation between education and cardiovascular outcomes, which in turn, can help initiate public health initiatives.

INTRODUCTION

According to the World Health Organization , an estimated 17.9 million people die from cardiovascular disease (CVD) each year, representing approximately 31% of all global deaths [1]. Individuals with lower levels of education often face a higher burden of CVD risk factors such as obesity, hypertension, and diabetes [1]. Additionally, populations with low education tend to have limited access to healthcare services, leading to undiagnosed or poorly managed cardiovascular conditions [1].

The pathology of CVD reveals the intricate relationship between education and its indirect impact on cardiovascular health. Poor education correlates with higher rates of smoking, unhealthy dietary habits, sedentary lifestyles, and stress—all contributing factors to the development and progression of CVD [3]. Moreover, the lack of health literacy stemming from inadequate education can result in delayed diagnosis, ineffective management of risk factors, and poorer adherence to treatment plans. These factors collectively underscore the urgency of addressing educational disparities to mitigate the burden of CVD.

The objective of this paper is to examine the relationship between level of education and cardiovascular outcomes by reviewing the existing scientific literature. Investing in education not only empowers individuals to make

informed health decisions but also lays the foundation for healthier communities by breaking the cycle of poor health outcomes associated with limited educational opportunities.

METHODS

This scoping literature review aimed to explore the correlation between education quality and its indirect influence on cardiovascular disease (CVD). The search was conducted through PubMed and Google Scholar using the key search terms CVD, socioeconomic status, education, and CVD. Articles published before the year 2000 were omitted, ensuring a focus on the most recent information. Systematic reviews or other literature reviews were excluded.

RESULTS

In a study involving over 210,000 participants (average age 46.3), around 8% had atherosclerotic cardiovascular disease (ASCVD) [2]. Educational levels varied, with approximately 14.7% having less than a high school education, 27% with a high school diploma, 20.3% with some college education, and 38% having completed college or more [2]. Over a follow-up time period of 4.5 years, mortality rates were notably higher among those with lower education levels, both for all causes and specifically for cardiovascular disease, compared to those with higher education levels [2]. Individuals with less than a high school education had a 40-50% increased risk of mortality in the overall population and a 20-40% increased risk in the ASCVD population for both all-cause and cardiovascular-related mortality [2]. Accounting for traditional risk factors lessened these associations but still showed significant differences, particularly for the group with less than a high school education level in the overall population [2]. This trend was consistent across various socio-demographic subgroups, including age, sex, race/ethnicity, income, and insurance status [2].

Khan et al. reported that approximately 20% of males and 25% of females were categorized as having “low” education, while around 42% of males and 41% of females fell into the “medium” education group, and roughly 38% of males and 33% of females were classified as “highly” educated [2]. A comparison between individuals with “low” and “high” education levels showed that those with “high” education had lower systolic blood pressure

(8% decrease), diastolic blood pressure (4% decrease), decreased blood glucose (6% lower), and lower total cholesterol (7% decrease) [1].

DISCUSSION

The education system plays a pivotal yet often overlooked role in shaping public health outcomes, including CVD. Access to education could be an indirect solution to making better health choices and behaviors. Individuals with higher levels of education tend to have greater awareness of health risks and are more likely to adopt healthier lifestyles. They're equipped with critical thinking skills that help them discern credible health information, leading to informed decisions regarding diet, exercise, and healthcare utilization. Moreover, education indirectly influences socioeconomic status, which in turn affects health outcomes [4]. Higher education levels may correlate with better job opportunities, income, and access to resources, including healthcare, all of which impact the prevalence and management of CVD.

Conversely, disparities in educational opportunities contribute to health inequalities, including the prevalence of CVD. Communities with limited access to education often face higher rates of CVD due to several factors. Limited health literacy stemming from inadequate education can lead to misunderstanding or ignorance of preventive measures against CVD. Additionally, lower educational attainment may intersect with lower socioeconomic status, leading to reduced access to healthcare services and healthy lifestyle resources, further exacerbating CVD risk factors. Addressing these disparities by improving educational access and quality can indirectly mitigate CVD prevalence by empowering individuals and communities to make informed health decisions and break the cycle of poor health outcomes associated with limited education.

Education plays a vital role in prevention from CVD as well as CVD mortality. People who have received a 'lower education' seem to be associated with CVD mortality and this was shown as there was a 40-50 % increased risk of mortality with individuals with less education [2]. Once again these findings display that there is a strong need for an education among populations and with that, we could start to see major decreases in CVD.

Similarly, another study also goes in depth with his correlation but between males and females [3]. It was once again found that no matter the gender, individuals with "high" education demonstrated better health outcomes, including lower blood pressure, improved lipid profiles, and reduced inflammation markers, in comparison to those with "low" education. Specifically, "high" education was associated with lower systolic and diastolic blood pressure, improved blood glucose levels, favorable lipid profiles, and reduced inflammation markers, even after adjusting for potential confounders. These results highlight the potential link between higher education levels and cardiovascular health benefits. I believe that the area of focus should be on policy and help the underprivileged in the area of schooling and healthy food options and transportation.

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Riti Meeniga

The Effect of Sleep on Cardiovascular Disease

ABSTRACT

Background: Poor sleep habits can increase an individual's chances of cardiovascular disease. This can occur due to Circadian misalignment from rapidly changing sleep habits or having poor sleep quality and short sleep periods. By learning how sleep can affect cardiovascular disease (CVD), individuals may be more motivated to improve their sleep habits and lead to an overall increase in population cardiovascular health.

Methods: This literature review used PubMed and included papers published within the last 10 years using key search terms sleep, cardiovascular disease, and CVD. Systematic reviews and meta-analyses were excluded.

Results: Circadian misalignment can lead to hypertension and tachycardia. Every one hour increase in sleep onset time can increase risk of CVD by 36%, and a decrease in sleep quality can account for a 9% increase in risk.

Discussion: Hypertension can lead to ruptures of blood vessels, and tachycardia can lead to hypertension. The evidence found in the review supports the idea that poor sleep habits lead to increased risk of CVD. More research should be done regarding the behavioral changes caused by sleep and how they lead to declining heart health.

INTRODUCTION

Significance of the Problem

Around 20% of the US gets less than six hours of sleep a night [1]. One in every five deaths is due to a cardiovascular disease (CVD), and lack of sleep can increase the risk of hypertension and coronary heart disease [1,2]. These effects can be magnified for those diagnosed with insomnia, the most prevalent sleep disorder plaguing the country [3].

Background Information

One of the main reasons unhealthy sleep habits lead to heart problems is due to Circadian misalignment, or the individual's internal clock no longer being in sync with the outside environment, such as the presence of sunlight. This can lead to elongated sleep cycles, feeling constantly sleepy

throughout the day, or even insomnia, where the body is in a state of constant hyperarousal and cannot rest at all. Two ways Circadian misalignment can occur is due to long-term shift work, defined as any work outside the hours of 7am and 6pm, or constantly changing your sleep routine.

Information Gap

While many studies have analyzed sleep as a determinant of cardiovascular disease, not many have talked about how the effects of sleep disorders are compared to sleep habits. By informing a wider audience about the true effects sleep can play into your life, it will hopefully encourage more people to put importance on getting a proper amount of sleep each night, which is defined as 7-9 hours by the National Institute of Health (NIH) [4]. In the case of disorders like insomnia, the study hopes to urge medical professionals into further expanding knowledge in this area by showing that these disorders may be even more dangerous than they seem. With sleep being one of the factors that determines cardiovascular health, a larger population getting more sleep will also help to reduce the number of deaths due to heart problems and lead to a higher overall level of health in the country.

Objectives

This literature review aims to explore how abnormal sleep patterns and sleep habits can lead to an increased risk for cardiovascular diseases and how different kinds can have different effects upon the body.

METHODS

Search Strategy

The search engine used for the literature review was PubMed. Key search terms included were sleep, cardiovascular disease, and CVD.

Inclusion and Exclusion Criteria

Only free full texts published within the past 10 years (2013 and earlier) were used to maximize the probability of any results being reflective of the present day in 2024. Systematic reviews and meta-analyses were excluded.

RESULTS

Circadian Misalignment

In one study discussing the effects of shift work on the heart, individuals with Circadian misalignment were found to have an increased overall blood pressure when studied over a 24 hour period [5]. Systolic blood pressure was increased by 3.0mmHg while diastolic blood pressure went up by 1.5mmHg [5]. When being exposed to Circadian misalignment for a longer period of time, individuals were also found to have an increased 24-hour heart rate by 1.6 bpm compared to those with Circadian alignment [5]. However, during wake-periods, heart rate was lower by 0.9bpm and higher by 3.6 bpm during sleep periods for Circadian misalignment [5].

Sleep Duration and Sleep Onset

Another study included 1,992 participants all known to be free of heart problems and evaluated the effects of sleep duration and sleep onset timing on CVD [6]. Every 1 hour increase or decrease from the average sleep duration was found to lead to a 36% higher chance of acquiring a cardiovascular disease, after adjusting for other sleep related factors and other risks of CVD [6]. When taking into account these same factors, every additional one hour increase in the average sleep onset timing led to an 18% increase in risk of CVD [6]. When calculating the risk by including both sleep duration and onset timing, there was a 14%-23% increase in CVD risk [6].

Joint Effects of Sleep Duration and Sleep Quality

A study used a calculated measure called sleep score to determine the joint effects of sleep duration and sleep quality on CVD [7]. A higher, and better, sleep score meant an individual had a better sleep quality and were more likely to be getting 6-8 hours of sleep when compared to an individual with a lower sleep score [7]. A better sleep score was found to greatly reduce the risk of CVD, with every 5 unit increase in the Sleep Score accounting for a 9% decrease in CVD risk [7]. When looking at sleep duration and sleep quality separately, those with worse sleep quality were found to have a 21%-40% increase in CVD risk while those with sleep durations less than 6 hours had an increased risk of 13% [7].

DISCUSSION

Having unhealthy sleep habits can lead to an increase in the risk of cardiovascular diseases. Through many of the studies reviewed, it was found that sleeping less than the recommended 6 hours a night and also having a lower quality of sleep can increase risk for CVD by over 21%, while sleeping

just one hour later than the average in a population consistently could lead to an 18% increase in CVD [1,6]. Through another study reviewed, it was found that some of the overall effects of Circadian misalignment due to unhealthy sleep patterns on the heart are an increase in blood pressure and fluctuations in heart rate [5].

The increased risk of CVD due to poor sleep quality and sleeping less shows us that having bad sleep habits can possibly lead to getting a heart disease in the future. High blood pressure, also known as hypertension, and fluctuations in heart rate are known risk factors of cardiovascular diseases and indicators of poor heart health [8]. Hypertension can lead to CVD since the pressure of the blood in blood vessels may be too much for the vessel walls to bear, causing them to rupture and leading to a stroke [8,9]. An increase in resting heart rate, or tachycardia, may not mean much, but if it continues consistently, it can lead to certain forms of hypertension, again leading to increased risks of strokes and worsening heart health [9]. Since unhealthy sleep patterns, such as sleeping too little or having poor sleep quality, is what leads to the increased heart rate and hypertension, these pieces of evidence also support that unhealthy sleep habits lead to more heart problems.Shockingly, simply sleeping one hour later than recommended can lead to drastic changes in the risk of CVD.

The results of my review are mostly in line with other existing literature, such as articles published by the Center of Disease Control (CDC). One such article mentions poor sleep habits can lead to high blood pressure, but it also says that sleep can affect behavioral characteristics that then lead to heart health, such as unhealthy eating choices and less motivation for physical exercise [10]. While my study did evaluate how sleep affected risk of CVD, it did not take into account lifestyle choices that could then lead to declining heart health with sleep acting as an indirect cause.

Going forward, this data can be used to help the public understand the true implications of staying up too late. Due to over 35% of the current US population sleeping less than 7 hours a night, cardiovascular diseases can be expected to become more prevalent over the coming years [10]. Individuals can reduce their risk of this by making sure to get enough sleep while also doing whatever they can to improve the quality of their sleep.

According to the CDC, there is an established connection between an individual's heart health and their mental health [11]. We can apply the information found through this study to also help decrease problems due to mental health. If we can increase overall heart health in a population by improving sleep, it is expected that mental health will also improve.
More research should still be done in this area, such as how exactly certain bodily processes during sleep are interrupted by poor sleep habits and how this can affect heart health. Another area that little is known about is the effects of sleeping too long and its potential impacts on CVD.

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Alexis Maria Mikhelis

Investigating the Connection Between Social Determinants of Health in Women with Low Socioeconomic Status, Differing Education, and Cardiovascular Disease

ABSTRACT

Background: Cardiovascular disease (CVD) is a leading cause of death in women, with one in five experiencing symptoms annually. Despite its prevalence, women are often excluded from cardiovascular clinical trials, highlighting a critical gap in research. This paper explores the significance of the problem, emphasizing the need for more research, especially in the context of social determinants like stress, socioeconomic status, and race impacting women's cardiovascular health.

Methods: This scoping literature review using PubMed focused on studies conducted in the US after 2000, involving women over 18 years of age. Key search terms included health insurance status, socioeconomic status, education, and their relation to cardiovascular disease. Exclusion criteria ensured a targeted and relevant selection of studies for analysis.

Results: The review revealed significant associations between social determinants and cardiovascular health in women. Women with higher cardiovascular health scores exhibited better outcomes, emphasizing the role of education. Socioeconomic factors such as low income and education levels were linked to increased risks of CVD.

Discussion: The findings underscore the intricate interplay of socioeconomic disparities and depressive symptoms influencing women's cardiovascular health, emphasizing education as a protective factor. This study adds depth to existing knowledge, emphasizing the imperative for targeted interventions addressing education and socioeconomic disparities to enhance cardiovascular health in women. The nuanced relationship between mental health, educational attainment, and cardiovascular outcomes challenged conventional assumptions, highlighting the need for personalized approaches.

INTRODUCTION

One in five women every year experience shortness of breath, pain in one arm, heartburn, and unusual fatigue, ultimately leading to a heart attack caused by their heart simply not getting enough oxygen [1]. An estimated

3,200,000 women suffer from a myocardial infarction, also known as a heart attack each year [2]. In addition to this, approximately 213,572 die of coronary artery disease each year, a disease that affects the heart, restricting its ability to send enough blood and oxygen to the heart muscles through the coronary arteries.

Being that it is such a life-threatening disease, it often leads to myocardial infarction and or strokes, as the arteries sometimes fill with plaques [2,3]. Interestingly enough, more women die and have heart attacks than men, making it the top cause of death for women worldwide [4]. Most importantly it is crucial to note that women are still not being included in many clinical trials regarding cardiovascular disease (CVD) and many of these trials do not report results based on gender, leaving a clear need for more research and attention toward the field of cardiology in women. [5]
One common determinant of a higher risk of heart disease in women is stress depression and physical inactivity. This paper will dig deep into the different social determinants of health, socioeconomic status, education, and health insurance status, in women and cardiovascular disease and how they affect the lifestyle of women, eventually causing heart failure and other cardiovascular diseases. This will emphasize the need for more clinical research in this field and act as an advocacy and awareness platform for young women to be aware of these determinants.

METHODS

Search Strategy

In this scoping literature review, PubMed was used to identify relevant articles. The key search terms used were (Health Insurance Status OR Socioeconomic Status OR education) AND (Cardiovascular Disease OR CVD OR myocardial infarction OR hypertension) AND (Women).

Inclusion and Exclusion Criteria

Studies included were conducted in the US, were limited to studies on participants over the age of 18, focused on women, and were completed after the year 2000. Studies were excluded if they were conducted outside of the US, involved participants under 18, or included non-biological women.

RESULTS

In a study that examined the relationship between cardiovascular health (CVH) and the incidence of CVD in postmenopausal women, 99% of women with an ideal CVH had a high school education of more [7]. When comparing women with high CVH scores with those with the lowest CVH scores, women with low scores had seven times the hazard of CVD [7]. Another study looked closely at how money and where women lived, specifically living quarters and living situation, affected their health when treating high blood pressure CVD [7].

In a study with 27,862 participants, 7.8% were among the lowest-income sites [8]. It was found that these participants were majority women, black, Hispanic, and had fewer years of education [8]. This 7.8% had greater all-cause mortality (hazard ratio [HR], 1.25; 95% CI, 1.10–1.41), heart failure hospitalizations/mortality (HR, 1.26; 95% CI, 1.03–1.55), end-stage renal disease (HR, 1.86; 95% CI, 1.26–2.73), and were less likely to achieve blood pressure control (<140/90 mm Hg) (odds ratio, 0.48; 95% CI, 0.37–0.63) [8].

It was also found in a study that delved into the mental health of 341 African American women with hypertension, through a secondary analysis of a randomized clinical trial, that 57% of women with less than a high school diploma and women who smoked with chronic health conditions had a higher prevalence of depressive symptoms [9]. Women's depressive symptoms had a negative association with postsecondary education (adjusted odds ratio [aOR], 0.492; 95% CI, 0.249-0.968) and a positive association with the number of chronic conditions (aOR, 1.235; 95% CI, 1.046-1.460) and smoking (aOR, 1.731; 95% CI, 1.039-2.881) [9]. The following factors were associated with better depressive outcomes: having greater than a high school education (unadjusted OR, 0.492; 95% CI, 0.265-0.916; adjusted OR, 0.492; 95% CI, 0.249-0.968) and annual income between $20,001 and $40,000 (unadjusted OR, 0.479; 95% CI, 0.263-0.870, adjusted OR, 0.518; 95% CI, 0.275-0.974) [9]. Smoking (unadjusted OR, 1.761; 95% CI, 1.113-2.787; adjusted OR, 1.731; 95% CI, 1.039-2.881) and having multiple comorbidities (unadjusted OR, 1.199; 95% CI, 1.025-1.404; adjusted OR, 1.235; 95% CI, 1.046-1.460) was associated with increased risk of being depressed [9]. It was not found that there was a significant

association between depression and age, annual income greater than $40,000, health insurance status, and drinking behavior [9].

Another study focused on atrial fibrillation (AF), a common heart rhythm issue affecting older individuals, particularly women [10]. Women with AF tended to have higher levels of financial, and traumatic life events, neighborhood stress, and lower everyday discrimination stress scores than women without AF [10]. Traumatic life event stress continued to be significantly associated with odds of AF in age-adjusted models [OR 1.37, CI (1.19-1.59), p <0.0001], as well as in fully adjusted models [OR 1.32, CI (1.12-1.52), p <0.0007] [10].

Lastly, a prevention program for community-based cardiovascular disease was implemented in rural communities and intervention sites reported high levels of fidelity (82%) and dose delivered (84%) [11]. Overall reach was 2.6% and program classes were rated as effective (3.9/5) [11]. Participants were satisfied with their experience and reported benefits such as camaraderie and awareness of healthy eating and exercise strategies [12].

DISCUSSION

In examining the relationship between social determinants of health and CVD in women, this literature review revealed compelling insights into the intricate interplay of factors influencing women's cardiovascular health. The findings underscored the importance of education as a significant determinant, with women having a high school education or less exhibiting a higher risk of CVD. Notably, 99% of women with an ideal CVH profile had a high school education or more, emphasizing the potential protective role of education in cardiovascular well-being. This suggests a link between educational attainment and overall health, emphasizing the need to address educational disparities to mitigate cardiovascular risks in women. Additionally, this review shed light on the pervasive impact of socioeconomic factors, exemplified by the association between lower income, education, and increased risk of CVD. The disparities observed in health outcomes among participants from lower-income sites, predominantly women of Black and Hispanic backgrounds, underscore the urgency of addressing socioeconomic inequalities in cardiovascular health interventions. These results were expected to some extent, aligning with prior research, yet the magnitude of the associations emphasizes the critical need for targeted

interventions addressing these disparities to alleviate the burden of CVD in vulnerable populations.

Surprisingly, despite the known relationship between mental health and cardiovascular outcomes, findings revealed a nuanced connection between depressive symptoms and educational attainment among women with hypertension. The studies illuminated the complex interplay of factors, showing a negative association between postsecondary education and depressive symptoms, but a positive association with the number of chronic conditions and smoking. This nuanced understanding challenges conventional assumptions and underscores the need for personalized approaches in addressing mental health within the context of cardiovascular disease.

Comparing these findings to current literature, our study provides nuanced insights into the multifaceted determinants influencing cardiovascular health in women. While some results align with existing knowledge, such as the impact of education and socioeconomic status, our exploration of the nuanced connections between mental health, educational attainment, and cardiovascular outcomes adds depth to the current understanding. Moving forward, these findings emphasize the imperative for targeted interventions addressing education and socioeconomic disparities to enhance cardiovascular health in women, ultimately advocating for more inclusive and tailored approaches in cardiovascular research and healthcare practices.

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Rupsa Mitra

Health Literacy and Frequency of Myocardial Infarctions and Hypertension in US Minorities

ABSTRACT

Myocardial infarction (MI), also known as a heart attack, and hypertension (HTN) are two types of cardiovascular disease (CVD) and are the leading causes of death in US minorities. While previous studies have found that genetics and other socioeconomic (SES) factors can explain the differences between the heart disease rates in Spanish-speaking Americans, African-Americans and their white counterparts, this research paper aims to explain how, specifically, health literacy can better explain the higher numbers of minorities suffering from MI and HTN in the United States. This literature review uses seven peer-reviewed papers that were published post 2000. These papers were found through PubMed and Google Scholar with the keywords being cardiovascular disease, health literacy, myocardial infarction, hypertension, minorities, and US. The results imply that health literacy plays a bigger role than others in determining one's health levels, and that targeted interventions, like CVD health camps, should be held to instruct minorities of the risks, treatments, signs, and symptoms of CVD. This literature review underscores the urgent need of such targeted health literacy interventions to address the rising incidence of MI and HTN among US minorities, highlighting disparities in risk factors and outcomes.

INTRODUCTION

According to the CDC, one person dies every 33 seconds in the United States from cardiovascular disease (CVD) [1]. Based on a study, despite just being approximately 28% of the population sample, Black participants had the highest incident rate of heart failure, with Hispanic patients following closely second [2].

A myocardial infarction (MI), also known as a heart attack, occurs when the blood flow to the myocardium is reduced or ceased. This can happen for a lot of reasons– from blood clots to an underlying coronary disease [3]. A person can experience chest discomfort, shortness of breath (SOB), nausea, and/or light-headedness during the onset of a MI [4]. A heart attack can also lead

to permanent heart damage, increasing the risk of arrhythmia and thrombosis [5].

Hypertension (HTN), a type of CVD, is caused when the pressure builds up in the arteries, pushing on the arterial walls. This results in severe headaches, fatigue, dizziness, and nosebleeds. In the United States, more than 35 million adults have uncontrolled blood pressure (BP), representing one-half of the adult population with hypertension [6]. Moreover, minority groups have a higher risk of negative effects due to hypertension than their white counterparts. Black adults have higher hypertension prevalence than White adults. Hispanic adults have lower awareness and treatment rates than White adults [6].

Socioeconomic status (SES) is a complex phenomenon predicted by a combination of financial, occupational, and educational influences [7]. These factors influence one's successful recovery from MI and HTN. Health literacy, influenced by SES, is the ability of an individual to gain knowledge and use that information to improve health appropriately [8]. Inadequate health literacy can be responsible for the difficulties in comprehension of health information, limited knowledge of diseases and lower medication adherence, which contribute to poor health, high risk of mortality, insufficient and ineffective use of healthcare, increased costs, and health disparities [8]. Approximately 24% of African Americans and 41% of Hispanic adults exhibit "below basic" health literacy compared to 9% of White adults, as measured by the National Assessment of Adult Literacy [9].

While previous studies have researched how genetics and other socioeconomic factors can explain the differences between the heart disease rates in Spanish-speaking Americans, African-Americans and their white counterparts, this research paper aims to explain how health literacy can better explain the higher numbers of minorities suffering from MI and HTN in the United States.

METHODS

A scoping literature review was conducted to identify articles published, after the year 2000, that investigate the higher frequency of MI HTN in minorities living in the US. The search strategy involved using databases such as PubMed and Google Scholar with keywords related to heart attacks,

hypertension, risk factors, minorities, USA, health literacy, genetics, socioeconomic status, Hispanic-Americans, and African-Americans. Additionally, using the process of backsearching from those sources, more relevant articles were found. Articles were included if they focused on comparing the minority population and the white population, studying the higher risk factors and symptoms in each minority, or providing general information, including statistics, on CVD, and statements regarding sSES, including specific definitions.
After checking abstracts and reading the full research report, a total of seven relevant articles were selected for extracting data.

RESULTS

In a trial that determined the effect of MI risk-associated genotypes among different racial groups, those who were African Americans (AA) had significantly higher frequencies of two out of three risk-associated genotypes compared to European Americans (EA). The frequencies of GJA4, MMP3, and PAI-1 were observed to be 20%, 78%, and 55% in AA, respectively, whereas in EA, they were 7%, 24%, and 16%, respectively [10]. Additionally, disparities in HTN control have also been proven to exist among racial groups by Aggarwal, R [6]. His team and he found Black adults have higher hypertension prevalence (45.3% versus 31.4%) but similar awareness and treatment rates as White adults [6]. Hispanic adults have similar hypertension prevalence, but lower awareness (71.1% versus 79.1%) and treatment rates (60.5% versus 67.3%) than White adults [6]. After interviewing several hypertensive African-Americans, Ogedegbe, G., et al. concluded knowledge about high blood pressure and the nature of hypertension is one of the key facilitators of the health outcome of hypertensive African-Americans [11]. Lor et al. argues that health literacy has a larger impact on health outcomes than other factors. They found 88.4% of their sample to have low adherence levels and 84.9% to have inadequate health literacy [13]. Moreover, Chaudhry et al. used external surveys to conclude that even after adjusting for factors such as education, black patients still exhibited significantly worse health literacy [12]. Furthermore, Lor et al. held a survey, which outlined the importance of health literacy levels in developing effective adherence interventions [12]. Finally, Macabasco-O'Connell et al. discovered that out of 97 hispanic adults, 68 were found to have low health literacy regarding heart failure, while 29 had adequate literacy [14].

Table 1: Article Summaries

Sources	**Claims**
1. Lanfear et al. [10]	9.1% of African Americans had all three high-risk genotypes for CVD
2. Aggarwal et al. [6]	Black adults have higher hypertension prevalence Hispanic adults have lower awareness and treatment rates
3. Ogedegbe et al. [11]	Knowledge about high blood pressure and the nature of hypertension facilitates health outcome of hypertensive African-Americans
4. Chaudhry et al.[12]	Black heart-failure patients were significantly more likely than white patients to have poor health literacy: 24.4% of black patients had poor health literacy and 9.7% of whites
5. Lor et al. [13]	Overall, the majority of participants had low adherence levels to antihypertensive medications and inadequate health literacy.
6. Macabasco-O'Connell et al. [14]	68 (out of 97 hispanics) were found to have low health literacy regarding heart failure

DISCUSSION

After reviewing the data, a consistent trend becomes apparent, and health literacy can be identified as an important risk factor among minorities in the United States experiencing MIs and HTNs. The study highlights significant health literacy disparities among African Americans and Hispanic Americans in the United States experiencing MIs and HTNs. This implies the urgent

need to prioritize health literacy interventions for minorities. White Americans are less likely to face these challenges compared to minorities, but disparities persist overall. These findings emphasize the importance of targeted health literacy initiatives. Comparisons between SES and genetic factors suggest that SES alone may not be the sole determinant of MI and HTN outcomes. Genetics, as demonstrated by Lanfear et al., can also be a factor in the outcomes, highlighting the need to address genetics and SES to determine MI and HTN outcomes in Black Americans and Hispanics [10]. Even if African-Americans and Hispanic-Americans are genetically more prone to CVD, health literacy can increase the awareness of these fatal diseases, and, in turn, decrease the probability of a worse health outcome.

Overall, the results underscore the urgency of addressing the health literacy gap. Preventive strategies should target improving health literacy, especially among the respective minorities. A comprehensive plan of action with tailored interventions for race-specific risk profiles and a focus on enhancing health literacy can significantly contribute to reducing the burden of MI and HTN in the minority population. Schools and employers, in areas with high Hispanic/African populations, can hold health-literacy camps to spread awareness on the signs, symptoms, risk factors, and precautions for MI and HTN. These camps should be a part of the students' curriculum or the employees' training. Additionally, community outreach departments can hold such programs in libraries, playgrounds, or other community centers. Addressing potential barriers to resources is important, so that the minorities are able to participate. If transportation is an issue for some people, online camps should be held. These camps should not only cover CVD topics, but also basic health instruction, such as CPR procedure, better sleep habits, balanced food diets, and basic English instruction [15]. Additional efforts can involve partnerships with hospitals, government agencies, faith-based organizations, social service agencies, and medical schools. Schools and hospitals can incentivize the med-students and doctors, who go to hospitals in neighborhoods with a high marginalized population, to offer heart screenings and checkups.

By addressing these problems, coming up with more creative solutions, and enforcing the above solutions, public health efforts can effectively improve outcomes for African Americans and Hispanic-Americans suffering from MI and HTN.

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William Nguyen

Sleeping Away Success: The Toll of Sleep Deprivation on Cognitive Function in Adolescents

ABSTRACT

This research study aimed to investigate the effects of consistent versus inconsistent sleep schedules on cognitive function in adolescents. Relevant research articles were accessed through PubMed using specific inclusion criteria, including keywords such that relate towards the importance of sleep. In addition, articles that were only published recently were selected. The results revealed that maintaining a consistent sleep schedule is crucial for optimal cognitive function in adolescents. Inconsistent sleep schedules negatively impacted attention, processing speed, and working memory. These findings emphasize the significance of promoting healthy sleep habits among adolescents and implementing interventions to improve sleep consistency. Future actions may involve the development of sleep education programs and interventions targeting sleep schedules in educational settings. Healthcare providers can also play a role in assessing and addressing sleep issues in adolescents.

INTRODUCTION

Everyone needs sleep. Sleeping is a human necessity that refreshes the human body and helps us function. However, this basic human necessity is restricted constantly among adolescents. A study found that the average high school student slept for 6.6 hours [1]. Ultimately, this contradicts the recommended 8-10 hours that most healthcare specialists recommend. In addition, 30% of boys and 49% of girls reported experiencing sleeping difficulties and 36% of 15-year-olds reported not having enough sleep to be able to concentrate on school work [2]. Researchers say that there are various reasons for adolescents not receiving enough sleep. One prevalent issue that researchers have found is that because of the competitive nature of school and college admissions, students are constantly required to sacrifice their sleep in order to have an advantage over their peers. These habits of getting less sleep to try and gain a competitive advantage over their peers might seem like a clever idea, but it ultimately could have the risk of impacting their cognitive function negatively.

Despite the acknowledged importance of sleep in adolescents, there is still a significant gap in our understanding of the relationships between sleep and cognitive abilities in adolescents. Extensive studies have gained increasing knowledge regarding the wide-ranging influence of sleep on cognitive function. However, there remains a lot of room for exploration concerning the intricate mechanism. Prior research has underscored the adverse outcomes arising from sleep deprivation, encompassing attention, memory, problem-solving, and decision-making [3]. The consequences of inadequate sleep have been associated with diminished academic achievements, compromised learning capacities, and heightened vulnerability to mental health challenges among young individuals [4]. Overall, this paper will strive to find the true consequences behind varied sleep and show that adolescents who exhibit higher variability in sleep duration will demonstrate poorer cognitive functioning and experience higher levels of mental health difficulties compared to those with more consistent sleep duration patterns.

METHODS

PubMed was utilized to access relevant research articles related to the study topic. The search was performed using specific keywords that include "adolescents", "cognitive function", "importance of sleep", "sleep" and filters to identify relevant publications.

Inclusion and Exclusion Criteria

To ensure the selection of relevant literature, specific inclusion and exclusion criteria were established.

The inclusion criteria were as follows:

- Participants: Adolescents aged between 10 and 19 years, as the studies used primarily focused on this age group.
- Outcome of Interest: Cognitive function, as the research aimed to investigate its relationship with sleep.

- Time Frame: Publications within the last 10 years (2013-2023) were considered to include the most recent and updated information.

The exclusion criteria were as follows:

- Participants: Studies involving adults or children younger than 10 years of age were excluded from the analysis.
- Outcome of Interest: Publications primarily focusing on physical effects such as eye strain or leg cramping were excluded.

Data Analysis

The analysis of the collected literature was qualitative. Key findings and information related to the impact of sleep on cognitive function in adolescents were identified and therefore used. A systematic review was used to explore patterns and relationships within the literature.

RESULTS

A total of five articles were found and utilized. The findings indicate that sleep restriction and total sleep deprivation have detrimental effects on cognitive function, attention, mood, and memory formation in adolescents and young adults. Additionally, the studies highlight the importance of length of sleep in relation to emotional regulation, cognitive function, academic achievement, sleep quality, physical activity levels, and depressive symptoms in children and adolescents. Shorter sleep duration is associated with a higher risk of depressive symptoms, while those who had a longer sleep duration were able to perform greater cognitive functions such as greater math abilities.

Author Name, reference number	Country	Study Type	Results	References
Lo JC, et al.	Singapore	Randomized Controlled	The sleep-restricted group showed deterioration in sustained attention, working memory, and executive function, increased subjective sleepiness, and decreased positive mood compared to the control group. Even after two recovery nights, subjective sleepiness and sustained attention did not return to baseline levels.	[3]
Lo JC, et al.	Multiple Countries	Experimental Study	Total sleep deprivation (TSD) led to a higher misinformation consistent response rate compared to the control group, indicating an increase in false memory formation. Partial sleep deprivation (PSD) did not reach statistical significance when compared to the control group. Ancova analyses suggested that the higher misinformation consistent response rate in the TSD group could not be solely attributed to impaired	[4]

			sustained attention or subjective alertness.	
Dutil C, et al.	United States and China	Literature Study	The sleep EEG amplitude and power decrease during adolescence, which is associated with reductions in cortical grey matter volume. Sleep deprivation impairs memory formation and consolidation in adults, but verbal memory in adolescents appears relatively unaffected. This suggests the presence of neural compensation or the sufficiency of conserved sleep in supporting cognitive performance.	[5]
	Canada	Systematic Review	Later sleep timing was associated with poorer emotional regulation, lower cognitive function/academic achievement, shorter sleep duration/poorer sleep quality, poorer eating behaviors, lower physical activity levels, and more sedentary behaviors in children and adolescents. Limited associations were found between sleep timing and adiposity, quality of life/well-being, accidents/injuries, and biomarkers of cardiometabolic risk.	[6]

Zhou T, et al.	China	Cross-Sectional Study	Chinese adolescents with shorter sleep duration (<6 hours/night) had a higher risk of depressive symptoms. Higher mathematics scores were associated with a lower risk of depressive symptoms. Cognitive function mediated the effect of sleep duration on depressive symptoms.	[7]

DISCUSSION

The study by Lo et al. investigated the effects of consistent versus inconsistent sleep schedules on cognitive function in adolescents [3]. Participants were randomized into a sleep restriction (SR) group and a control group, with the results showing that a more consistent sleep schedule led to better cognitive function. The SR group, with 5 hours of sleep per night, performed significantly worse on cognitive tasks compared to the control group with 9 hours of sleep.

Similarly, another study by Lo et al. investigated the effects of consistent versus inconsistent sleep schedules on cognitive function in undergraduate students [4]. Participants were divided into control, sleep restriction (PSD), and total sleep deprivation (TSD) groups. Cognitive performance was assessed using reliable measures such as the misinformation paradigm, psychomotor vigilance task (PVT), and Karolinska Sleepiness Scale (KSS). The results showed that both PSD and TSD groups exhibited impaired cognitive performance compared to the control group. Specifically, the TSD group demonstrated a higher susceptibility to incorporating false information into their memory. Although the difference between the PSD and control groups was not statistically significant, there was a medium impact on cognitive performance.

Tarokh et al. also performed a study aimed to investigate the effects of consistent and inconsistent sleep schedules on cognitive function in adolescents [5]. Participants were randomly assigned to different sleep pattern groups, and the results showed that maintaining a consistent sleep

schedule led to increased bilateral hippocampal grey matter volume. Like the studies performed by Lo et al., Tarokh et al. agreed that maintaining a consistent sleep schedule is crucial for optimal cognitive function such as memory testing abilities in adolescents. Inconsistent sleep schedules, characterized by sleep restriction, negatively impacted attention and an increase in memory under cognitive testing conditions. These findings highlight the importance of promoting healthy sleep habits and implementing interventions to improve sleep consistency. Factors such as circadian rhythm and sleep duration likely influenced the outcomes observed. One way to mitigate these factors is deploying programs such as sleep education programs in schools and involving healthcare providers in assessing and addressing sleep issues. Future research should aim to include more diverse populations and explore underlying mechanisms to develop targeted interventions and strategies for promoting healthy sleep habits and optimizing cognitive outcomes.

Although length of sleep is important, sleep timing should be considered in shaping various health outcomes and support the implementation of interventions to support optimal cognitive development and emotional well-being in children and adolescents. Dutil authored a systematic review aimed to examine the effects of sleep timing on health outcomes in children and adolescents [6]. The review included 37 articles and found that consistent sleep timing was associated with better emotional regulation, cognitive function, academic achievement, quality of life, and well-being. Conversely, irregular sleep timing was linked to negative outcomes such as anxiety, depressive symptoms, stress, mood disturbances, hyperactivity, and impulsivity. The review also identified associations between sleep timing and accidents and injuries, biomarkers of cardiometabolic risk, eating behavior, sleep duration and quality, and physical activity and sedentary behavior. The findings support the hypothesis that a more consistent sleep schedule leads to better cognitive function and academic achievement in children and adolescents. The review provides an up-to-date and comprehensive overview of the literature on sleep timing and health outcomes, considering both observational and experimental studies. However, it is important to acknowledge potential biases in the included articles and limitations such as recall bias and the predominance of cross-sectional designs. The generalizability of the findings should also be considered. Future research should aim to include diverse populations to enhance the external validity of

the findings. Based on the results, future actions can be taken to promote regular sleep patterns and optimize cognitive function in children and adolescents. Educational campaigns can raise awareness about the importance of consistent sleep schedules and provide strategies for establishing healthy sleep habits. Healthcare professionals can play a role in assessing and addressing sleep issues, offering guidance on sleep hygiene practices and monitoring sleep patterns.

In addition to cognition, sleep may also have consequential effects on mental health. The present study written by Zhou et al. examined the relationship between sleep duration, cognitive function, and depressive symptoms in Chinese adolescents [7]. The findings showed that shorter sleep duration was associated with a higher risk of depressive symptoms, while a longer sleep duration was linked to better mental health. Cognitive function mediated the relationship between sleep duration and depressive symptoms. These results support the hypothesis that consistent sleep schedules, indicated by adequate sleep duration, are associated with a decreased likelihood of experiencing depressive symptoms in Chinese adolescents. The study contributes valuable insights within the context of the Chinese adolescent population. It is important to consider potential biases in the study, such as reliance on self-reported data and the cross-sectional design, which limits establishing causality. Future research should employ longitudinal designs and comprehensive assessments of sleep patterns and quality to further explore the mechanisms underlying these associations and develop targeted interventions for promoting mental health and well-being in adolescents.

The overall consensus emerges on the pivotal role of maintaining consistent sleep schedules for optimal cognitive function among adolescents and young adults. While studies by Lo, Tarokh, and others underscore this agreement, nuances arise regarding sleep’s influence on mental health, with an emphasis on depression. Lo’s findings hint at the protective potential of adequate sleep duration against depressive symptoms, while Tong Zhou’s research introduces the intricate interplay between sleep duration, cognitive function, and depression in Chinese adolescents. Limitations, like reliance on self-reporting and small sample sizes, echo across these studies and our synthesis, underscoring the need for robust methodologies and broader participant representation. Amidst these findings and constraints, these studies propel understanding of sleep’s impact on cognitive and emotional

well-being, advocating for proactive measures such as sleep education programs. Yet, unanswered questions persist about underlying mechanisms and individual variations. Moving forward, this collective research points to promising interventions for cognitive enhancement and mental health mitigation, necessitating rigorous studies to unravel sleep's intricate effects comprehensively and guide holistic adolescent flourishing.

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Monica Pace

The Impact of Economic Status and Social Support on Cardiovascular Health

ABSTRACT

Economic status, an important social determinant of health, impacts quality of life and cardiovascular health. But how does economic connectedness, or the economic status of our friends impact upward mobility and the ability to improve our socioeconomic status and health? This report explores the deep connection between economic status and social support on cardiovascular health, and how social networks may play a role.

INTRODUCTION

Economic status and social relationships play a significant role in cardiovascular health as people with lower income often face barriers to living healthy lifestyles due to factors like limited access to nutritious food, safe places to exercise, and good quality healthcare [1]. If people have a lower economic status it could lead to not getting the professional care that is required for their health [1]. Likewise, social interactions can influence cardiovascular health [1]. For example, hostility which is defined as aggressive behavior or conflict, may lead to increased stress and poor health outcomes [2]. On the other hand, social support including emotional, financial, and practical support, can lead to decreased levels of stress and better health outcomes [2].

Data from the Center for Disease Control (CDC) show 20% of high income individuals have healthier levels of biomarkers for cardiovascular disease [3]. Higher income individuals have greater access to primary care and more frequent screening for CVD meanwhile 80% of low-income individuals have hypertension, high cortisol levels, and these conclude risk factors that include higher chances for cardiovascular abnormalities [3].

If we could improve access to health care for low income individuals it could lead to better life quality, a healthier life, and a more happier and active community. The efforts to better health have tried to be provided to the public such as offering free exercise programs, and creating a garden. But this doesn't address the full problem, these "solutions" are not sustainable.

People with a lack of money are more likely to not get the education they need to know about what more they can do to prevent cardiovascular abnormalities based on economic status.

This paper aims to examine the impact of social networks of low-income individuals on cardiovascular health.

METHODS

I conducted a scoping literature review by searching PubMed and Google Scholar using the key words cardiovascular disease and economic status. Studies performed in the United States were included while systematic reviews and literature reviews were excluded.

RESULTS

Knox et al., reports on the relationship between hostility, social support, and carotid artery atherosclerosis [2]. Social support decreased the odds of atherosclerosis in women at a high risk of coronary heart disease [2]. High risk women were more impacted by social support than high risk men, or medium and low risk men and women [2]. In high-risk men and women, hostility negatively impacted atherosclerosis, however, hostility plus low social support had even higher odds of developing atherosclerosis [2]. High risk women were impacted by high hostility, however low to medium risk men and women had no significant association [2].

Orth-Gomér et al., reports that marital stress caused a 2.9-fold increase in recurrent coronary events as compared to work stress, which did not significantly increase recurrent coronary events [4]. After statistically taking into account covariates including the severity of angina pectoris symptoms, sedentary lifestyle, personal history of high blood pressure, family history of coronary heart disease, body mass index, and total cholesterol level the result was still significant [4]. It is important to note that marital status itself was not associated with an increased risk of recurrent events [4].

Evidence from Hubinette et al., shows how lower social classes have less social support, reduced health benefits and limited healthcare access [5]. This correlation between socioeconomic status on cardiovascular disease shows that lower social classes have a higher risk of heart disease [5]. According to Chetty et al., people within the same social class tend to be

friends more than people who aren't in the same social class [6]. If there are alot of high socioeconomic (SES) people in one place like a neighborhood, you'll likely find a lot of high SES people in other places like school's [6]. Friending bias, or the tendency to make friends with certain types of people, is usually the same across different places [6]. So, low bias group's (like religious groups) in one area will probably be low bias in other areas too [6]. Where you live can affect how many high SES individuals you meet, but the groups you're part of really determine how much you interact with these high SES peers [6].

DISCUSSION

The study from Knox et al. underscores the protective role of social support against atherosclerosis in high-risk women, highlighting the significance of strong social connections in promoting cardiovascular health [2]. Moreover, the differential impact of social support across gender and risk groups suggests the need for gender-specific interventions targeting social support to mitigate cardiovascular risk factors as women were more likely to rely on social support. Additionally, Knox et al. reveal the adverse effects of hostility on atherosclerosis, particularly in high-risk individuals, emphasizing the detrimental consequences of hostile attitudes and behaviors on cardiovascular health. The exacerbation of atherosclerosis risk in individuals experiencing high hostility and low social support underscores the importance of addressing both psychosocial factors in cardiovascular disease prevention efforts.

Orth-Gomér et al.'s study highlights the association between marital stress and the risk of recurrent coronary events [4]. This underscores the critical impact of marital relationships on cardiovascular health outcomes and emphasizes the need to consider the quality of marital relationships when assessing cardiovascular risk. While both work stress and marital stress put strain on the heart, only marital stress was associated with increased risk of having recurrent coronary events. This suggests that the social support one would receive from a partner, which is typically protective of heart health, is not present. The results emphasize the imperative of targeted interventions aimed at mitigating marital stress to reduce the burden of recurrent coronary events in at-risk populations.

The Hubinette et al. study demonstrates that low-income individuals have increased risk of cardiovascular disease, compared to high-income

individuals, while Chetty et al. demonstrates that people tend to befriend those in similar SES positions [5,6]. High income-individuals may have more connections than people with lower incomes, which leads me to believe that people with higher incomes may have more efficient healthcare than people lower income individuals.

Ultimately, assessing the current literature on social support, this paper has found that a lack of a strong support system usually negatively impacts cardiovascular health. To increase social support, doctors can provide information to patients on therapy and group support. Future researchers should do a needs assessment for social support among low-income communities and then study effective interventions to increase social support.

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Kushal Paul

Analysis of the Relationship Between the Legalization of Marijuana and a Potential Increase in Teenage E-cigarette Use

ABSTRACT

Adolescent vaping has increased from 3% in 2011 to 27% in 2014, leading to concerns for respiratory health. In this paper, we look at the increased availability of nicotine products stemming from the legalization of marijuana as a possible cause for increased teen e-cigarette usage. The Youth Behavioral Risk Survey and Healthy Kids Colorado Survey were analyzed to investigate this hypothesis. Through a Google Scholar and PubMed literature search, the following key terms were utilized: “adolescent”, “nicotine”, and “vaping”. The evidence suggests that following the legalization of marijuana in Colorado, teenage e-cigarette use increased two times from 2013 to 2021 and sits at a higher rate than the national average (36.2%). There is strong support for the theory that the legalization of marijuana increased e-cigarette use amongst adolescents through increased availability of such products.

INTRODUCTION

Since 2014, the most popular nicotine product in teens has been vapor products/e-cigarettes(1). A large majority of e-cigarette use is continued due to the addictive properties of nicotine. Nicotine addictions are problematic as e-cigarette use has been shown in clinical studies to cause increased rates of respiratory disease and adversely alter pulmonary function in teens(2). Another problem caused by e-cigarette use in teens is increased stress levels(3). Stress can cause poor health conditions and life-threatening illnesses(4). Furthermore, stress can lead to using other narcotics in teens, causing further physical harm(3).

To minimize the negative consequences of adolescent e-cigarette use, we must look at the potential causes. Common theories suggest aggressive marketing and ineffective education regarding the consequences of frequent e-cigarette use(1,2). However, the research on these causes is not concrete enough to discredit looking for new solutions, such as the increase of teen e-cigarette usage through the lack of perceived harm. Therefore, this study will explore the concept that e-cigarette use is increasing through the

availability and subsequent perceived lack of danger stemming from recreationally legal marijuana in Colorado, as it is one of the states with the highest adolescent e-cigarette usage(5,6).

Starting in 2012, Washington and Colorado legalized recreational marijuana statewide. Afterwards, many other states have followed in their footsteps and brought up similar legislation(7). The evidence and analysis will explore the relationship between the legalization of marijuana and the corresponding increase of e-cigarette based nicotine addiction. Therefore, we hypothesize that the legalization of marijuana is a major contributor to the increase of e-cigarette based nicotine addictions.

METHODS

This paper reviewed a combination of previously published research and reports on national/statewide databases.

Search queries and Search Engine

Pubmed and Google Scholar were searched with the keywords: "addiction", "adolescent", "Colorado", "e-cigarette", "marijuana","nicotine", "teen","vaping","weed" in different combinations to find literature that reported on trends of e-cigarette use in adolescents.

Databases/Surveys

The Youth Risk Behavior Surveillance Survey (YRBSS) or Youth Risk Behavior Survey (YRBS) conducted by the Center of Disease Control (CDC) asked a series of questions to students in grades 9-12 regarding key health habits that contribute to the highest teen mortality rates. For the purpose of this analysis, we reviewed questions regarding e-cigarette usage. These questions include: "Have ever used e-cigarette products" and "Used e-cigarette products in the last 30 days." This source was primarily used to compare Colorado to other states and overall national data. This survey is administered every odd year and we use the most recent date available as of 2023(9).

A second database called Healthy Kids Colorado Survey (HKCS) focuses on a questionnaire asked toward Colorado students. This survey is done during the fall on odd years. Similar to the nationwide survey, we are focusing on answers regarding questions on e-cigarette use. This source was used to

observe trends regarding e-cigarette use overtime in Colorado students. We look at data from 2013-2021 as these are years where specific e-cigarette focused questions were asked.

The provided online portal for both sources were used to filter specific questions about e-cigarette use in teens and students.

Exclusion Criteria

We excluded data regarding other types of nicotine products (traditional cigarettes, nicotine gum, nicotine patch, dip, chew), side effects of adult e-cigarette use, and teenage substance abuse other than e-cigarettes. We excluded data outside of the U.S and before 2010.

RESULTS

Literature results

A paper published by Hadland et al. interprets a general surgeon report from 2016 stating that from 2011 to 2015 e-cigarette use increased from 3% to 27%. Furthermore, data from the same paper states a 78% increase of e-cigarette use among high schoolers from 2017-2018(8).

Lin et al. shows the trends of e-cigarette use amongst teens in California between the years 2014 and 2018. In these years Lin found a 55.2% increase in e-cigarette use amongst teens.(9)

Another article by Wang et al. notes an increase in e-cigarette products sales from US retail scanner data. The units sold increased to 1,547 per 100,000 in 2016 from 667 per 100,000 in 2012 nationwide. Alongside this increase of sales there was a reported average decrease of 48% regarding the prices of e-cigarette products(10).

Database results

The YRBS finds the percent increase of e-cigarette use in teens compared to the national average in Colorado, Alaska, Washington DC, Nevada, and California to be 4.1%, 9.6%, 3.8%, 0.2%, 6.1%, respectively. While the percent decrease compared to the national average in Massachusetts and Maine to be -5.2% and -4.5% (11)(Figure 1).

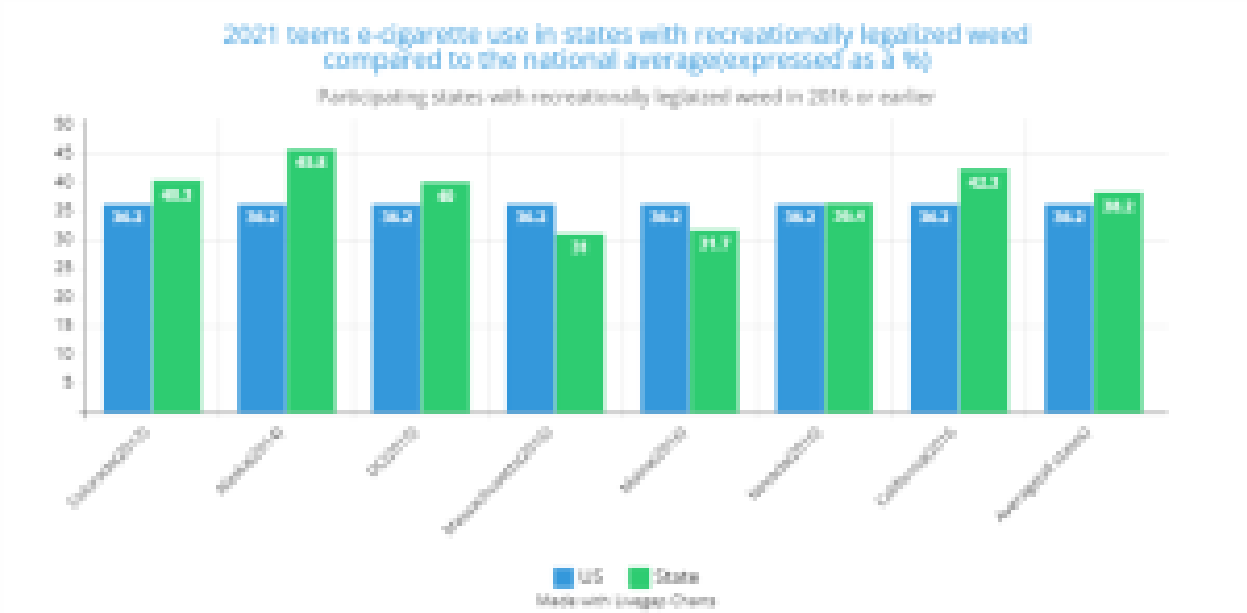

Figure 1. The graph shows results of the YRBS. The years next to the names of states refer to the year the state recreationally legalized weed. The two columns on the furthest right refer to the average of all the states shown on the graph(11).

The Healthy Kids Colorado Survey shows an increase in teenage e-cigarette use following the legalization of weed in 2012 (Figure 2). In 2013, the percentage of students that answered “yes” if they have ever used an e-cigarette product was 15.1%. This proportion increased to 46.2%, 44.2%, 45.9%, 30.3% in the years 2015, 2017, 2019, 2021 respectively(12).

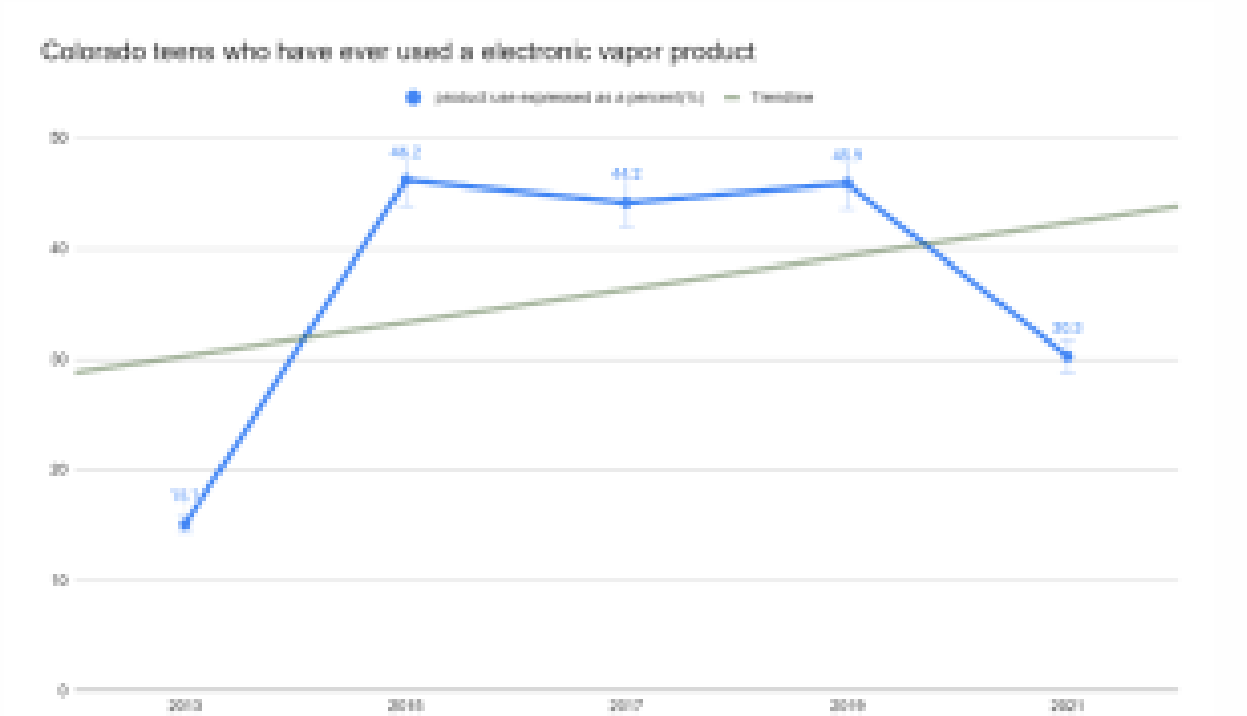

Figure 2. The percentage of Colorado teens that have ever vaped from 2013-2021. The trendline demonstrates the overall change of percentage values each year the survey was conducted(12).

DISCUSSION

There has been extensive research into the damages of teen e-cigarette-based nicotine addiction. However, the research for identifying specific causes of e-cigarette-based nicotine addiction has been limited to mostly targeted marketing and underwhelming education about the risks of e-cigarettes. We believe we are the first study to attempt to link marijuana use to a perceived lack of risk perception as a potential cause to explain the increase in adolescent e-cigarette use.

It is clear that within the last decade, e-cigarette use in teens has increased substantially nationwide from 3 to 27% in 4 years.(8). E-cigarette retail sales increased nationwide from 667 to 1547 per 100,000 between 2012-2016(10). Specifically, states with recreationally legalized marijuana have higher teen e-cigarette use than states without recreationally legalized marijuana(6). Data from the YRBS supports this conclusion as states that have had marijuana legalized for over 7 years have a higher e-cigarette use than the national average (2% increase for recreationally legalized

marijuana). When focusing on Colorado specifically, we can see an increase of 4.1% higher than the national average(11). Looking at longitudinal trends in Colorado, we can see an approximately 100% increase in teen e-cigarettes following the legalization of marijuana between late 2012 to 2021(12). This issue is not state specific; California, another weed-legal state, has also demonstrated increased e-cigarette use between 2014-2018(55.5%) and also sits higher than the national average(6%) in 2021(9).

Following legalization in Colorado, there has been a massive increase in overall marijuana use(13). According to the Colorado Department of Revenue, since initial legalization, 78% of marijuana sales have been retail-based (14). These retail sales include physical and online store options. Therefore, we can infer that with the increase in demand, there is an increase in supply in the form of more stores that sell marijuana products. Based on a study from Berg et al., the managers/owners of vape shops agree that many e-cigarette consumer base shares with the marijuana use consumer base. Therefore, many managers/owners are willing to sell both at the same store(15). From these previous assertions, we can infer that from the legalization and further increase of marijuana use, there has been a sharp increase in demand, leading to more retail stores that often sell both marijuana and e-cigarette products. According to a study done by Broman, the increased availability of substances during adolescence shows an increase in substance use for the adolescents exposed(16). In conclusion, existing research and data suggest that a potential cause of e-cigarette use in teens stems from the legalization of marijuana and its subsequent increase in demand, which causes the availability of similar products, such as e-cigarettes, to increase. This increase in supply leads to more availability that causes adolescents' use of e-cigarettes to increase.

<u>Limitations</u>

A limitation of this study is that we could not effectively compare e-cigarette trends between states. This is because most statewide surveys differed in their collection methods, questions, sample sizes, and time when data was collected. Therefore, it could not be determined how Colorado's increased rate of teen e-cigarette compared to other marijuana-legal states or marijuana-illegal states.

Another limitation of the study is that the link between the increased availability of substances and the further use of the substance in teens does not focus on the exact scenario of our paper(16). Instead, it primarily focuses on increasing the availability of substances in a household instead of increasing availability in the overall environment. However, we still used this source since an article by the National Institution of Drug Abuse corroborates a similar relationship between adults and heroin use, showing a higher rate of heroin use following the increase in availability(17).

Future Studies

New research studies should focus on addressing the limitation of the inconsistent sampling methods between states. To do this, creating a standardized survey that can run in consistent timeframes in each state and nationally is recommended. With potential information from this method, more accurate correlations can be made between the legalization of marijuana and an increase in teenage e-cigarettes at state and national levels.

The psychological relations between the increase in the availability of nicotine products and the potential subsequent increase of use in teens should also be further explored. To our knowledge, there has not been an in-depth study focusing on this relationship with a focus on nicotine products.

Conclusion

We have found strong evidence to suggest a potential cause of nicotine addictions in teens stemming from the legalization of marijuana. With further research, this concept can be further strengthened. Therefore, legislatures should consider the increase in nicotine addiction among teens as a potential side effect of legalizing marijuana. Systems should be put in place to minimize this consequence when considering the legalization of marijuana.

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Varsha M. Ramachandran

Peer Pressure and Social Media Impact of Smoking/Vaping and its Neuroscience Correlation

ABSTRACT

This research investigates the correlation between peer pressure, social media influence, and smoking/vaping behavior, with a focus on the neuroscience behind these behaviors. Through a literature review, the study explores the impact of peer pressure and social media on smoking and vaping behaviors among adolescents and young adults. Findings reveal that these factors significantly contribute to smoking/vaping behaviors. The study emphasizes the long-term consequences of nicotine exposure during adolescence on brain development and function, highlighting the disruption of nicotinic acetylcholine receptors and changes in neurotransmitter release and reward-related brain regions. The research underscores the need for accurate information, interventions addressing social norms and peer influence, and regulation of e-cigarette portrayal on social media, understanding the underlying neuroscience mechanisms to mitigate the negative health consequences of smoking/vaping.

INTRODUCTION

Electronic cigarettes (e-cigarettes) have increased exponentially among adolescents [7]. The increasing popularity of e-cigarettes, or vaping, among adolescents and young adults is problematic due to the potential associated health risks. The rise of vaping has prompted researchers to investigate the factors contributing to its prevalence. One such factor is the influence of peer pressure and social media [5] on smoking and vaping behavior. This research paper explores the correlation between peer pressure, social media validation, and the neurological rewards of nicotine in amplifying the propensity for smoking and vaping in modern society.

Existing research highlights the association between social media use and e-cigarette initiation among US adolescents [1] and found that exposure to nicotine product placement increases the likelihood of e-cigarette use among young adults [2]. Furthermore, research shed light on the misleading nature of e-cigarette marketing, which often portrays these products as less harmful than traditional cigarettes and as aids for smoking cessation [3].

However, there is no evidence supporting their effectiveness in quitting smoking [3]. Research also found that social status, as measured by the number of friends and leadership among peers, alone did not significantly impact e-cigarette use. However, perceived social impact, or beliefs about the social consequences of e-cigarette use, and peer influence increased the likelihood of accepting a cigarette and increasing the frequency of use. Those using e-cigarettes more frequently and those with friends using e-cigarettes perceived e-cigarette use as having a positive social effect [8]. Lastly, a study demonstrated that adolescents who use nicotine are more likely to initiate and continue using other drugs, emphasizing the potential long-term consequences of nicotine exposure during adolescence [6].

While the influence of social media, peer pressure, and underlying neural mechanisms were studied in isolation, we aim to examine the interplay between peer pressure, social media validation, and the neurological rewards of nicotine and provide valuable insights into the underlying mechanisms that drive smoking/vaping behavior in modern society. These findings will contribute to developing targeted interventions and preventive measures to mitigate the negative health consequences of smoking/vaping among adolescents and young adults.

I hypothesize that the combination of social media validation and the neurological rewards of nicotine contributes to an increased likelihood of engaging in smoking and vaping behaviors. Reviewing existing literature, this paper seeks to improve understanding of the complex mechanisms underlying smoking and vaping habits and inform potential interventions to address this public health concern. In conclusion, this research paper seeks to shed light on the complex relationship between peer pressure, social media validation, and the neurological rewards of nicotine in influencing smoking/vaping behavior. By addressing the gaps in existing literature, we can gain a deeper understanding of the factors contributing to the prevalence of smoking/vaping and develop effective strategies to combat this growing public health concern.

METHODS

I conducted a scoping literature review to gather existing research on the relationship between social media validation, nicotine rewards, and smoking/vaping behaviors. I searched PubMed and Google Scholar using the

keywords “social media,” “peer pressure,” “nicotine rewards,” “smoking,” and “vaping.”

I included studies that focused on the influence of social media on smoking and vaping behaviors, as well as studies that explored the neurological rewards of nicotine. I also considered studies that examined the role of peer pressure and social influence in smoking and vaping initiation. I focussed the search on recent papers by limiting the search to the last 10 years to ensure the most recent and relevant research is included.

The literature review included quantitative and qualitative studies, review articles and meta-analyses. I excluded opinion pieces, editorials, letters to the editor, and other non-peer-reviewed articles.

The research paper is limited to studies published in English and may not include all relevant research on the topic. Additionally, the paper does not include primary data collection or analysis but instead synthesizes existing literature to comprehensively understand the topic.

RESULTS

Users on social media such as Facebook, Google Plus, YouTube, LinkedIn, Twitter, Tumblr, Instagram, Pinterest, or Snapchat were classified into three categories: never users, non-daily users, and daily users. Social media use is associated with increased susceptibility and initiation of e-cigarette use among adolescents[1]. Exposure to tobacco content on social media may encourage tobacco use initiation and normalize tobacco use behaviors among regular social media users, like adolescents and young adults [4]. Exposure to tobacco content in social media had greater odds of reporting lifetime tobacco use, past 30-day tobacco use, and susceptibility to tobacco use amongst never-users [4]. Exposure to nicotine product placement in marketing is associated with increased use among adolescents. E-cigarette marketing often portrays these products as less harmful than traditional cigarettes and as aids for smoking cessation, despite no evidence supporting their effectiveness in quitting smoking [3].

Social status is considered in two forms. One is an individual’s perceived number of friends. This measure indicates perceived popularity. The second form of social status, leadership among peers, refers to the degree to which

an individual controls or directs the actions of a peer group (i.e., choosing what to do). Results showed that social status alone did not have any significant association with e-cigarette use. However, those reporting using e-cigarettes more frequently, as well as those reporting more friends using e-cigarettes, perceived e-cigarette use as having a positive social impact. The perceived social impact was positively related to the frequency of e-cigarette use and the likelihood of accepting an e-cigarette offered by a friend [8].

Nicotine use during adolescence can disrupt the normal development and function of nicotinic acetylcholine receptors in the brain, leading to changes in neurotransmitter release and reward-related brain regions. This can impact reward regulation and cognition and increase the reinforcing effects of other drugs. Furthermore, nicotine exposure during adolescence can have long-lasting effects on brain chemistry and function. It can lead to persistent increases in deltaFosB, a protein associated with reward, in the nucleus accumbens. It can also impair GABA signaling in the ventral tegmental area and alter gene expression in reward regions. These changes in brain function and behavior can increase the vulnerability to substance abuse and addiction later in life [6].

These findings suggest that the combination of social media validation, being with other users, and the neurological rewards of nicotine contributes to an increased likelihood of engaging in smoking and vaping behaviors which in turn increase the vulnerability to substance abuse and addition to other drugs later in life.

DISCUSSION

The objective of this study was to investigate the correlation between peer pressure, social media influence, and smoking/vaping behavior, with a particular focus on the neuroscience underlying these behaviors. The findings of this research provide valuable insights into the interplay between social media, peer pressure, and the neurological rewards of nicotine in shaping smoking and vaping behaviors among adolescents and young adults.

Social media platforms have emerged as powerful influencers in shaping smoking behaviors. Daily social media use among adolescents who never

used e-cigarettes was associated with a higher likelihood of being susceptible to e-cigarette use, past e-cigarette use, and current e-cigarette use [1]. Additionally, the influence of online social networks through platforms such as YouTube can promote the perception that e-cigarette use is socially accepted behavior, further contributing to susceptibility among young people [9]. Therefore, it is crucial to address the role of social media and peer pressure in preventing adolescent smoking and vaping behaviors. We can leverage social media platforms to create engaging and informative campaigns that highlight the negative health effects of smoking and vaping, while also promoting positive alternatives and providing support for those looking to quit. The influence of social media on smoking behaviors highlights the importance of regulating the portrayal of smoking-related content and providing accurate information about the risks associated with smoking.

Peer pressure is another significant factor contributing to vaping behaviors among adolescents and young adults. The study found that social status, including the perceived number of friends and leadership among peers, did not directly correlate with e-cigarette use. However, individuals who reported using e-cigarettes more frequently and had friends who also used them viewed e-cigarette use as positively affecting their social lives. This perception was linked to the higher frequency of e-cigarette use and a greater likelihood of accepting an offered e-cigarette from a friend [8]. Interventions targeting peer influence and promoting resistance skills can help mitigate the impact of peer pressure on vaping behaviors.

The neuroscience aspect of smoking and vaping behaviors reveals the complex interplay between the rewards of nicotine and the brain’s response. Research shows that nicotine activates the brain’s reward system, leading to feelings of pleasure and reinforcing the behavior of smoking or vaping [6]. This reward system plays a significant role in the development and maintenance of addiction [6]. Nicotine stimulates the release of dopamine in the brain, a neurotransmitter associated with feelings of reward and reinforcement [6]. This reinforcing effect can make smoking or vaping behaviors more difficult to quit or resist. Understanding the neurobiology of smoking and vaping behaviors allows for the development of targeted interventions that address the underlying mechanisms driving addiction. Chronic nicotine exposure can lead to changes in the brain’s reward pathways, reinforcing the addictive nature of smoking and vaping [6]. The

neurological effects of nicotine highlight the importance of understanding the underlying mechanisms to develop targeted interventions for smoking cessation.

The interplay between social media, peer pressure, and the neuroscience of smoking and vaping behaviors is evident. The portrayal of smoking as a desirable behavior on social media can amplify the influence of peer pressure and increase the likelihood of engagement in smoking and vaping behaviors. The neurological rewards of nicotine further strengthen the addictive potential of smoking and vaping, making it challenging to quit. It is important to consider factors such as peer pressure, social media exposure, and family influences when addressing interventions for young adults. Young adults may feel pressured to engage in risky behaviors such as substance abuse or dangerous activities to fit in with their peers, influenced on social media, leading to feelings of inadequacy or the desire to conform influenced by the values, beliefs, and expectations of their family members, which can impact their decision-making and behavior. By understanding these interpersonal influences, strategies like peer support can be implemented to reduce vaping behavior. Additionally, limiting celebrity endorsements of e-cigarette products could help in reducing positive perceptions of vaping within social networks. Therefore, it is crucial that comprehensive vaping interventions for young adults incorporate these interpersonal influences, including peer pressure and social media exposure [9].

In conclusion, this study highlights the significant influence of social media and peer pressure on smoking and vaping behaviors among adolescents and young adults. To effectively address and target these behaviors, interventions need to take into account the interplay between social media, peer pressure, and the neurological rewards associated with smoking and vaping. By understanding these influences, interventions can be designed to effectively mitigate the impact and reduce the prevalence of vaping among young adults. Furthermore, implementing strategies such as peer support and limiting celebrity endorsements of e-cigarette products can significantly contribute to reducing the positive perceptions of vaping within social networks.

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Meghna Ramaswamy

The Neurological Problems Caused By Scoliosis And The Possible Treatment Options

ABSTRACT

This literature review explores the neurological effects of scoliosis and the effectiveness of different treatments. It highlights that scoliosis can cause several neurological problems such as radiculopathy, which can impair motor control and cause tingling sensations. This review suggests that treatments like bracing, surgery, and exercise can help reduce these neurological symptoms. However, the effectiveness of each treatment varies depending on the patient, highlighting the importance of consulting medical professionals for individualized treatment plans. The review emphasizes the need for more studies to further understand the neurological effects associated with scoliosis. It also mentions that limited studies with relevant data hinder the research findings. Nonetheless, methods that have been proven to improve the Cobb angle and dexterity can potentially benefit the neurological health of scoliosis patients. The review aims to provide information that can educate patients on the importance of scoliosis treatment and its impact on their quality of life.

INTRODUCTION

Scoliosis is well identified as a physical deformity causing excessive curvature of the spine. Scoliosis is further categorized by the extent of the curvature, which is done using a measurement called the Cobb angle. A Cobb angle greater than 10 degrees is considered scoliosis. Idiopathic scoliosis is the most common type of scoliosis and is most commonly diagnosed in adolescents age 11 and older. It is more prevalent in girls than boys and is more likely to be found in those who have close relatives also with scoliosis. Screening can be done in many ways with the most common ways being physical examinations or x-rays. Severe or untreated scoliosis can affect the life span of patients, although this is dependent on the patient’s curve severity. In some cases scoliosis affects their quality of life and neurological health. This paper discusses the neurological problems that impact patients with moderate to severe cases of scoliosis, defined as a Cobb angle of 40 degrees or higher. The problems include conditions such as tension headaches, issues with the recirculation of cerebrospinal fluid (CSF),

radiculopathy, and compressed nerves. All forms of scoliosis can lead to life altering neurological symptoms. Adolescent Idiopathic Scoliosis (AIS) is prevalent with about 2-2.5% of adolescents diagnosed with it [1]. Because it is so common, there have been many types of treatment explored for the deformity correction of scoliosis including core stabilization exercises, Schroth exercises, surgery, and bracing. These methods have been proven to improve the Cobb angle, dexterity, and in turn help with the neurological health of patients. However, treatment plans may vary depending on the individual patients age, condition severity, curvature location, and condition type [2].

This review aims to explain the neurological issues relevant to scoliosis as well as the mitigation of scoliosis related issues through treatment. It can be used to help patients better understand scoliosis and the potential treatment they can receive, as well as help identify the gaps in the research of scoliosis. This data can be used to educate patients on the importance of scoliosis treatment and the impact treatment has on patient quality of life.

METHODS

This review employs published studies found on the Pubmed database and independent research articles written by reputable medical professionals. For the research question defined as "How can early diagnosis along with treatment affect the severity of scoliosis neurologically?," key terms such as "Scoliosis back pain", "Scoliosis nerve pain", "Scoliosis risk factors", "Untreated scoliosis", "Undiagnosed scoliosis", "Scoliosis brain", "Scoliosis symptoms", "Scoliosis side effects", and "Brain damage scoliosis" were used to search on Pubmed. Phrases such as "Scoliosis effect on the nervous system" and " How scoliosis affects that body?" were used when conducting internet searches. Seven peer-reviewed articles were selected based on authenticity, relevance to research questions and being published within the last 15 years.

RESULTS

Both Nalda [2] and Lowenstein [3] found that there were numerous neurological problems caused by scoliosis. Both studies explained that it causes radiculopathy which is defined as a painful tingling sensation capable of impairing fine and gross motor control. Both studies stated that the

prevention of recirculation of CSF can leave the brain with inadequate levels of CSF surrounding it, leading to tension headaches and sometimes migraines.

Romberg et al [4] and Weinstein et al [5] talked about the effectiveness of bracing and how it affects scoliosis patients. Romberg et al [4] focused primarily around the strength and dexterity of patients who underwent bracing and was able to find that early bracing was significantly more effective by comparing Early Onset Scoliosis (EOS) patients to AIS patients. Weinstein et al [5] focused on AIS patients and concluded that bracing reduced the progression of severe curvature of the spine. The study further concluded that the longer the braces were worn the more the benefit, including a greater likelihood of reaching skeletal maturity with less than a 50 degree curve and less warrant for surgery.

Smith et al [6] found that within a two-year follow up evaluation, patients who underwent surgical treatment had reduced back pain and disability which improved overall patient health. Improvements were measured through a Numerical Rating Scale (NRS), determined by patient numerical estimation of their back pain. The mean NRS in non-operative patients did not significantly improve while the mean NRS in those with operative treatment were found to have significantly improved. The NRS was lower for patients who underwent surgery indicating that surgery can reduce back pain and improve patient quality of life.

Both Kocaman et al [1] and Anwer et al [7], found that exercises improved Cobb angle, angle of trunk rotation, peripheral muscle strength, thoracic kyphosis angle, lumbar lordosis angle, spinal mobility, and quality of life. Kocaman researched two specific exercise treatments including the Scroths exercise and core stabilization exercises. Schroth method was found to reduce Cobb angle and improved spinal mobility, while the core exercises were more effective with improving peripheral muscle strength. The study done by Anwer provides evidence that a broader set of exercises improved quality of life for those with scoliosis. All studies showed that there are many neurological problems associated with severe cases of scoliosis and the treatment options that can be used to improve high-risk scoliosis and its neurological symptoms.

DISCUSSION

Information gathered from both Kocaman et al [1] and Nalda [2] suggests that Scroths exercises might be most successful for boosting overall health and reducing neurological issues due to scoliosis. Nalda [2] concludes that symptoms including radiculopathy, nerve problems, impairment of fine and gross motor control, decreased levels of CSF surrounding the brain, and tension headaches are related to the curvature of the spine with the symptoms escalating with the severity of the curve. Because the Scroths exercise has demonstrated it reduces the Cobb angle, it can be more useful for treating these neurological symptoms than other exercises. Additionally, as Anwer et al [7] implies, many forms of exercise are beneficial for scoliosis and exercises are generally useful for patients with scoliosis.

From the information gathered from both Smith et al [6] and Lowenstein [3], surgical treatment is recommended to reduce back pain by treating curvature of the spine in scoliosis. As explained in Lowenstein [3], the curvature of the spine compresses nerves and creates radiculopathy that leads to pain for those with scoliosis. Surgery to treat scoliosis reduces the curvature of the spine thus reducing the issues causing the back pain. Information from studies Nalda [2] and Weinstein et al [5] suggest that bracing can decrease the risk of curve progression and reduce the possibility of neurological symptoms. It is concluded in Weinstein et al [5] that bracing has a high success rate with a positive correlation between the number of hours worn and its success. The larger the Cobb angle, the more the likelihood and severity of these neurological problems increases. Therefore, by reducing curve progression bracing lessens the risk of these neurological problems.

Although both Romberg et al [4] and Weinstein et al [5] conclude that bracing is beneficial, Romberg et al [4] emphasizes that it is best to treat patients by bracing when they are younger. Romberg et al [4] even concludes that EOS patients have better results than ASI patients with bracing. Each study listed a multitude of reasons as to why this occurs. In Weinstein et al [5] only AIS patients were studied for bracing without any comparison made with age, making it unclear how beneficial bracing actually will be or what ages bracing should be used. For these reasons the effectiveness and age ranges for bracing should be more explored and further researched.

This literature review suggests that with treatments such as bracing, surgery, and exercise we can reduce many neurological symptoms of scoliosis patients. Additionally, the effectiveness of each treatment is dependent on the patient and treatment plans should be discussed with medical professionals. However, there were limited studies with relevant data, meaning the research findings are repressed. There were a restricted amount of sources talking about neurological symptoms of scoliosis. Although many databases were inspected, few articles actually reported the neurological damages. For these reasons, more studies exploring the neurological problems in scoliosis need to be conducted, including those that investigate the presence of neurological problems in scoliosis and its impact on a patient's quality of life.

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Ryma Saha

How Can Artificial Intelligence Be Used to Diagnose Autism Spectrum Disorder?

ABSTRACT

Autism spectrum disorder (ASD) is a neurodevelopmental disorder that is characterized by difficulties in social interaction and repetitive patterns of behavior. As ASD cases increase worldwide, there is an increased need for innovative and effective interventions. This literature review aims to assess the potential of Artificial Intelligence (AI) to diagnose ASD. I searched Google Scholar and PubMed using the key search terms Autism Spectrum Disorder diagnosis, MRI, facial recognition, emotion in speech, and fMRI to identify four relevant studies, excluding any studies before 2017. The results of the four reviewed articles revealed that AI has a strong potential in improving the assessment of ASD. However, data limitations, differing views, and lack of collaboration between clinicians, must be addressed before AI can be used to effectively diagnose ASD.

INTRODUCTION

Autism spectrum disorder (ASD) is a neurodevelopmental disorder that affects people's behavior and the way they function, ranging from mild to severe. ASD affects social communication and interaction. Challenges include not understanding sarcasm, taking things literally without understanding abstract concepts, and having difficulty processing information [1]. Signs of ASD also include repetitive and restrictive behavior. According to the CDC, nearly 1 of 36 children in the US have been diagnosed with ASDas of 2023 [1]. ASD is four times more commonly seen in males compared to females and has occurred in all racial, ethnic, and socioeconomic groups [1]. Globally, an estimate of 75 million people were reported to have ASD [1]. The diagnosis of ASD is difficult due to the lack of any specific medical tests or blood tests [1]. Currently, the diagnosis of ASD mainly depends on behavioral, parent-report, and interview assessments,which are very subjective, and time-consuming [1]. Two common tools used by medical specialists to diagnose ASD are MCHAT-R and ADOS [2]. However, these tools often miss the diagnosis or overdiagnose the case due to multiple barriers such as language barriers. Using this screening (check) tool, ASD could be detected as early as 18-months-old, but diagnosis has shown to be

more reliable at age 2-years-old and up. However, many children do not receive their final diagnosis until they are much older due to the shorter availability of psychologists, psychiatrists, and developmental pediatricians [3]. Early diagnosis may lead to early intervention which could be beneficial to both patient and family.

As technology advances, medical professionals continue to see artificial intelligence (AI) as a potential tool for the diagnosis of ASD. AI is a machine or software that performs and mimics human intelligence to perform cognitive functions. AI could assist in the diagnosis of ASD efficiently using the algorithm to analyze large datasets of developmental milestones, behavioral patterns, and medical records. These can lead to early screening, diagnosis, and intervention, which is crucial in order to improve the outcomes of individuals with autism. Although AI has shown promising potential in assisting the diagnosis of ASD, it does not replace the expertise of trained professionals. The purpose of this paper is to assess the combination of currently used screening tools and how AI could possibly speed the process of the diagnosis of ASD and increase the accuracy, leading to early intervention.

METHODS

The inclusion criteria included AI being used to diagnose ASD. The articles stated the AI algorithms used to conduct the study such as using brain imaging using fMRI, emotion detection in speech, facial expression detection, and behavioral observation using skeletal movement. The articles also stated how participants of different gender and ethnicity were used in the studies conducted.

Articles that used specifically AI algorithms such as brain imaging using fMRI, emotion detection in speech, facial expression detection, and behavioral observation using skeletal movement were also included. Finally, The article/study includes that they tested both males and females as well as all ethnicity groups in order to determine the prevalence rate in gender and ethnicity groups.

The exclusion criteria of this study states the study must not be earlier than 2017. This is because AI was not implemented at the time therefore there is no data available. The study must also not test patients diagnosed with chronic conditions such as seizure disorder or hearing impairment. It may

interfere with the AI algorithm result interpretation due to similar behavior. Finally, case reviews were excluded from this scopus review.

Based on the inclusion and exclusion criteria, four articles were reviewed from databases such as PubMed, Google Scholar using the key search terms: AI, Autism Spectrum Disorder diagnosis, MRI, facial recognition, emotion in speech, and fMRI. Using these keywords, four articles were chosen for this Scoping review amongst all others.

RESULTS

Four articles were chosen to investigate the results of the different studies conducted [4-7]. Several computer aided design systems (CADS) were used based on AI techniques such as Machine Learning and Deep Learning (ML and DL methods) by using MRI modalities (sMRI and fMRI) which was less time consuming. Among various MRI modalities data sets Autism Brain Imaging Data Exchange (ABIDE) was the most complete and freely available database for MRI. Different pipelines methods were used for preprocessing of data sets, ABIDE was found to be the most popular pipeline. The most common method used for feature reduction/selection was principal component analysis (PCA) due to its ability to find a minimal number of features required for classification. For classification, which is the final step, support vector machines (SVM) have been widely used with AUC of 62%-99.52%. Random Forest (RF) classification method had the highest accuracy rate of100% and had 100% sensitivity and specificity followed by LR. (Linear Regression). ML method is more time consuming and complex to design than DL method, because DL method automatically performs the steps from deep layers feature extraction to classification and requires little human intervention to function properly. Combination of both techniques may give more accurate results in diagnosing ASD.

A study by Song, et. al. reviewed thirteen studies using ML techniques and different classification methods used for multiple behavioral observation in ASD and typically developing (TD group) of children [4]. Among all behavioral symptoms eye tracking was the most important characteristic with accuracy of 88.51% when compared to other behaviors. Typically the developing group spent more time looking at the right eye while the ASD group spent more time on the left eye. This paper reviewed the eye tracking scan path in diagnosing ASD by using ML technique where 59 children participated that included both normal and ASD children. They watched a set

of photography and age appropriate video. Eye tracking scan paths were converted to visual representation as a set of images when convolutional neural network (CNN) was used to classify the image. Results showed that an eye tracking scan path was able to differentiate children from ASD to non ASD groups.

CNN model can provide prediction accuracy of 90%. In this study eye movement velocity was compared with CARS (Childhood Autism rating Scale) scores which revealed possible correlation between the level of autism and eye motion velocity [5]. Individuals with high autistic traits tend to have shorter and less frequent saccades compared to low autistic traits. The comparison between eye movement velocity with CARS helped to mitigate the effect of outliers in eye tracking experiments.

Table 1: Article Machine Learning Models and Outcomes

Source	Machine Learning Model	Outcome
Megerian et al.	Gradient boosted decision tree machine learning algorithm	The algorithm enabled timely diagnostic evaluation with a high degree of accuracy for a third of the primary care sample.
Song et al.	Support Vector Machines (SVM)	SVM has the highest accuracy in analyzing behavioral symptoms.this study also showed this classification method were able to exclude duplicate items to reduce the amount of time and effort needed

		in the assessment process.
Cillia et al.	Machine Learning – scan paths system used to transform eye tracking into a set of visual images and then the CNN model used to perform image classification	Eye tracking could be an objective tool for diagnosis of ASD, CNN model could provide prediction accuracy close to 90%, study also revealed that individuals with high autistic traits have shorter and less frequent saccades compared to others with low autistic trait.
Parikh et al.	Neural Network Model	Personal characteristics data plus neuroimaging provided the highest sensitivity and specificity for neural network models to predict ASD diagnoses.

DISCUSSION

The results of the reviewed papers showed the use of AI in the diagnosis of ASD has shown promising results [4-7]. ABIDE was the most popular method to retrieve data sets [4-7]. DL technique was proven to be superior to ML technique [4-7]. SVM classification was the most commonly used method among all other methods [4-7]. Eye tracking characteristics were proven to be the best method compared to other behaviors [4-7]. It was also well accepted by both clinicians and parents because of the ease of administration [4-7]. The biggest challenges of each of these studies was gathering standardized data sets, data complexity and use of a wide variety of classification methods [4-7]. These challenges required collaboration

between AI researchers, clinicians and individuals with ASD to develop effective and responsible AI based diagnostic tools.

These findings suggest that AI has the potential to improve the accuracy and efficiency of ASD diagnosis leading to earlier interventions and support for individuals with the conditions. Based on the study reviewed, I would recommend that clinicians should implement AI for the diagnosis of ASD in addition to standard screening methods for earlier diagnosis and intervention. Future study can focus on the development of more accurate and reliable AI algorithms for early detection and diagnosis of ASD. Another area of exploration could be the integration of AI in the existing diagnostic tools. This could involve the development of AI powered screening tools and interactive systems that can assist clinicians in making more accurate and efficient diagnosis in the clinics.

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Zohaib Sarfraz

The Rising Trend of Myocardial Infarction in Young Individuals

ABSTRACT

Myocardial infarctions (MIs), commonly known as heart attacks, have been increasingly affecting younger individuals, raising concerns among researchers, healthcare professionals, and policymakers worldwide. This review investigates the concerning trend of increasing MIs in younger individuals and explores its implications for public health. MIs in those under 45 account for 10% of acute MIs in the United States, mainly caused by atherosclerotic coronary artery disease. Smoking is the most prevalent risk factor in young adults experiencing heart attacks. Gender differences in symptom presentation and risk profiles were observed, emphasizing the need for tailored interventions. Despite being younger, very young MI patients showed similar outcomes to older age groups, such as death, or prolonged health problems. The study underscores the urgency of addressing this emerging health challenge through targeted interventions, recognizing atypical symptoms, and individualized risk assessments to reduce the burden of heart attacks in the younger population.

INTRODUCTION

Myocardial infarctions (MI) are when a blockage prevents blood reaching to the heart. MI's are a prevalent and serious medical condition that affects millions of individuals worldwide. Traditionally associated with older age groups, there has been a growing concern regarding the increasing incidence of heart attacks in younger people (1). This alarming trend has sparked significant interest among researchers, healthcare professionals, and policymakers alike, necessitating a thorough investigation into the underlying causes, risk factors, and potential implications for public health. The purpose of this review is to provide a comprehensive examination of heart attacks occurring more frequently in younger individuals. By synthesizing and analyzing relevant studies, data, and expert opinions, this paper aims to shed light on the factors contributing to this trend and explore its potential implications for affected individuals, healthcare systems, and society at large. Moreover, it seeks to identify gaps in current knowledge, propose areas for future research, and ultimately, contribute to the development of effective preventive strategies and interventions.

METHODS

In this literature review, a comprehensive search was conducted to identify relevant articles published within the last five years (2018-2023), investigating heart attacks in younger individuals. The search strategy involved using databases such as PubMed, Google Scholar, and Web of Science with specific keywords related to heart attacks, myocardial infarction, risk factors, symptoms, and surveys on heart attacks. Articles were included if they focused on comparing age groups, studying risk factors and symptoms, examining young patient populations, or providing general and important information on heart attacks/myocardial infarction. Only peer-reviewed articles were considered to ensure the reliability of the findings.

After screening titles and abstracts and subsequently reviewing full-text versions, a total of relevant articles were selected for data extraction. The synthesis of findings organized into themes related to heart attacks in younger individuals, including prevalence, risk factors, symptoms, and studies specific to young patients. Additionally, the review aimed to offer a broader understanding by including general and important information on heart attacks/myocardial infarction.

RESULTS

The results of this literature review reveal that MI in patients under the age of 45 accounts for a notable 10% of acute MIs in the United States, with the majority of cases occurring in men (3). Atherosclerotic coronary artery disease emerges as the leading cause, responsible for approximately 80% of these cases. Notably, smoking is the most prevalent risk factor in young adults experiencing MI. The relative number of MI cases in young patients is on the rise, indicating a concerning trend (3). Young women are also affected, with over 30,000 hospitalized with MI annually in the USA alone (4). This increase in MI incidence among younger women may be linked to factors such as metabolic syndrome, diabetes mellitus, and non-traditional risk factors like stress, anxiety, and depression (2).

Gender differences were observed in symptom presentation, with young women being less likely to present with chest pain compared to men, but young men, older men, and older women also occasionally presented

without chest pain (4). However, despite lower symptom severity, young women still reported chest pain and discomfort (4). When comparing very young MI patients (under 40 years old) with those aged 41 to 50 years, similarities were found in their risk profiles, with the exception of higher substance abuse prevalence and lower hypertension rates in the very young group (3). Nonetheless, despite being on average 10 years younger and having less extensive coronary artery disease, very young MI patients exhibited similar 1-year and long-term outcomes when compared with the 41 to 50 years age group (3).

DISCUSSION

After reviewing the data, a concerning trend becomes apparent: an increase in myocardial infarctions among the younger population. Atherosclerotic coronary artery disease is identified as the principal underlying cause, with smoking emerging as the most prevalent risk factor among young adults experiencing MIs. This underscores the imperative for targeted interventions to promote smoking cessation within this demographic.

The study highlights significant gender differences in symptom presentation and risk profiles among young individuals experiencing heart attacks. Young women are less likely to present with chest pain compared to men but report a higher number of symptoms overall (4). These findings emphasize the importance of recognizing atypical symptoms, like chest pain and discomfort, and addressing gender-specific risk factors to ensure timely diagnosis and appropriate management in young women (4).

Comparisons between very young MI patients (under 40 years old) and those aged 41 to 50 years reveal similarities in risk profiles, suggesting that age alone may not be the sole determinant of MI outcomes (3). Substance abuse prevalence and hypertension rates differ between the two age groups, highlighting the need for individualized approaches considering various risk factors and comorbidities.

Overall, the results underscore the urgency of addressing the rising incidence of heart attacks in younger individuals. Preventive strategies should target smoking cessation, promote healthy lifestyles, and consider both traditional and non-traditional risk factors. Tailored interventions for gender-specific risk profiles and comprehensive assessments of individual

risk factors can significantly contribute to reducing the burden of MI in the younger population. By addressing these findings, public health efforts can effectively mitigate the impact of this emerging health challenge and improve outcomes for young individuals at risk of myocardial infarctions.

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Avighna Sastrula

Cardiovascular Disease Affected by Access to Healthcare in America

ABSTRACT

Background: Cardiovascular disease affects 40% of Americans. The purpose of this review is to see the linkage between access to healthcare and cardiovascular disease.

Methods: PubMed, and Google Scholar were searched using the following inclusion criteria:10 years, and had study designs of original research articles. Meta-analyses and literature reviews were excluded from the reviewed articles.

Results: Regular screenings prevented diseases a lot more which comes with more access to healthcare. Participants that had insurance were more likely to use screenings to their advantage to help them check for CVD. Participants who tend to take care of their overall health would find ways to get access to healthcare and decrease their risk of CVD.

Discussion: Using other healthcare methods such as medicare to help more people who need this to ensure that people get proper screenings to keep them from getting CVD. We need to see which areas need help more and supply those areas so people get equal opportunity to live better lives.

INTRODUCTION

Significance of the Problem

Cardiovascular disease (CVD) is predicted to affect 40% of Americans by 2030 [1]. It is also the leading cause of death in the United States [2]. CVD has been the leading cause of death since 1950 [2]. Health insurance plays a big role in how we are able to afford medical care. I hypothesize that the lack of access and ability to afford healthcare or health insurance impacts the ability to get screenings to check for and treat CVD.

Objectives

The aim of this paper is to determine how having access to healthcare or insurance will affect immigrants mainly women under the age of 50 who have unusual intervals of care.

METHODS

Search Strategy

PubMed and Google Scholar were searched using key search terms related to "cardiovascular disease," "healthcare access," and "prevention factors."

Inclusion and Exclusion Criteria

Articles were included if they specifically explored the link between healthcare access and cardiovascular disease with a focus on prevention factors, were published within the last 10 years, and had study designs of original research articles. Systematic reviews, meta-analyses, and case studies were excluded.

RESULTS

Alcalá et al., demonstrate that relationships between insurance status, usual source of care, and CVD prevention factors did not depend on the presence or absence of CVD clinical risk/disease [1]. Among those with no CVD clinical risk/disease, insurance and a usual source of care were associated with increased odds of healthcare utilization, screenings, and discussions about lifestyle factors [1]. The sample primarily consisted of individuals under the age of 50, predominantly women, with a high school education or less [1]. Having a regular source of care was associated with increased odds of physician visits, timely blood pressure and cholesterol screenings, as well as discussions about weight, eating, and exercise with healthcare professionals [1]. When insurance status was included in the model, all previously significant associations remained significant [1].

A study by Barghi et al., showed that participants with insurance had significantly higher odds of utilizing healthcare services, timely cholesterol screening, and discussions about exercise with healthcare professionals. However, insurance status was not associated with discussions about weight or eating habits, nor did it increase the odds of engaging in healthy behaviors [3]. Most participants were foreign-born, bilingual in English and Spanish, currently insured, and had a usual source of care [3].

While a study by Stacy showed that participants with a usual source of care also had increased odds of less frequent consumption of sugar-sweetened beverages [4]. Participants generally reported timely screenings for blood pressure and cholesterol, with varied engagement in healthy behaviors [4].

The inclusion of the usual source of care in the model rendered the association between insurance status and exercise discussions nonsignificant [4].

DISCUSSION

Healthcare Access and Utilization linked to Insurance Status

The significant association between insurance status and healthcare utilization aligns with existing literature highlighting the role of insurance in facilitating access to healthcare services. Insured individuals had higher odds of engaging in timely cholesterol screening and discussions about exercise with healthcare professionals [3]. However, the non-significant association between insurance status and discussions about weight or eating habits, when controlling for a usual source of care, suggests that factors such as accessibility and age play a role [1].

Usual Source of Care

The robust relationship between having a regular source of care and various healthcare utilization metrics underscores the importance of continuity in healthcare services. Individuals with a usual source of care had increased odds of physician visits, timely screenings for blood pressure and cholesterol, and discussions about lifestyle factors [4]. This finding aligns with the notion that consistent healthcare relationships contribute to more proactive preventive care.

Moderation by CVD Clinical Risk/Disease

The moderation analysis did not reveal significant differences in the relationships between insurance status, a usual source of care, and CVD prevention factors based on the presence or absence of CVD clinical risk/disease [4]. This suggests that the benefits of insurance and a regular source of care extend across different risk profiles, emphasizing their universal relevance in promoting cardiovascular health.

Implications for Public Health Policy

The studies' results underscore the importance of addressing healthcare access barriers to enhance cardiovascular health. Policies aimed at increasing insurance coverage and promoting the establishment of a regular source of care may contribute to better preventive care. Efforts to facilitate

discussions about lifestyle factors within healthcare settings could further enhance the impact of these policies.

Limitations and Future Directions

While this paper provides valuable insights, some limitations should be acknowledged. Studies included with a cross-sectional design limits the establishment of causality. Additionally, any self-reported data, especially regarding lifestyle behaviors, may be subject to bias. Future research could explore longitudinal data and incorporate objective measures to strengthen the evidence base.

Conclusion

In conclusion, this study contributes to the ongoing discourse on the interplay between healthcare access and cardiovascular health. The findings emphasize the need for holistic approaches in healthcare policies to address disparities and enhance preventive care, ultimately contributing to improved cardiovascular outcomes within diverse populations.

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Ankith Sureddi

How Race Impacts an Individual's Cardiovascular Health in the U.S.

ABSTRACT

Background: Gaps in resources and racial groups can cause increases in cardiovascular disease (CVD) risks and decreases in overall cardiovascular health. Unfair treatment such as discrimination, stereotyping, and social stigmatization can play a large factor in terms of increased risk of CVD between minority groups and races. Implicit bias and racial gaps have led to solutions such as health campaigns from not being as effective.

Methods: I used PubMed and Google Scholar to better understand and study how an individual's race can impact their cardiovascular health. I specifically studied various races in the U.S. and their cardiovascular health and risk for Cardiovascular Disease (CVD). I did not include any non U.S. populations in the research.

Results: The highest (as in the best and healthiest) cardiovascular health is found in white individuals compared to their black counterparts. Black Americans had some of the lowest levels of cardiovascular health and instead some of the highest CVD rates. Out of the Asian American cohort, Chinese Americans had the lowest prevalence of CVD compared to their Southeast Asian and Pacific Islander counterparts..

Discussion: The results show a correlation between race and CVD. Minority groups such as Black Americans face higher CVD risk because of low socioeconomic status, less resources, less opportunities, institutional racism, perceived/personally mediated racism, and internalized racism. More educational programs and training healthcare workers regarding health disparities will minimize the impact of health disparities.

INTRODUCTION

Significance of the Problem

Cardiovascular disease (CVD) is the leading cause of morbidity and mortality across the globe, making CVD a major public health issue [1]. One person dies every 33 seconds in the United States from CVD and every year, 805,000 people have a heart attack [2]. Race can impact an individual's cardiovascular health in the U.S. due to gaps in resources and access between racial groups. Black adults are more than twice as likely to pass away from cardiovascular diseases (CVD) compared to their white

counterparts [3]. Minority groups, especially Black Americans, in the U.S. face social stigmatization, leading to unfair treatment, such as discrimination and stereotyping [4]. The reason underserved minorities face disparities in terms of access to high-quality cardiovascular health care is related to neighborhood, poor diet quality, and physical inactivity [5]. The disparities faced by underserved minority populations in the United States need to be addressed to better the cardiovascular health of these communities. Public health campaigns, policy changes, and community outreach have all been done in the past to reduce CVD, but the issues that made some of these initiatives less efficient were due to racial gaps in health and implicit bias in healthcare [6].

Objectives

This paper will investigate how race impacts cardiovascular health outcomes by investigating disparities and the risk factors as well as the incidence of disease among various races in the United States. The paper aims to educate and provide more insights to promote equitable cardiovascular health regardless of race and to increase better healthcare practices and policy initiatives to reduce the health disparities in terms of CVD.

METHODS

I searched Pubmed and Google Scholar using the key search terms racism, cardiovascular health, United States, and language barriers. The inclusion criteria were studies that focused on populations in the U.S., and minority populations such as Hispanics, Black Americans, and Asians, immigrants, CVD risk also related to stress. Systematic reviews and meta-analyses were excluded.

RESULTS

Race impacts cardiovascular health in the U.S. through high blood pressure (which 75% of respondents reported), racial discrimination (32% reported), and depressive symptoms (50% reported) [7]. These impacts were due to stressors related to the navigation of manhood (especially seen in Black American men), having high expectations with lack of resources, managing healthcare needs despite fear of discrimination, and environmentally through the people one is surrounded by [7]. It was also found that white women have the highest cardiovascular health level [8]. After white women, it is followed by white men, black men, and then black women with the lowest

cardiovascular health level [8]. When observing older Black women, reports of recent stressful events correlated with CVD [9]. It was also found that there was a lower prevalence of CVD among Asian Americans while there was a higher prevalence among the Pacific Islander population in the U.S. Among the Asian race, Filipinos had the highest CVD prevalence while Chinese had the lowest [10].

DISCUSSION

The most significant results from the research showed that White individuals had overall better cardiovascular health compared to their Black American counterparts. The results show this is due to factors such as lack of resources, fear of discrimination, and the environment some of these minority groups are surrounded by impoverished communities in the U.S. This is also due to three types of racism Black Americans in the United States often face such as institutional racism, perceived/personally mediated racism, and internalized racism. Through these various types of racism minority groups, especially Black Americans, face outcomes such as limited socioeconomic mobility and opportunities, less access to resources, and poor living conditions. In terms of perceived/personally mediated racism can cause psychological reactions such as stress. There is also internalized racism, which is especially seen in the U.S., due to possible self-evaluations that could be negative and related to negative cultural stereotypes. These factors all largely play a role in impacting cardiovascular health. It was also found that in terms of Asian Americans, Chinese Americans had the lowest prevalence of CVD compared to their Filipino and Pacific Islander counterparts [10]. This could be due to many Asian Americans having higher education and more resources as a result. These results find a positive correlation between race and cardiovascular health due to racism and more resource accessibility. This research points to the need for programs to address disparities in cardiovascular health. This can be done by improving access to care by tailoring culturally relevant interventions, training healthcare professionals to be more competent and culturally understanding, promoting more screenings and checkups for CVD, and advocating for policies related to health equity promotion.

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Emma Sutandi

Loneliness Increases the Risk for Cardiovascular Disease

ABSTRACT

Cardiovascular disease has been the leading cause of death for more than 20 years. Loneliness is a form of stress that releases cortisol, a hormone that raises blood pressure, causing hypertension and in turn, myocardial infarction and stroke. The goal of this scoping review was to find how much the risk of cardiovascular disease increases due to loneliness. I searched PubMed and Google Scholar using the key terms loneliness, cardiovascular disease, stroke, myocardial infarction, heart disease, and social isolation. I excluded all literature reviews, any health outcome that wasn't a cardiovascular disease, and studies that were older than 10 years old. Overall, loneliness increased the risk of cardiovascular disease by 13-42% and increased the risk of dying from it by two times. People who had a low amount of close contacts were found to be 4.9 times more susceptible to myocardial infarction and 4.1 times more susceptible to stroke. In line with the hypothesis, loneliness substantially increases cardiovascular disease risk. However, more work needs to be done to address the lack of information on loneliness' impact on physical health, teens, as well as if the duration of loneliness changes outcomes.

INTRODUCTION

Cardiovascular disease (CVD) is the number one leading cause of death in the world, and it has been since 2001 [1]. On average, it kills around 17.5 million people worldwide, but the death rate increases at an exponential rate [1]. CVD refers to any kind of disease that's related to the heart or blood vessels [2]. One's lifestyle can have a substantial impact on the risk of getting a cardiovascular disease and also the risk of dying from it; these factors of one's lifestyle are called the social determinants of health which include economic stability, education, transportation, access to healthcare, and social surroundings [1]. That means that low social support or loneliness can cause cardiovascular disease because it's a form of stress, which releases a hormone called cortisol [3]. High levels of cortisol from prolonged or high levels of loneliness can cause high blood pressure or hypertension [3], which then can cause stroke and myocardial infarction, two dangerous forms of cardiovascular disease [2].

Loneliness affects 1 in 3 people in developed countries, with 1 in 12 people experiencing very high levels of loneliness [4]. I defined loneliness as the feeling of sadness because one's social life is not what one wants it to be – even though loneliness and social isolation are sometimes used interchangeably, social isolation is not having adequate social interactions. A person who has a very active social life can still feel lonely, making the two terms not interchangeable. Loneliness affects every age group but is most common in older adults because friends and family may have passed away, they may not spend as much time with their children, and lower physical ability prevents them from going out as much. In the past, loneliness has been ignored as a health problem and has been shoved aside in the eyes of public health officials, but especially after COVID-19 left us all isolated at home, loneliness is becoming a more prevalent problem [4], becoming the US Surgeon General Dr. Murty's priority for the health of the US. The goal of this scoping review is to see how much loneliness increases the risk for cardiovascular disease among older adults in different countries, which will bring light to the pressing problems that loneliness induces.

METHODS

To gather the data for this scoping review, key search terms like loneliness, cardiovascular disease, stroke, myocardial infarction, social isolation, and heart disease were entered into Pubmed and Google Scholar. The inclusion criteria were articles about loneliness' connection to cardiovascular disease and original research. The exclusion criteria were articles about diseases other than cardiovascular disease, any kind of review, and articles that were more than 10 years old.

RESULTS

To measure loneliness, the studies analyzed conducted a questionnaire including questions about one's social interactions like "How often do you get to interact with family?," "How often do you go out?," "Do you feel included," and most of all, "Do you feel lonely?" These results were either put on an index or given on a numerical scale of 1-10 that later translated into a word scale like sometimes or frequently. All of these factors were then put together to find out if a participant was lonely, and the people who were identified as lonely were compared with the people who weren't identified as lonely to compare how many new cases of CVD were reported.

Studies found that people who are lonely or have poor social health are anywhere from 13-42% more likely to develop CVD, two times more likely to die from it [5,6], and 48% more likely to have a hospital admission because of CVD than people who have a good social network [7]. A study from Russia in 2013 found that 57.1% of women in this study had a low amount of close contacts (CCs), and 77.7% were found to have a low social network (SNs) [8]. Women with a low number of close contacts were 4.9 times more likely to get a heart attack and 4.1 times more likely to get a stroke, and a low social network increased heart attack risk by 2.9 times and 2.7 times for stroke [8]. It was also found that women who were married or worked hard physical jobs had a lot less understanding and knowledge about how to keep themselves heart healthy and also CVD in general, and a major amount of women in Russia fell under these categories [8]. One English study found that in a six-year time frame, 571 out of the 5397 people in the study developed a new case of CVD, which was more than 10% of the participants [9], and another English study found that 17% of their participants reported a new CVD case and 16% had a cardiovascular-related hospital visit in the almost 10 year monitoring period of the participants [7].

Table 1: Article Summaries

Authors	**Participants**	**Age**	**Gender**	**Country**	**Results**
Freak-Poli et al., 2021	11486	70+	Female & Male	Australia	People with poor social health were 42% more susceptible to CVD and two times as likely to die from it.

Gafarov et al., 2013	870	25-64	Female	Russia	Women with low CCs and SNs were 4.9 & 2.9 times more likely to get a heart attack and 4.1 & 2.7 times more likely to get a stroke
Golazewski et al., 2022	57825	65-99	Female	United States of America	Women who experienced social isolation and loneliness were found to be 13-27% more susceptible to CVD.
Valtorta et al., 2020	5397	50+	Female & Male	England	571 new cases of CVD were found out of the 5397 participants: more than 10% of the participants.
Bu et al., 2020	10437	50+, mean age of 64	Female & Male	England	17% of participants reported a new case of CVD and 16% had a

					CVD-related hospital visit.

DISCUSSION

Overall, loneliness does increase the risk for CVD by 13-42% [5], and having a low social network increases the risk for CVD by 4.5 times [8]. The cardiovascular diseases most associated with loneliness were stroke and myocardial infarction. While I was not surprised that loneliness impacts CVD, I was very surprised by the magnitude and how much the risk increases. Considering that 1 in 3 people suffer from loneliness in developed countries – 1 in 12 suffering from it severely – how loneliness affects health, in general, should be studied more deeply, especially after seeing how much it increases the risk for CVD. I noticed that compared to other social determinants of health, not much research has been done in the past to examine the impact of loneliness on CVD, even though loneliness is a very prevalent problem.

Additionally, all of these studies were mainly done on older adults, except for Gafarov et al., 2013 which included adults 25-64 years of age [8]. While myocardial infarction and stroke are more common in older adults, everyone can experience loneliness. The stressful teenage years can be very lonely and may have an impact on future CVD outcomes. The role of social media and the sense of social belonging in teenagers in the development of CVD over time may be an area of future research.

Another piece of research that should be looked into is if there's any health difference between a person who's been lonely their whole life and a person who's been lonely only for a short period of time. Most of these studies asked if the participant felt lonely now, but not how long they've felt lonely for. Being lonely for a prolonged period of time rather than a short one and how it affects CVD could be interesting to look into because there would probably be some difference.

Overall, this scoping review provides evidence that loneliness increases the risk for CVD substantially. We can use this information to recognize the significance of loneliness in the health world and dig deeper into the devastating results of this growing feeling.

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Mihir Thakur

Health Literacy as an Effector on Hypertension Levels and Knowledge

ABSTRACT

Background: Hypertension remains an ongoing health issue, contributing to 10 million deaths each year globally. While health literacy is a critical determinant of hypertensive outcomes, current interventions have fallen short in addressing this complex issue comprehensively. This paper aims to explore the role of low health literacy in contributing to hypertensive conditions.

Methods: I searched PubMed to find reputable research papers and studies done with the keywords including "blood pressure control," "hypertension," "health literacy," and "treatment adherence."

Results: Patients with adequate functional health literacy had a mean knowledge score of 16.5± 2.3, while the mean scores for patients with marginal and inadequate literacy were 15.2± 2.2 and 13.2± 3.1, respectively. Apart from literacy, the amount of time of hypertension, years of school completed, and age showed correlations to hypertension knowledge scores in analysis.

Discussion: The demographic distribution revealed a concerning trend, with a large percentage of minorities exhibiting inadequate functional health literacy, further highlighting the prevalence of health literacy disparities. The mostly minority population in the studies, limits the generalizability of findings. Future research should explore these relationships in more general and holistic populations or societies.

INTRODUCTION

Hypertension, characterized by elevated blood pressure levels and recognized as a primary risk factor for cardiovascular diseases (CVD) and other adverse health outcomes, affects approximately 1.13 billion individuals globally, contributing to nearly 10 million deaths annually [2]. While the intricate physiological mechanisms of hypertension have been extensively studied, an emerging area of focus revolves around the influence of health literacy, or lack thereof, in exacerbating this condition. Hypertension leads to

cardiovascular diseases including heart disease, heart failure, and arrhythmia. On top of that, an elevated blood pressure puts individuals at a much higher probability for stroke as the blood vessels are damaged in the brain, increasing hemorrhaging or clot formation. Kidney failure can also be attributed to chronic hypertension as it can damage the kidneys' blood vessels and impair their filtering ability.

The impact of hypertension transcends geographic boundaries, impacting individuals across diverse socio-economic strata, yet the burden disproportionately affects populations with limited health literacy [7]. Health literacy, defined as an individual's capacity to obtain, process, and understand basic health information and services to make appropriate health decisions, is intricately linked to hypertension management. The correlation involved, intensifies the risk of complications, including heart disease, stroke, kidney failure, and premature mortality. In a study of 402 individuals conducted by Williams et al., found that 55%of patients who had hypertension and diabetes had low health literacy and did not understand that a blood pressure reading of 160/100 mmHg was abnormal, along with 60% of patients who did not know that exercise lowers blood pressure [1].

Despite the recognition of health literacy as a crucial determinant of hypertensive outcomes, existing interventions have fallen short in addressing this complex issue comprehensively. Conventional approaches have predominantly focused on medical management and lifestyle modifications, assuming a high level of health literacy among patients. However, the reality often reveals substantial gaps in understanding, navigating healthcare systems, interpreting medical instructions, and adhering to treatment regimens, particularly among individuals with limited health literacy skills.

Furthermore, these gaps perpetuate disparities in health outcomes, contributing to a cycle of inequities among vulnerable populations. The inadequacy of current strategies in accommodating diverse health literacy levels has underscored the urgent need for interventions that bridge the gap between hypertension management and varying levels of health literacy.

This research aims to explore the multifaceted factors of low health literacy that contribute to hypertensive conditions. By exploring the intricate

intersections between health literacy, hypertension management, and health outcomes, this study seeks to uncover the specific challenges faced by individuals with limited health literacy.

METHODS

In this literature review we conducted a search of the relevant articles published from 1998 to 2024, a broad yet encapsulating timeframe of key changes to the scientific contextualization, which can characterize the different social aspects of health literacy and education.

The search strategy was devised to identify relevant literature addressing the intersection of health literacy and hypertension. Electronic databases including PubMed and Google Scholar were searched using key terms including "health literacy," "hypertension," "blood pressure control," and "treatment adherence." Boolean operators (AND, OR) were employed to refine search queries.

Papers were included if they focused on the direct relationships between health literacy and hypertension and focused on the characterization of linkage between health literacy, education, and CVD. Papers were excluded if they were systematic reviews or meta-analyses.

Initially, titles and abstracts of identified articles were screened for relevance to the research topic. Full-text articles meeting the inclusion criteria were subsequently reviewed to assess their suitability for incorporation into the scoping literature review.

RESULTS

Williams et al. did a study of individuals with either hypertension or diabetes that delved into the relationship between the patients' health literacy level, 69% of Spanish speaking patients in Los Angeles, 22% of English speaking patients in Los Angeles, and 57% of patients in Atlanta had inadequate functional health literacy [1]. Merely 42% of individuals with insufficient literacy recognized a blood pressure measurement of 130/80 mmHg as normal, while 45% were unaware that a blood pressure reading of 160/100 mmHg indicates high blood pressure [1]. High proportions of patients with inadequate functional health literacy also lacked knowledge about the effect of lifestyle and dietary factors on blood pressure [1]. Among all patients with

hypertension, mean scores of knowledge (range, 0-21) were strongly related to literacy [1]. Patients with adequate functional health literacy had a mean (±SD) score of 16.5± 2.3, while the mean (±SD) scores for patients with marginal and inadequate literacy were 15.2± 2.2 and 13.2± 3.1, respectively, (P<.001) [1]. Apart from literacy, the duration of hypertension (P<.001), years of schooling completed (P<.01), and age (P<.01) showed notable correlations with hypertension knowledge scores in initial analysis [1]. Upon accounting for these variables, the patient's reading proficiency emerged as the most robust predictor of hypertension knowledge [1]. Despite the strong correlation between inadequate literacy skills and low disease knowledge, there was no found significant relationship between literacy and disease outcomes.

Wang et al. conducted a cross sectional study from a randomized control trial measuring health literacy and self-management efficacy on the health-related quality of life of hypertensive patients in a western rural area of China [4]. The Health-related Quality of Life (HRQL) scores among all patients were evaluated, alongside an examination of the correlation between demographic traits, health literacy, self-management efficacy, and HRQL. The results obtained through the Chew test revealed a notable trend: as age increased, HRQL scores tended to decrease. Specifically, distinct age groups exhibited significant differences in Mental Component Summary (MCS) scores ($p = 0.05$), although no significant variance was observed in Physical Component Summary (PCS) scores [4]. Moreover, discernible differences were evident among various education levels, showcasing an increase in HRQL scores corresponding to higher education levels [4]. Furthermore, PCS scores exhibited significant variance across different levels of hypertension cognition ($p = 0.029$), whereas MCS scores varied significantly among distinct health literacy levels ($p = 0.001$) [4]. Conversely, factors such as gender, annual family income, and yearly family medical expenses did not demonstrate a discernible influence on HRQL [4]. The findings depict that patients who had higher health literacy and self-management efficacy get better HRQL [4]. However, patients who are more elderly with lower education levels havelower HRQL.

A trial conducted by Karami et al. investigated the effect of health literacy intervention based on the medication adherence among uncontrolled hypertensive patients using mobile health [5]. Prior to the intervention, the

health literacy total scores were 33.34 and 33.14 in the intervention and control groups, respectively [5]. Following the intervention, these scores rose to 40.36 and 34.20 in the intervention and control groups, respectively. The increase was statistically significant within the intervention group ($p = 0.01$) [5].

DISCUSSION

Hypertension is the most important modifiable risk factor for all-cause morbidity and mortality worldwide and is associated with an increased risk of cardiovascular disease [6].

The current studies explored the intricate relationship between health literacy levels and the understanding of chronic diseases, particularly hypertension and diabetes, among a predominantly racially minority population. The demographic distribution revealed a concerning trend, with a substantial percentage of Spanish-speaking patients in Los Angeles (69%), English-speaking patients in Los Angeles (22%), and patients in Atlanta (57%) exhibiting inadequate functional health literacy [1]. This highlights the prevalence of health literacy disparities, particularly among minority groups, which can significantly impact the management of chronic conditions.

Consistent with existing literature, our findings underscore a strong correlation between functional health literacy and patients' knowledge of their chronic illnesses [1]. Individuals with inadequate literacy were notably less accurate in responding to knowledge queries, exemplified by their limited understanding of blood pressure readings and the influence of lifestyle and dietary factors. The observed disparities in knowledge scores emphasize the critical role of health literacy in empowering patients with the information necessary for effective self-management.

Despite the robust correlation between inadequate literacy skills and low disease knowledge, intriguingly, no significant relationship emerged between literacy and disease outcomes [1]. This suggests that while health literacy is a pivotal factor in shaping patient knowledge, other elements may influence the translation of that knowledge into tangible health outcomes. Further exploration is warranted to identify these contributing factors and develop

targeted interventions to bridge the gap between health literacy and improved disease outcomes.
The study by Wang et al., conducted in a rural area of China, adds a cross-cultural dimension to our understanding of the impact of health literacy [4]. The findings revealed age-related trends in health-related quality of life (HRQL) scores, with notable differences across education levels. Higher health literacy and self-management efficacy were associated with better HRQL, emphasizing the global relevance of health literacy in influencing patient-reported outcomes [4]. Interestingly, age and education level were significant determinants of HRQL, highlighting the need for tailored interventions based on demographic characteristics.

Karami et al.'s trial focusing on mobile health intervention demonstrated a significant improvement in health literacy scores among uncontrolled hypertensive patients [5]. This underscores the potential of technology-driven interventions to address health literacy gaps and empower patients with the knowledge necessary for effective self-management [5]. The study provides valuable insights into the feasibility and effectiveness of mobile health platforms in enhancing health literacy, thereby contributing to improved medication adherence among hypertensive individuals [5].

While these studies contribute to our understanding of the complex interplay between health literacy, disease knowledge, and outcomes, several limitations must be acknowledged. The predominantly minority population in our study limits the generalizability of findings. Future research should explore these relationships in more diverse populations. Additionally, the focus on short-term outcomes in the mobile health intervention study calls for longitudinal studies to assess the sustainability of health literacy improvements over time.

In conclusion, our study, complemented by findings from diverse settings, reaffirms the pivotal role of health literacy in shaping patient knowledge and influencing health-related outcomes. Addressing health literacy disparities is crucial for empowering patients with the information necessary for effective self-management. Future interventions should consider demographic variations, cultural nuances, and the potential of technology-driven approaches to bridge the health literacy gap and enhance the overall well-being of individuals with chronic conditions.

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Peter Truong

Medicaid on Cardiovascular Health

ABSTRACT

Background: In America, heart disease has become one of the most prevalent conditions, ensuring that almost half of all Americans have at least one risk factor. However, given the rising costs in the healthcare industry, fewer people can afford private health insurance. However, a governmental program, Medicaid, which provides health insurance to people with unstable financial situations, was expanded through the 2010 Affordable Care Act.
Methods: Using PubMed and Google Scholar, articles about Medicaid expansion's effects on cardiovascular health were selected from the key search words of Medicaid, Cardiovascular disease, CVD, and Affordable Care Act. This study excluded articles that were older than 10 years to ensure relevancy.
Results: There was a decrease in the number of uninsured patients with acute myocardial infarction and an increase in vascular-related surgeries in expansion states compared to non-expansion states. Racial disparities in terms of access to treatment dropped in expansion states. In states that have expanded Medicaid, they have seen more increases in insured treatment procedures and hospitalizations that are equitable. In non-expansion areas that saw similar albeit smaller results.
Discussion: If Medicaid is expanded to all states, we could see comparable results in non-expansion states. This study, however, cannot answer how Medicaid can be expanded in a fashion that will gain universal political support. However, further avenues of research can find a way to implement Medicaid in a way that will garner support or find an alternative to achieving more equitable access to healthcare insurance.

INTRODUCTION

According to the Center of Disease Control and Prevention (CDC), 47% of all Americans have at least one risk factor for heart disease [1]. Typically, people have a higher risk of heart disease if they have unhealthy cholesterol levels, high blood pressure, diabetes mellitus, or obesity. One type of heart disease is coronary artery disease (CAD), a condition that affects the flow of blood to the heart. Common symptoms of CAD include arrhythmia, heart failure, and myocardial infarction. In fact, more than 365,000 people have

an out-of-hospital cardiac arrest in the United States each year, and 60-80% die before they reach the hospital [2].

The Affordable Care Act (ACA), a reformative healthcare law passed in March 2010, expanded access to Medicaid, a governmental program that provides health insurance to adults and children with limited resources and income. In states that expanded Medicaid, access was expanded to preventive screening, services, and treatments. According to the American Heart Association (AHA), 64% of studies on tracking insurance of cardiac treatments done on a meta-analysis of 30 studies found an increase in insured cardiac treatments in states with Medicaid [3]. Despite this, there are still states that have not yet expanded Medicaid, so doing so would grant access to more people to prevent and alleviate the effects of heart disease.

The objective of this scoping review is to examine the difference in cardiovascular outcomes across states with and without expanded Medicaid in the United States. If increasing access to healthcare by expanding Medicaid improves cardiovascular health outcomes it could inform policymakers in the future to ensure equitable access to cardiovascular health care across all demographics.

METHODS

In this literature review, Google Scholar and PubMed were searched with key search terms including Medicaid, cardiovascular disease, CVD, and Affordable Care Act. In addition, studies were chosen only from the past 10 years (from 2013 to 2023) to ensure the relevancy of these papers for the scoping review. Articles that compared areas of Medicaid expansion and non-expansion to cardiovascular-related treatments, access to treatments, and fatalities were selected in order to measure the effects of Medicaid expansion to use them as a predictor of expansion in every state. Systematic reviews and meta-analyses were excluded.

RESULTS

Medicaid on Cardiovascular Mortality

A paper by Khatana et al. used a longitudinal, observational design, using a difference-in-differences approach with county-level data from counties in 48 states (excluding Massachusetts and Wisconsin) and Washington, DC, from 2010 to 2016 found that counties in expansion states had 146.4 to 146.5

cardiovascular deaths per 100,000 people compared to counties in non-expansion states, which had 176.3 to 180.9 cardiovascular deaths per 100,000 people [4]. Furthermore, adjusting for demographic, clinical, and economic differences, counties in states that had expanded Medicaid had 4.3 fewer deaths out of 100,000 people than counties in states that have not [4].

Medicaid on Access to Insured Treatment

Wadhera et al. used a retrospective cohort study at hospitals participating in the National Cardiovascular Data Registry Acute Coronary Treatment and Intervention Outcomes Network Registry from patients who were hospitalized with acute myocardial infarction (AMI) [5]. They found that the chances of defect-free low-income adults in expansion states increased by 76.3% to 76.5%, but only increased in non-expansion states by 76.2% to 74.5% [5]. In addition, uninsured hospitalizations of AMI patients declined from 18% (4395 out of 24358 hospitalizations) to 8.4% (2638 out of 31382 hospitalizations) in expansion states compared to 25.6% (7963 out of 31137 hospitalizations) to 21.1% (8668 out of 41120 hospitalizations) [5].

A paper by Glance et al. used a retrospective analysis study to analyze data from hospitals participating in the University Health Systems Consortium, now renamed the Vizient Clinical Database from 2010 to 2018 [6]. Changes were analyzed between white and non-Hispanic black revascularization therapy patients hospitalized with ST-segment elevation (STEMI) and non-ST-segment elevation acute myocardial infarction (NSTEMI). The study found that for STEMI patients, differences between white and non-Hispanic black revascularization rates decreased by 2.9% in expansion states versus non-expansion states when the data was adjusted for patient and hospital differences [6]. In addition, the researchers concluded that the expansion of Medicaid had resulted in a decrease in the number of uninsured black patients to uninsured white patients [6].

A paper by Eguila et al. used the Healthcare Cost and Utilization Project State Inpatient Database to find patients undergoing care for major vascular pathology from 2010 to 2014 of patients aged 18 to 64 [7]. Inpatient admissions for insured patients in expansion states with an abdominal or thoracic aneurysm and carotid stenosis diagnosis grew greatly compared with non-expansion states. There were also more vascular-related surgeries

for carotid endarterectomy, lower extremity revascularization, lower extremity amputation, and arteriovenous fistula in expansion states than in non-expansion states [7].

A paper by Eslami et al. used data that was gathered from patients who had infrainguinal bypass procedures performed due to occlusive pathology from 2010 to 2017 [8]. Interrupted time-series analyses were used to analyze their primary outcomes from 1-year follow-ups. The researchers found that among non-acute cases, elective procedures increased in Medicaid expansion states by 3.9% with a decrease in annual mortality rates by 0.4% [8]. According to the researchers, the results were statistically significant after comparing them to annual trends of states that did not expand Medicaid [8].

DISCUSSION

Based on the evidence from the studies, the data suggests that there is generally a decrease in cardiovascular mortality rates and an increase in treatment for cardiovascular disease in states that have expanded Medicaid [4]. In addition, Medicaid expansion also appears to decrease disparities between demographics in terms of health insurance access [6]. This suggests that there could be a similar effect if Medicaid is expanded to other states.

Overall, one clear pattern throughout the data is shown: expanding Medicaid results makes access to cardiovascular treatment more equitable. Initial expectations were that Medicaid expansion would result in higher numbers of insured treatments for cardiovascular disease patients. The results mostly met my expectations that access to health insurance would help address concerns about cardiovascular mortality. However, one unexpected result was that there was a decrease in the number of uninsured AMI patients in states that did not have Medicaid expansion [5]. One explanation for this could be that people living in a coverage gap in non-expansion states could have moved to expansion states to get health insurance. This would result in a higher percentage of insured patients, but would not be an accurate assessment of that particular state’s access to medical insurance. Another explanation could be that as more people had access to free healthcare in expansion states, private insurers lowered their prices in response in both expansion and non-expansion states. This would result in a higher level of access to health insurance in non-expansion states.

These results are significant because states that have not expanded Medicaid yet could see similar results if Medicaid was expanded, building on existing evidence that it allows more people to access lifesaving treatments. In addition, it ensures that there is also more equitable access to healthcare that does not discriminate against certain demographics. Due to the benefits of increased access to treatments, Medicaid should be expanded in all states to minimize cardiovascular deaths that can be prevented.

This study can conclude that Medicaid expansion's positive effects on insured access to cardiovascular treatment and potentially cardiovascular mortality can be replicated in states that have not expanded it. However, it is beyond the scope of this study to find a solution to expanding Medicaid across all states because that is a political issue. It cannot determine whether Medicaid should be expanded because other factors need to be considered. Political factors, economic implications, ethical considerations regarding social justice, and existing legal frameworks must also be considered to determine further whether or not to expand Medicaid in all states. However, this study effectively answers that Medicaid expansion will result in greater and more equitable access to insured treatment, leading to increased odds of surviving severe symptoms of heart disease.

Further research is required to see if Medicaid expansion can have a positive economic effect through ensuring longer life spans. Another avenue of research would be to see if perhaps another solution exists in broadening access to health care insurance by working with the private sector. Alternatively, another area of potential research could be investigating how to lower costs in the healthcare industry. However, with the current state of the healthcare system, expanding Medicaid in all states would be the best avenue for ensuring an increase in access to cardiovascular treatment.

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Pranavi Vadi

Determining Whether Diet And Exercise Is More Advantageous Than Insulin Pumps In Managing Type 2 Diabetes

ABSTRACT

Type 2 Diabetes (T2DM), caused by the body's inability to respond properly to insulin produced by the pancreas affects 35 million Americans. The purpose of this study is to determine how beneficial diet and exercise is compared to insulin pumps in diabetics. I searched Google Scholar using terms such as type 2 diabetes, insulin pumps, diet, and exercise. During high intensity exercise irisin levels increased 88.4%, betatrophin levels rose to 667.3%, and insulin levels rose to 100.2%. Those who adapted an intermittent fasting diet achieved a mean weight reduction of 5.93 kg and 47.2% of the individuals achieved remission. Insulin pumps are used in type 2 diabetics if the patient needs higher doses of insulin, but it has been proven to lower A1C levels by using less insulin than other devices. Further research should be done to test if insulin pumps are as effective in those who don't need a high dose of insulin.

INTRODUCTION

Type 2 diabetes (T2DM) affects nearly 35 million Americans [1]. It can be found in adults, but it is also becoming increasingly prevalent in children and teens. It is caused by the body's inability to respond properly to insulin produced by the pancreas, or the body does not produce enough insulin. Insulin is a hormone produced by beta cells in the pancreas that acts as a signal for cells to take in glucose, a lack of response leads to high blood sugar in those affected by diabetes. For type two diabetics, managing their condition to remission, or when diabetes is not present anymore, is ideal [2]. Diabetes affects many organs in our body: the heart, blood vessels, the eyes, and the kidney. Recently, diabetes has been linked to various cardiovascular diseases, such as congestive heart failure or a stroke. Cardiac problems cause 80% of deaths in diabetic patients. Recent studies have shown a need to control glucose management and cardiovascular disease risk factors in diabetic patients [3].

There is no cure for diabetes; patients often focus on lifestyle changes instead. Although patients can achieve remission, in which A1C levels are

very low there is no guarantee remission will be permanent. The Diabetes Prevention Program detailed that changes in lifestyle can prevent type 2 diabetes by 58% over 3 years [3]. Additionally, there are medical devices commonly used to manage glucose levels and insulin administration. There are many devices available. Some include insulin pumps, multiple daily injections (MDI), insulin pens, and implantable glucose monitors. Insulin pumps offer results over a long period of time and are often more affordable than other devices available. Furthermore, devices must manage nocturnal hypoglycemia, when blood sugar levels become low while a person is sleeping [4]. Devices are able to do this by constantly monitoring glucose levels and releasing small amounts of insulin when necessary.

The usual diet advised to patients is a balanced diet with calorie restriction [2]. Furthermore, research has been conducted on the various dietary options available, including low fat/low carbohydrate diets, mediterranean diets, and Chinese medicine dietary therapy (TCMDT). Consisting of whole grains and traditional Chinese medicines, TCMDT is often used to battle hyperglycemia and reduce obesity in patients [5]. Nonetheless, currently there is not enough data on which diet is most effective after four years. In a study comparing a diet or fitness change to a group that made no change, 46% of patients achieved remission in the tested group compared to the 4% that made no change to their normal routine [6]. Not many reviews have shown the true effect of either a medical device and diet on managing diabetes.

The purpose of this study is to determine how beneficial diet and exercise is compared to insulin pumps in diabetics. In these trials, patients were used to study the effect of insulin pumps and diet or exercise. By analyzing the data already gathered we can understand which factor is better at controlling diabetes. Positive results are indicated by a decrease in A1C levels or weight loss over a given amount of time.

METHODS

I used Google Scholar to find relevant sources by using the search terms: Type-2 diabetes, insulin pumps, diet, and exercise. Other inclusion criteria were articles written in English y in the last five years. Exclusion criteria included papers about Type-1 diabetes, other diabetic devices, like insulin

injections or glucose monitors, and any studies on pregnant individuals or children.

RESULTS

In a trial that determined the effect of intermittent fasting on Type-2 diabetes remission, those who practiced intermittent fasting for three months, 47.2% achieved remission and achieved a mean weight reduction of 5.93 kilograms. In the control group, 2.8% achieved remission and had a mean weight reduction of 0.27 kilograms [6]. Yang L, Lin H et al. demonstrated that exercise helps insulin resistance in Type-2 diabetics. Exercise also leads to a decrease in blood sugar and an increase in glucose uptake, by increasing glucose oxidation. Additionally, a decline in branched amino acids was observed, which promotes glucose and lipid metabolism [6]. It was concluded that a high fat diet leads to an increase in firmicutes which break down carbohydrates. Daly and Hovorka detail the various types of technology available to those with Type-2 diabetes (see figure 1); they concluded that there is no significant benefit of self monitoring glucose levels longer than a year, since there was no difference compared to the control which used normal basal insulin once a day [7].

Enteshary et al. conducted studies on the levels of irisin, betatrophin, and insulin during exercise. They concluded combined aerobic training led to increased irisin levels by 88.4% in the high intensity group and 36.7% in the moderate group. Betatrophin levels rose 667.3% in the high intensity group. In the moderate group it rose by 1.6%. Insulin levels rose to 100.2% in the high intensity group. In the moderate group it rose 56.7%. Participating in high intensity training increases insulin levels in diabetic patients by 100.2% [8]. Since the various hormones released during exercise facilitate the release of insulin, blood sugar reduces in the body [8]. A narrative review describes the various dietary strategies beneficial for type 2 diabetics, concluding that low carbohydrate diets were effective in reducing weight to achieve remission. But patients were unable to continue the diet due to the low requirement of carb needed [9].

Brown et al. 14.7% of the participants achieved remission by taking part in the Mediterranean diet [9]. In Grunberger et al. they compared the benefits of Insulin pumps against MDI. The study observed that the mean difference in A1C in CSll was -1.27% as opposed to the 0.85% from MDI. Fasting

plasma glucose in Continuous subcutaneous insulin infusion (CSII) was -33.9mg/dL. In MDI the difference was -35.6 mg/dL. It was concluded that both treatments are effective in reducing A1C. But, the CSII group achieved a greater reduction with much less insulin compared to the MDI group [4]. A clinical overview of insulin pumps detailed that many studies have shown that A1C has decreased by 1% or more when individuals use insulin pumps [10]. Ruiyu, Wu et al. used the TCMDT diet and studied how it affected gut bacteria, concluding that it increased diabetes-improving bacteria (Coriobacteriaceae, Bacteroidaceae, and Peptostreptococcaceae) by 7-26 times [5].

The difference in fasting blood glucose between those dieting and using an insulin pump is also significant. Those who adopted an intermittent fasting diet achieved a reduction in blood glucose of 1.82 mmol/L [2], while those who utilized an insulin pump for another study achieved a reduction of 1.9 mmol/L [4]. Furthermore, a reduction in mean A1C was observed in the diet group of 1.75% [2], while in the CSII group there was a 1.27% difference in mean A1C [4].

Table 1: Article Summaries

Article	Purpose	Independent Variable	Dependent Variable
Effect of an Intermittent Calorie-restricted Diet on Type 2 Diabetes Remission: A Randomized Controlled Trial	Determine the effect of an intermittent fasting diet on type 2 diabetes remission.	Intermittent Fasting Control	Percent of those who achieve remission A1C levels Fasting blood glucose level
Exercise Ameliorates Insulin Resistance of Type 2 Diabetes through	To understand how exercise aids in insulin resistance in type 2 diabetics.	High fat diet Control	Fasting blood glucose levels Intestinal flora levels Glucose uptake capacity

Motivating Short-Chain Fatty Acid-Mediated Skeletal Muscle Cell Autophagy			
Comparison of the Effects of Two Different Intensities of Combined Training on Irisin, Betatrophin, and Insulin Levels in Women with Type 2 Diabetes.	Understand how different intensities of exercise can affect hormone levels beneficial in diabetics.	High-intensity training Moderate-intensity training Control	Irisin levels Betatrophin levels Insulin levels
Human regular U-500 insulin via continuous subcutaneous insulin infusion versus multiple daily injections in adults with type 2 diabetes: The VIVID study	To determine if CSII is more beneficial than MDI.	Continuous subcutaneous insulin infusion (CSII) MDI	A1C levels Fasting plasma glucose Weight change Hypoglycemia
Effect of a Chinese medical nutrition therapy diet on gut microbiota and short chain fatty acids in the	To study the effect of the TCMDT diet on gut microbiota.	TCMDT diet Control	Composition of gut micro bacteria Microbiota activity

simulator of the human intestinal microbial ecosystem (SHIME)			

DISCUSSION

Type-2 diabetes stems from insulin resistance in the body which is when the body does not react to insulin produced by the pancreas [1]. Studies have shown that exercise helps with blood sugar levels. As exercise is performed, hormones are produced such as irisin and betatrophin. Irisin increases the expression of betatrophin, which increases beta cell growth [8]. This increases insulin production, as its source in the body increases. Additionally, insulin levels also increase with exercise [1]. The higher the intensity of exercise the higher the hormone levels are. Exercise promotes the decrease of blood branched-chain amino acids, which are substrates that produce energy. Therefore, promoting lipid and glucose metabolism in patients [6]. Exercise regulates the concentration of short fatty acid chains (SCFA) in diabetic individuals. When the whole body vibrates it increases the amount of SCFA producing bacteria [6]. Bacteria found in the gut (intestinal flora) can regulate the secretion of hormones of endocrine cells, which regulates appetite and the release of insulin. Exercise can change the composition of intestinal flora, the barrier of the gut, and can lead to an increase in Bacteroides which can battle obesity and help insulin resistance [6].

A decrease in A1C was observed as well as an increase in remission rate when diabetic individuals adopted an intermittent fasting diet. [2]. To clarify, remission only occurred in those who had lower A1C levels and were on fewer antidiabetic medications. But it was found that many did achieve remission despite having the disease for a minimum of 6 years [2]. Intermittent fasting promotes the production of beta cells which helps control the body’s metabolism and the production of insulin [5]. With the introduction of intermittent fasting in their diets, patients greatly decreased their medicine costs by using less antidiabetic drugs. Furthermore, research has found that a high fiber diet is found to promote a group of SCFA’s that focus on easing the effects of diabetes [2].

Insulin pumps have been a trusted mode of insulin delivery for many years by efficiently administering insulin. Specifically, in Type-2 diabetes it is only used in those who need higher doses of insulin. In a study comparing insulin pumps to MDI, both devices lead to a lower A1C [4]. However, the group tested with insulin pumps achieved this lower level by using less insulin. The insulin pumps vastly increased insulin release in the morning to modify the amount of insulin released at night. Additionally, both groups experienced similar amounts of weight gain [4]. Moreover, the occurrence of nocturnal hypoglycemia actively decreased as the study continued in the insulin pump group. Therefore, as the device discovered the trend it adapted the amount of insulin released during the night to avoid hypoglycemia. In many occurrences insulin pumps have been proven to be the best device for managing diabetes, since they are much more practical, easy to use, and cheaper than other devices [7].

After reviewing my findings, I have found that daily aerobic exercise aids in reducing insulin resistance and intermittent fasting reduces A1C levels in order to achieve remission in Type-2 diabetics. As opposed to using an insulin pump to control blood sugar, changes in exercise and diet have shown a great amount of promise to control type 2 diabetes leading to weight loss and a decrease in A1C levels. Insulin pump therapy did show great promise with decreasing total A1C and managing nocturnal hypoglycemia, but it was not as beneficial as diet and exercise. Often, insulin therapy is used in type 2 diabetics with those who require high amounts of insulin. This has become a novel approach as there are no other practices [6].

This research can be utilized to modify insulin pump technology in the future. As my research has shown that lifestyle changes are more beneficial. After analyzing the positive effects of lifestyle changes, insulin pumps do not seem to be the best choice for those with type 2 diabetes. Furthermore, these results highlight how vital diet and exercise is to treating diabetes. Future research on the use of insulin pumps in individuals that do not need high doses of insulin is vital to see if insulin pumps can be advantageous to everyone with Type-2 diabetes.

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Health Disparities within Racial Minorities effect on Cardiovascular Health

ABSTRACT

Background: Heart diseases in racial minorities differ as social determinants affect every community differently. Specifically, in minorities such as American Indians, Alaskan Natives, and Hispanics, cardiovascular disease (CVD) is more prevalent due to underrepresentation, underreporting, and health factors, where the risk of CVDs is higher. This literature review will compile information from other studies to address the difference between low income racial minorities.

Methods: Using search engines, Google Scholar and PubMed, this review was based on studies that reported on racial minorities living in low income communities, with a lack of access to affordable, quality healthcare, lack of a stable socioeconomic status, and other social determinants.

Results: Racial minorities in low-income communities are the leading factor in heart related deaths in the US. About 58% of the lower class are composed of racial minorities, where 67% of hispanic women were affected by ischemic heart disease. CVD and other heart diseases such as coronary heart diseases are 12% more prevalent in American Indians and Alaskan Native, compared to the white population. Native Americans and Alaskan Natives are also found to be underreported in CVDs by 21% because of lack of representation within the healthcare community.

Discussion: Much of the higher rates of CVD in racial minorities were due to the increase of unhealthy behavior, because of the lack of health education within these communities. In American Indians and Alaskan Native, the numbers may not even be accurate due to the underrepresentation and underreporting, which is why not many programs are in place to fight against these higher rates. Hispanics in low income communities often dismiss health warnings, because of cultural “norms” and continue on with unhealthy lifestyles with excess drinking, smoking, and dietary habits.

INTRODUCTION

Heart disease is the leading cause of death in the United States, and largely differs by race and ethnicity [1]. Non-hispanic black persons were more than twice as likely to die of heart diseases in 1999 and 2017 [1], because of dietary factors, exposure to chemicals, and higher risk of hypertension, within these communities compared to other racial groups. Among American Indians, and Alaska Natives, higher rates of heart failure and arrhythmia and other forms of CVD are more prevalent, but lack proper defense due to limited data [2] and underrepresentation in the health care system.

These racial minorities are often underrepresented within the system, and are exposed to a higher risk of CVD, because of lack of education, lack of quality health care/insurance, and other social determinants of health [2]. Racial minorities in low income households aren't often included in national surveys, and because of different lifestyle habits, have a higher risk of cardiovascular diseases [2]. About 58% of low income communities are those of racial minorities, often living lifestyles that contribute to CVD risks [5]. Hispanic and non-Hispanic black adults, age 20 and over were the most likely to have hypertension, obesity, diabetes, and high total cholesterol from 2015-2016 [1], which may not be an accurate measure in numbers due to the lack of representation within these communities.

There are many factors when determining what creates higher risks in CVD for these communities. This review will address what creates racial differences between American Indians, Alaskan Natives, African Americans, Hispanics and their white counterparts and how the healthcare system fails to cater, and causes an increase of risk for CVD in these groups. This paper will also address the programs put in place to try to fix the numbers of CVD in these minorities, and incentives placed in low income areas to lower the risk of CVD. This paper will aim to promote health equity, and analyze the lifestyles of these minorities, to find what leads to this higher mortality rate when considering cardiovascular health.

METHODS

In this literature review, using studies from 1991-2023, Google Scholar, and PubMed were used to identify studies that reported on the health disparities within specific racial and ethnic groups. Using key search terms, "access to healthcare relationship with cardiovascular health," articles were chosen if

they included the racial/ethnic groups in low income with high risk to particular heart conditions or CVD. After thoroughly scanning abstracts, and full articles, the list of articles were narrowed down to original studies on how racial disparities in low income communities affect access to health care, and its relationship with cardiovascular health.

RESULTS

This literature review found that largely low income racial minorities were the leading group in heart related deaths in the United States. Among women in 1995, the leading group in heart related deaths were Hispanic women of whom 67% had ischemic heart disease [3]. CVD and coronary heart disease (CHD) rates are 12% higher likely in American Indians and Alaskan Natives compared to the white population in the United States, and are even believed to be underreported by 21% [2]. It was found that diabetes mellitus (DM), and higher rates of coronary heart disease among these two groups, with additional factors such as low-density lipoprotein cholesterol levels (one of the lipid groups), hypertension, renal diseases, age and sex were the leading CVDs in American Indians and Alaskan Natives [2].

Among Hispanics, in a study from 2015, participants were relatively healthy with only a third reporting having ever been told they had any type of heart disease, heart failure, high cholesterol, or diabetes/hyperglycemia [4]. Over a quarter of the sample had been told that they had high blood pressure [4]. Participants in the sample were mostly under the age of 50 years, women, had a high school education or less, were foreign-born, and spoke both English and Spanish at home. Most participants were currently insured, had a usual source of care, and had more than one physician visit in the last 12 months [4]. Hispanics, the largest ethnic minority group in the United States, have higher rates of CVD behavioral risk factors such as smoking and lack of physical activity than non-Hispanic whites [4]. In another study from 2022, 70.4% of the participants had treated hypertension, who had also graduated high school and had a college degree [5]. As the level of education completed went lower, the cases of untreated hypertension increased within the group [5].

DISCUSSION

The leading factor to the increase of CVDs in American Indians and Alaskan Natives is mainly exposure to toxic material due to smoking and those living

in low income communities. Some programs and resources have reduced CVD risk in higher risk communities, but systemic issues such as the underreporting of American Indian, and Alaska Native population largely underestimates the “extent of CVD” within these groups. Forms of CVD, such as heart failure and arrhythmia are also understudied within American Indian and Alaskan Indian populations, and social determinants vastly contribute to both the lack of access to quality care, and the underrepresentation of these groups within the healthcare system [2]. The data suggests that due to this underrepresentation, these population groups are more susceptible to untreated CVDs, and dismiss early stages of hypertension and other symptoms that are indicators of CVDs.

Increasing rates of ischemic heart disease among Hispanic women is largely due to the unhealthy and cultural “norms” that are common within the community. Compared to results in 1995, in 2022 CVDs affect 42.7% of Hispanic women, and 52.7% of Hispanic men [6]. Due to social and economic challenges, CVD remains the leading cause of death within Hispanics, because of the significance of morbidity and mortality in this group [6]. Compared to their white counterparts, with only 36% of the population affected by CVDs, Hispanics are more susceptible to heart diseases due to core health behaviors like smoking, physical inactivity, diet, and weight [6]. This also includes health factors, such as cholesterol, blood pressure, and glucose control which levels vary among races [6]. The results might suggest that it is due to the high lipid diets that are often present in these communities. However, a more plausible explanation is a lack of physical activity, and excess smoking and other unhealthy behaviors. The results build on existing evidence of the difference between racial groups in general. Those in low income Hispanic and black communities often have less healthier lifestyles, which increases the risk for CVDs [5]. This is why initiative should be taken to educate these communities on the importance of cardiovascular health and the importance of studying the implications within these racial minorities.

Future research is needed to establish proper procedures to help these low income communities. Often because of the underrepresentation and underreporting of these groups, few options are there to maintain and support individuals of these communities. It is beyond the scope of this

study to specifically dive into areas of the US, but this review addresses the clear differences between racial minorities.

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