THE IMPORTANCE OF HAND HYGIENE IN THE ERA OF ANTIMICROBIAL STEWARDSHIP

Muhammad Faheem Afzal[1]
1. Professor of Pediatrics: Program Director, Pediatric Infectious Diseases, King Edward Medical University, Lahore

Article Info

EDITORIAL

Corresponding author:
Muhammad Faheem Afzal: Professor of Pediatrics: Program Director, Pediatric Infectious Diseases, King Edward Medical University, Lahore
Email: mfaheem169@gmail.com

Vol: 3 | Issue:1

ISSN Print: 2960-2580
ISSN Online: 2960-2599

Publisher:
Medical Research and Statistical Consultancy Training Centre (SMC-PRIVATE) Limited

Keywords:
Hand Hygiene, Antimicrobial Stewardship, Pediatrics, Infectious disease

In the current healthcare landscape, the increasing prevalence of antimicrobial resistance (AMR) has brought into sharp focus the need for more stringent antimicrobial stewardship plans. These agendas aim to improve the use of antibiotics and few other antimicrobial agents to preserve their effectiveness. However, one essential yet often overlooked component of infection control - hand hygiene - plays a pivotal role in the success of these stewardship initiatives.[1]

Hand hygiene, the process of cleaning hands using either soap and water or alcohol-based hand sanitizers, has long been recognized as one of the most effective methods for preventing the spread of infections in healthcare settings. This practice is not just a foundational element of infection control, but also a critical strategy in reducing the transmission of multi-drug-resistant organisms (MDROs) that contribute to the growing AMR crisis.[2]

Enhancing hand hygiene compliance is not just about preventing infections—it is also a critical strategy for antimicrobial stewardship, as it helps reduce the need for antibiotic interventions in the first place. In the era of antimicrobial stewardship, the role of hand hygiene becomes even more significant. The improper use or overuse of antibiotics, combined with lapses in infection control practices like hand hygiene, can accelerate the emergence of resistant strains of bacteria.[3] This gap in practice underscores the necessity for constant education, training, and monitoring to ensure that hand hygiene is consistently practiced as part of daily routines.

The importance of hand hygiene cannot be overstated in the fight against antimicrobial resistance. By improving hand hygiene practices, we can reduce the spread of infectious diseases, limit the use of unnecessary antibiotics, and slow the emergence of antimicrobial resistance.

REFERENCES

1. Gammon J, Hunt J. The neglected element of hand hygiene - significance of hand drying, efficiency of different methods and clinical implication: A review. J Infect Prev. 2019;20(2):66-74.
2. Kapil R, Bhavsar HK, Madan M. Hand hygiene in reducing transient flora on the hands of healthcare workers: an educational intervention. Indian J Med Microbiol. 2015;33(1):125-8.
3. Mathur P. Hand hygiene: back to the basics of infection control. Indian J Med Res. 2011;134(5):611-20.

LIFE STYLE MEDICINE: AN EMERGING HEALTHIER FUTURE

Rabiah Mahwish[1]

1. Associate Professor: Community Medicine department, Khawaja Muhammad Safdar Medical College Sialkot

Article Info

GUEST EDITORIAL

Corresponding author:
Rabiah Mahwish: Associate Professor: Community Medicine department, Khawaja Muhammad Safdar Medical College Sialkot
Email: dr.rabiah5@gmail.com

Vol: 3 | Issue:1

ISSN Print: 2960-2580
ISSN Online: 2960-2599

Publisher:
Medical Research and Statistical Consultancy Training Centre (SMC-PRIVATE) Limited

Keywords:
Life style, Digital health, Emerging, sedentary life style

Burden of deaths due to non-communicable diseases is increasing worldwide. Low- and middle-income countries share 70-80% of this burden. WHO Pakistan country profile shows that approximately 80 million population is suffering from one or more noncommunicable diseases. About 25.3% population has hypertension, 19% cardiovascular diseases, 17% diabetes mellitus while 8% cancers and about 6% suffer from chronic respiratory diseases.[1]

Traditional medicines mainly deal with symptoms of diseases by medications and treatment procedures but do not address the underlying causes of chronic diseases at primordial and primary level of health care. Life style medicine bridges this gap by intervening in daily life habits and behaviors. Life style medicine is an evidence-based approach that works mainly in 6 domains of life style.

These domains include nutrition, physical activity, stress management, avoidance of harmful substances, positive socialization and sleep hygiene. Interventions in life style of these domains help in preventing underlying causes of non-communicable diseases. American College of Lifestyle Medicine recommends the following practices in lifestyle:

1. A regular physical activity for at least 30 minutes per day for five days in a week or 150 minutes per week is considered a healthy lifestyle. Regular exercise decreases triglycerides and low-density lipoprotein and increases high density lipoprotein which regulates lipid profile and helps in decreasing blood pressure as well as glucose levels. It also reduces weight gain.
2. Practicing a peaceful sleep of 9–10 hours per day is recommended. Any kind of sleep deprivation may increase the risk of hypertension, diabetes and cardiovascular diseases.
3. Avoiding alcohol, smoking and substance abuse.

4. Adopting nutritional modifications for a healthy balanced diet comprising of 45-65% carbohydrates, 25-35% fat and10-30% protein of total caloric requirements. Avoiding high caloric foods, soft beverages and junk food promotes a healthy lifestyle.
5. Taking measures for stress management and participating in healthy activities. Nature such as forests, gardens, mountains and seas can not only improve psychological health but also physical health of individuals.
6. Building a healthy relationship with family and friends. A healthy social relationship improves mental health and enhance self-esteem.[2]

Recently our world has faced COVID-19 pandemic which left many challenges to maintain a healthy lifestyle and promote a good quality of life. Evidence showed that high prevalence of morbidity and mortality was associated with unhealthy lifestyle practices. The main pillars of lifestyle medicine supported the healthcare workers and patients to combat during this pandemic and even after this to lead a healthy life.[3] Health care providers can enhance the effectiveness of their treatment by incorporating comprehensive treatment along with life style medicines. This step will not only reduce the burden of pharmacological medicines but also increase the quality of life. Individuals can take informed choices to avoid unhealthy behaviors and combat modifiable risk factors of chronic diseases.[4] Although evidence based new research is required to develop guidelines for lifestyle medicine, but following recommendations are necessary to adopt healthy behavior by individuals:

Firstly, health education and Awareness: Health education programs, awareness campaigns and outreach community health programs can sensitize individuals about unhealthy behaviors. Individuals can make healthier lifestyle choices.

Secondly, healthcare multidisciplinary Integration: Train health care providers to incorporate lifestyle medicine with comprehensive treatment. This incorporation of lifestyle medicine will help to reduce the modifiable risk factors of non-communicable diseases.

Thirdly, healthy environmental policy: Adopt healthy environmental policy to promote lifestyle medicine by providing good space in residential areas for physical activity, easy and cheap availability of healthy food, no access to harmful substances and providing healthy working environment.

Lastly, Social support systems: Provision of social support programs through community participation can provide a platform to individuals for maintaining healthy lifestyles.[5,6] Life style medicine is a paradigm shift in the field of healthcare, providing prevention and improving quality of life and well-being. Through sustainable lifestyle modifications, we can address the modifiable risk

factors of the non-communicable diseases and reduce the burden of our tertiary health care facilities in terms of cost and patient turn over. So, it is imperative for healthcare providers, individuals and policy makers to adopt lifestyle medicine as a fundamental component of their health and wellbeing. As we are moving forward, combined efforts can carve the way to a better, healthier and more well-suited lifestyle.

REFERENCES

1. Naeem Z. Pakistan–double burden of diseases. Journal of Shifa Tameer-e-Millat University. 2019 Jul 21;2(1):1-2.
2. John NA, John J, Tarnikanti M, Kalpana M, Kamble P, Singhal A, Ganji V, Gaur A, Umesh M, Katta R, Saktivadivel V. Implications of lifestyle medicine in medical practice. Journal of Family Medicine and Primary Care. 2023 Feb 1;12(2):208-12.
3. Smirmaul BP, Chamon RF, de Moraes FM, Rozin G, Moreira AS, de Almeida R, Guimarães ST. Lifestyle medicine during (and after) the COVID-19 pandemic. American Journal of Lifestyle Medicine. 2021 Jan;15(1):60-7.
4. Farrokhi M, Taheri F, Bayat Z, Damiri M, Farrokhi M, Ghadirzadeh E, Tosi YE, Dahouei AA, Kazemi MH, Lahijan LZ, Esfahani A. Role of Lifestyle Medicine in the Prevention and Treatment of Diseases. Kindle. 2024 Mar 15;4(1):1-219.
5. Vodovotz Y, Barnard N, Hu FB, Jakicic J, Lianov L, Loveland D, Buysse D, Szigethy E, Finkel T, Sowa G, Verschure P. Prioritized research for the prevention, treatment, and reversal of chronic disease: recommendations from the lifestyle medicine research summit. Frontiers in medicine. 2020 Dec 22;7:585744.
6. Lippman D, Stump M, Veazey E, Guimarães ST, Rosenfeld R, Kelly JH, Ornish D, Katz DL. Foundations of Lifestyle Medicine and its Evolution. Mayo Clinic Proceedings: Innovations, Quality & Outcomes. 2024 Feb 1;8(1):97-111.

FREQUENCY OF PLANTAR FASCIITIS AMONG PHYSIOTHERAPISTS IN LAHORE

Hira Zahid[1], Iqra Waseem[2], Sana Akram[3]
1. University institute of physical therapy, university of Lahore
2. Assistant Professor: University Institute of Physical Therapy, University of Lahore
3. Sana Akram: Assistant Professor, University Institute of Physical Therapy, University of Lahore

ARTICLE INFO

ORIGINAL ARTICLE

Corresponding author:
Hira Zahid, University institute of physical therapy, university of Lahore
Email: hirazahid158@gmail.com

Vol: 3 | Issue:1

ISSN Print: 2960-2580
ISSN Online: 2960-2599

Publisher:
Medical Research and Statistical Consultancy Training Centre (SMC-PRIVATE) Limited

Author's contributions
Hira Zahid: Idea conception, data collection and write up
Iqra Waseem: write up
Sana Akram: Literature search and write up

Keywords:
Prevalence, Plantar fasciitis, Pain, Heel Pain, Physiotherapists, Selfcare

ABSTRACT

Background: Plantar fasciitis is known as burning and aching plantar heel pain. It results from inflammation or irritation of plantar fascia, a thick band of tissue responsible for supporting the foot's arche and absorbing shock during movement. **Objective**: To assess how common plantar fasciitis is among physiotherapists in Lahore. **Methodology**: The total of 64 physiotherapists aged between 25-50 years was included with plantar fasciitis pain. A plantar fasciitis pain scale questionnaire administered among physiotherapists and performed a windlass test. SPSS version 24.0 software was utilized for data analysis. **Results**: The findings revealed that approximately 19% of the physiotherapists of Lahore suffered from plantar fasciitis. Of the population suffering from pain about 11(17.2%) have little pain, 10 (15.6%) have moderate pain and 3(4.7%) have severe pain. In which 33(51.6%) have pain withstanding for a prolonged time. And about 45 (70.3%) have not been suffering because of wearing appropriate shoes and maintaining proper posture. **Conclusion**: Physiotherapists may develop plantar fasciitis throughout their life and the intensity of pain can vary depending upon standing hours, and shoe wear.

INTRODUCTION

Physiotherapists are healthcare professionals and part of a multidisciplinary team. For decades, they are playing an important role in the healthcare system.[1]Musculoskeletal disorders arise due to poor posture, which imposes overpressure on the muscles and soft tissues.[2] Plantar fasciitis, often referred to as plantar heel pain, is a condition that effect approximately 11 to 15% of adult seeking professional care for foot issues.[3]The plantar heel consist of the deep fascia at the base of the foot, which plays a critical role in connecting the calcaneus to the toes. Anatomically, the fascia is divided into 3 elements: medial, lateral, and central. [4]

Plantar fascia occurs due to inflammation and degenerative changes in the plantar fascia, often caused by repetitive injury and stress, especially at its attachment point on the medial calcaneal tuberosity of heel. This leads to pain in the central to medial heel pain. Studies indicate that 11 to 15% of adults with foot pain experience plantar heel pain. While plantar fasciitis is typically a self-limiting condition, the pain can become chronic and disabling, often requiring several months of the rehabilitation.[5]

It plays a crucial role in the foot's normal biomechanics, and even small changes in foot structures or pain can impact balance and gait. This often results in slower walking pace, shorter stride length and unbalanced walking pattern.[6]Plantar fasciitis pain is often described as a burning or aching sensation. Plantar fasciitis more often affects unilateral, although approximately 30% of patients have bilateral symptoms.[7]

A key clinical feature of plantar fasciitis is heel pain, primarily affecting the medial part of the heel and sometimes radiating into the medial arc of the foot. The discomfort is typically most severe in the morning. Pain may also be worse during overuse conditions of the foot such as running.[8] The main cause of plantar fasciitis is repetitive strain injury to the ligament of the sole. Other causes include being overweight, prolonged standing or walking, and heel spurs.[9]

Plantar fasciitis is the leading cause of heel pain, affecting approximately 7% to 10% of the population. Risk factors include a high body mass index or sudden weight gain. Diagnosis is primarily based on symptoms, with ultrasound often used for confirmation. Research indicates that up to 49% of individual with plantar fasciitis continue to experience symptoms 1.5 to 5 years after onset.[10]

Heel pain is commonly observed in older teachers, possibly due to decrease elasticity of the plantar fascia. The prevalence of plantar fasciitis has been studied in the population with varying findings of 2.7-17.55%. A large-scale population in the US reported a prevalence of 0.85% among adults. Additionally, within 12 to 24 months of diagnosis, 60-80% of patients have been reported to experience plantar fasciitis.[11]

Of the working females of Multan, about 72% suffered from plantar fasciitis and were above 50 years. About 54% of the individual with plantar fasciitis report experiencing deep pain, while 46% describe it as superficial pain. Around 34% of those affected suffer from pain throughout the day at regular intervals.[12] The condition has been found to affect about 8.14% of physicians and 13.11% of nurses. Compared to the general population, the risk of developing plantar fasciitis is lower among physician but higher among nurses.[13]

Patient may experience pain along the central band of the plantar fascia, particularly in the area of the foot's medial arch.[14-17]

A cross-sectional online survey was conducted with a total of 695 participants. The prevalence of plantar fasciitis among them was found to be 37%. The study identified that extended period of walking or standing at work, along with inadequate workplace facilities,[17]were linked to a higher risk of developing the condition. Additional contributing factors included middle age, prolonged exercise, and gastrocnemius muscle tightness. The findings highlight the importance of raising public awareness about the risk factor and management strategies for plantar fasciitis through health education initiatives.[16]

Pallavi Yamini did her study in 2020. He took 100 subjects for the trial. A total of 100 female students from Galgotias University participated in the study, divided equally into 2 groups: one group of 50 individual with flat feet and the other 50 who wear high heels. The finding revealed 20% of subjects experienced the plantar fascia stretch, and 7% tested positive in the windlass test. Additionally, 27% of the total population were found to be risk of developing plantar fasciitis, with 19% of them being regular high heel users. Notably, all participants who tested positive in the windlass test were among those who wore heels.[17]

In a 2021 study conducted by Bhoir and GD, 100 healthy nurses from the Pravara institute were examined, including 30 male and 70 female nurses, all aged between 20-50 years. Among participants, 21% tested positive for sign of plantar fasciitis - 4% of males and 17% of females. The study concluded that female nurses have higher likelihood of developing plantar fasciitis compared to their male counterparts.[18]

In 2019, Abidin Haneef conducted a cross-sectional study among the police force in Peshawar to assess the prevalence of plantar fasciitis. He sent his 364 questionnaires. 360 have cooperated well. In the 360 population, 38 had had heel pain and 322 had not. In 38 population only 5 had plantar heel pain other 32 had not. [19]

Binu conducted a study comparing 100 individuals diagnosed with heel pain to a control group of people with no history of heel pain. Both groups completed a questionnaire assessing environmental and lifestyle factors. Among those with heel pain, the majority lived in homes with mosaic flooring, followed by marble, vitrified tile or carbonate flooring. However, when analyzing both groups, the highest prevalence of heel pain was found in individuals living in marble-floored houses. In contrast, the lowest incidence was seen in those residing in wooden floored or traditional cow-dung floored houses. The study suggests that type of flooring may influence the development of plantar fasciitis with marble flooring being associated with the highest incidence. [20]

The purpose of this study is to ascertain if plantar fasciitis occurs among physiotherapists because of their prolonged standing and lengthy workdays. Data was collected through self-reported forms completed by

physiotherapists, utilizing a visual analog scale to assess the severity and frequency of the condition. This approach aimed to evaluate the correlation between their occupational demands and the occurrence of plantar fasciitis.

MATERIALS AND METHODS

Study Design: This research was conducted as a cross-sectional study

Setting: Data was collected from different departments of physical therapy in different hospitals of Lahore i.e.:

- Pediatrics neuro-rehabilitation center of the University of Lahore Teaching Hospital,
- Physiotherapy Department of Wazir Hospital
- Al Shabir Health Care Center.

Duration of study: The study was finished within 6 months.

Sample Technique: It was Nonprobability purposive sampling.

Sample size: A total of 64 participants were included in the study. The sample size was determined by using the epi tool.

Sample selection criteria:

Inclusion Criteria:

- Willing to divulge details about pain.
- Windless test was used.
- Both gender male and female physiotherapists are included.
- Between the age of 25-50 years
- Working hours 7-8

Exclusion Criteria:

- Plantar fasciitis history before entering the field of physiotherapy.
- Physiotherapists who have not been working in the field for at least one year.
- Physiotherapists who have a past history of fracture.

Data collection procedure

This was an observational study conducted as a project involving physiotherapists in Lahore. The participants ranged in age from 25-50 years. A sample size of 64 was calculated using 5% margins of error and 95% of confidence intervals through the epi tool. To diagnose plantar fasciitis, windlass test was administered. Additionally, relevant demographic and medical information such as age, gender, weight, and medical history was collected using a predesigned proforma. Planar fasciitis pain scale (PFPS) questionnaires was also distributed among participants, consisting of pain related and control

questions specifically targeting symptoms of plantar fasciitis. After the approval of synopsis data was collected from a questionnaire used to gather the information. The SPSS-24.0 version was used to analyze the collected data for descriptive statistics. Frequency distribution was utilized to display the participant's demographic data. After the data analysis, the findings were summarized in accordance with the consultation with the statistician.

RESULTS:

There were 64 participants in this study. the participant's age ranges from 25-50 years the minimum age is 25 and the maximum age is 50. There were 64 participants of which 28% were male and about 72% were female. There were 64 participants, of which about 70.3 % were alright and 29.3% were suffering from planter fasciitis

		Frequency	Percent
Age groups	25-30	44	68.8
	31-35	10	15.6
	36-40	1	1.6
	41-45	2	3.1
	46-50	7	10.9
Gender	Male	18	28.1
	Female	46	71.9
Windlass test	Positive	19	29.7
	Negative	45	70.3

DISCUSSIONS

Plantar Fasciitis is the swelling of plantar fascia and tissue in the foot used during walking and foot movement. The windless test is used as a diagnostic procedure to assess plantar fasciitis. Khired and Najmi conducted an online cross-sectional survey. The total number of people who took part in the study was 695. Plantar fasciitis was observed to affect 37% of the individuals. And our study concluded about 29.3% of prevalence of Plantar fasciitis among physiotherapists.[18]

Abidin Haneef conducted a cross-sectional study in 2019 to investigate the prevalence of plantar fasciitis among security personal in the police force of Peshawar. The finding revealed that 10.6% of participants reported experiencing heel pain, while the prevalence of plantar fasciitis specifically was higher, at 13.2%.[21]

Pallavi Yamini completed her studies in 2020. He recruited 100 people for the trial. From Galgotias University. The findings revealed that 20% of the participants experienced a stretch in the plantar fascia, and 7% tested positive on the windlass test. Notably, 19% of individuals in our study demonstrated a positive windlass test result, which is significantly higher compared to the previously mentioned study. This suggests a greater prevalence of plantar fasciitis indicators in our sample. [20]

CONCLUSION

According to this study, Most of the Physical therapists from Lahore did not suffer from plantar fasciitis because they wear appropriate shoes but 46.9% suffered from plantar fasciitis because they wear inappropriate shoes

LIMITATIONS:

The study had a small sample size, the results may not be statistically significant or may lack the power to detect meaningful differences or associations. A small size can limit the reliability and validity of the findings. Height and body mass index were not reported, which had impact on how generalizable the findings were.

RECOMMENDATIONS

To improve the generalizability of the findings, researchers could aim for a larger and more diverse sample This would help capture a broader range of experiences and potential risk factors. Investigate the specific workplace factors contributing to plantar fasciitis pain, such as physical demands, workload, and adherence to ergonomic guidelines. Assessing these factors would help identify modifiable elements that can be targeted for intervention.

REFERENCES

1. Mahmood T, Salam A, Waseem I, Khalid A, Maqsood U. The Attitude of Physical Therapy Students Towards their Profession and Education in Punjab. Journal of Islamabad Medical & Dental College. 2022 Sep 28;11(3):182-7.
2. Mahmood T, Afzal MW, Waseem I, Arif MA, Mahmood W. Comparative effectiveness of routine physical therapy with and without instrument assisted soft tissue mobilization for improving pain and disability in patients with neck pain due to upper crossed syndrome. Annals of Punjab Medical College. 2022 Mar 31;16(1):45-50.
3. Jessup RL, Oates MJ, Johnston RV, Buchbinder R. Shockwave therapy for plantar heel pain (plantar fasciitis). The Cochrane Database of Systematic Reviews. 2019 Nov;2019(11).
4. Wearing SC, Smeathers JE, Urry SR, Hennig EM, Hills AP. The pathomechanics of plantar fasciitis. Sports medicine. 2006 Jul;36:585-611.

5. Hashmi R, Naeem L, Arif S, Habiba U, Irfan R, Zafar M. Frequency of Plantar Fasciitis among Females in Teaching Profession. Journal of Aziz Fatimah Medical & Dental College. 2020;2(2):53-7.
6. Dudoniene V, Balnytė M, Kuisma R. Comparison of static balance and gait between subjects with plantar fasciitis and age-matched controls. Journal of Back and Musculoskeletal Rehabilitation. 2022 Nov 10(Preprint):1-8.
7. Palomo-López P, Becerro-de-Bengoa-Vallejo R, Losa-Iglesias ME, Rodriguez-Sanz D, Calvo-Lobo C, López-López D. Impact of plantar fasciitis on the quality of life of male and female patients according to the Foot Health Status Questionnaire. Journal of pain research. 2018 Apr 27:875-80.
8. Latt LD, Jaffe DE, Tang Y, Taljanovic MS. Evaluation and treatment of chronic plantar fasciitis. Foot & ankle orthopaedics. 2020 Feb 5;5(1):2473011419896763.
9. Sandhu TB, Jamil A. Muscle energy technique versus passive manual soft tissue therapies on planter fasciitis: a systemic review. Pakistan Journal of Rehabilitation. 2023 Jul 6;12(2).
10. Sharma D, Rawat SK, Vadher S, Mani C, Shah F. Prevalence of Plantar Fasciitis Among Post Graduate Medical Students.
11. Hansen L, Krogh TP, Ellingsen T, Bolvig L, Fredberg U. Long-term prognosis of plantar fasciitis: a 5-to 15-year follow-up study of 174 patients with ultrasound examination. Orthopaedic journal of sports medicine. 2018 Mar 1;6(3):2325967118757983.
12. Rasenberg N, Bierma-Zeinstra SM, Bindels PJ, van der Lei J, van Middelkoop M. Incidence, prevalence, and management of plantar heel pain: a retrospective cohort study in Dutch primary care. British Journal of General Practice. 2019 Nov 1;69(688):e801-8.
13. Aiman U, Malik L, Zahoor a. pain and difficulty level in working females having plantar fasciitis of multan city: Pain & Difficulty in Working females with Plantar Fasciitis. Pakistan BioMedical Journal. 2022 Aug 31:46-50.
14. Sung KC, Chung JY, Feng IJ, Yang SH, Hsu CC, Lin HJ, Wang JJ, Huang CC. Plantar fasciitis in physicians and nurses: a nationwide population-based study. Industrial health. 2020;58(2):153-60.
15. Cornwall MW, McPoil TG. Plantar fasciitis: etiology and treatment. Journal of Orthopaedic& Sports Physical Therapy. 1999 Dec;29(12):756-60.
16. Arshad S, Sharif MW, Waseem I, Mahmood W, Ilyas HS. Individual and organizational barriers faced by physiotherapists in implementing evidence based practice-an analytical study. JPMA. The Journal of the Pakistan Medical Association. 2022 Oct;72(10):2030-3.
17. Khired Z, Najmi MH, Akkur AA, Mashhour MA, Bakri KA, Najmi M, Mashhour M, Bakri K. The

prevalence and risk factors of plantar fasciitis amongst the population of Jazan. Cureus. 2022 Sep 21;14(9).

18. Pallavi S, Yamini S, Megha Y, Jyoti S, Mangalam K, Sakshi A. To Compare the Prevalence of Plantar Fasciitis among Females Wearing Flat Foot Wear and Heels in Young Adults. Indian Journal of Physiotherapy & Occupational Therapy Print-(ISSN 0973-5666) and Electronic–(ISSN 0973-5674). 2020 Apr 25;14(2):177-80.
19. Bhoir KS, GD VV. Prevalence of Plantar Fasciitis Among Nurses at a Tertiary Care Centre in a Rural Area: A Cross Sectional Study. Int J Physiother Res. 2021;9(4):3900-06.
20. Abidin SZ, Haneef K, Malik NR, Mashal M, Zeb A, Rahman MU. Prevelance and Associated Risk Factors For Plantar Fasciitis Among Security Forces Personnel In Peshawar. Annals of Allied Health Sciences. 2019 Dec 31;5(2):20-3.
21. Binu MG, Iesa M, Vinodini C, Nizar A. Relationship of Prevalence of Plantar Fasciitis to the Type of Flooring-A Community Based Observational Study. Indian Journal of Public Health Research & Development. 2013 Apr 1;4(2): 54-56

ENERGY BAR FROM A COMPOSITE OF UNRIPE BANANA, SESAME, AND SEMOLINA FLOURS: DEVELOPMENT, SENSORY EVALUATION, PROXIMATE ANALYSIS, AND KEEPING QUALITY EVALUATION

Masuma Yaseen[1, 2], Nakyinsige Khadijah[1], Babirye Khadijah[1-3], Abbas Kisambira[1, 2], and Aqsa Abid[4]

1. Department of Food Science &Nutrition, Faculty of Agribusiness and Natural Resource Sciences, Islamic University in Uganda, P. O. Box 2555, Mbale, Uganda
2. Halal Assurance Institute Uganda, Plot 26, Wampewo Avenue, Kololo, Kampala, Uganda.
3. University Institute of Food science and Technology, Faculty of Allied Health Sciences, The University of Lahore. 1-Km, Defence Road, Lahore, Pakistan.
4. University Institute of Physical Therapy, Faculty of Allied Health Sciences, The University of Lahore. 1-Km, Defence Road, Lahore, Pakistan.s

ARTICLE INFO

ORIGINAL ARTICLE

Corresponding author:
Department of Food science and Nutrition, Faculty of Agribusiness and Natural Resource Sciences, Islamic University in Uganda, Main campus. P.O. Box 2555, Mbale, Uganda.
Email:
babirye.khadijah@iuiu.ac.ug
Vol: 3 | Issue:2
ISSN Print: 2960-2580
ISSN Online: 2960-2599

Publisher:
Medical Research and Statistical Consultancy Training Centre (SMC-PRIVATE) Limited

Author's contributions
Masuma Yaseen: Conceptualization, data collection, and writing original draft. **Nakyinsige Khadijah-** Supervision**; Babirye Khadijah-** Formal analysis, Review and editing**; Abbas Kisambira-** Methodology and review; **Aqsa Abid:** Data curation

Keywords:
Uganda plantain, consumer acceptability, unripe bananas, free fatty acid content, peroxide value

ABSTRACT

Background: Globally, the popularity and market for energy bars steadily increases, especially with increasing urbanisation. Fresh bananas suffer much postharvest losses mainly due to their short green life. Semolina, a by-product of wheat is usually destined to waste despite its nutritional profile. **Objective:** To developing an energy bar from a composite of unripe banana (*Musa accuminata)*, sesame (*Sesanum indicum)* and semolina flours, assessing the consumer acceptability and determining the nutrient plus keeping quality of the most accepted formulation. **Methodology:** A total of five energy bars from different composite flour formulations namely; A (20:20:60), B (20:10:70), C (10:10:80), D (30:30:40), and E (10:30:60) for unripe banana flour: sesame flour: semolina flour respectively was developed and tested for consumer acceptability using a nine-point hedonic scale. The most accepted energy bar formulation was further profiled for its proximate composition, selected minerals' content, and keeping quality using standard AOAC protocols. **Results:** Formulation D provided the most accepted energy bar regarding the texture, taste, plus overall acceptability, whereas the least accepted sample for most sensory attributes was from formulation A. No significant difference existed in colour for all the formulations. The most accepted energy bar contained 5.72% moisture, 1.02% ash, 5.39% crude protein, 2.94% crude fat, and 4.33% dietary fibre. This energy bar provided a high percentage of carbohydrates and energy value i.e. 72.98% and 1440 KJ/100g (360 calories) respectively. Its mineral analysis revealed 110.51mg/100g of calcium, 38.407 mg/100g of iron, and 399.291 mg/100g of potassium. Indices for the keeping quality of the most accepted energy bar showed a peroxide value of 1.87 and a free fatty acids content of 0.33.

Conclusion: This study developed a calories-rich energy bar which can be quickly utilized to fuel plus replenish the body in periods of high energy demand or as an energy-dense snack for people who dont have enough time for meal preparation, manifested in its high carbohydrate content and energy value obtained. Other than increasing the accessibility to cheap high-energy food sources, the present study showed potential of reducing post harvest losses of unripe bananas.

INTRODUCTION

Energy bars are ready-to eat nutritious foodswith good sensory characteristics,and targeted at people that require quick energy but do not have time for a meal.[1] A typical energy bar usually provides 200-300 calories, a low fat content of about 3-9 g, and is shaped in form of a bar or any other convenient form.[2] Numerous energy bars have been developed and are known by different names such as nutrition bars, sports bars, granola bars, meal replacement bars, protein bars, nutraceutical bars, snack bars, diet bars, cereal bars, among others. [2] Lately, energy bar sales and consumption have increased globally,[2] mainly because modern lifestyles require more time away from home.[3] There is need for a nutritious, pleasing, ready-to-eat, shelf-stable, proportionedyet portable method of food transport and availability; an energy bar can satisfy this demand. [2]

The bars which are well known by most rural households are compressed food bars used in emergency situations. Compressed food bars comprise of soya protein concentrate, baked wheat flour, sugars, vegetable fat, plus malt extract, and are used in relief effort for disasters if local foodis not available or accessible[4] In Uganda, there are few energy bar micro enterprises, for example in Nakasongola district which improves food security and income of farmers.[5] Composite flour is a binary or tertiary mixture of different flours, mostly wheat flour, with flours of differentcrops. Composite flour is found beneficial especially in developing countries becauseit encourages the use of locally available crops as flour. Local raw material replacement instead of wheat flour is rising because of the growing market for confectionaries.[6]

Banana is one of the fifth most crucial food crops across the globe. [1] More than 75% of farmers in Uganda grow bananas, making it the world's second largest producer after India[7]. Banana (*Musa accuminata spp.*)is a staple starchy food for about 80 million people in East Africa and a vital income source.[7] It is a reliable source of starch and energy hence ensuring food security for millions of people.[7] There are more than 120 banana varieties in Uganda, whichhave not yet been identified and/or documented anywhere else in the world. *Musa spp*, consisting of plantain plus dessert bananas,isone of the world's leading food crop as a source of energy in people's diet especially those residing in tropical or humid areas. [1] Although Uganda is regarded as food self-sufficient due to its productive soil and bi-annual rainfall, not all regions

of the nation have enough food, and some may experience food insufficiencies for some period in the year[5]. Therefore, the population in some areas of the country suffers from food insecurity and befalls victim of emergency situations[5].Despite the fact that Uganda is the leading producer of plantain plus banana in the sub-Sahara African region, this crop suffers post-harvest losses to a tune of 20-60%.[8]This mainly arises because of bananas' limited green life plus damage from improper produce handling after harvest. [1] A large amount of the produce thus goes to waste, leading to high physical and economic losses. [1] Therefore,using unripe banana in flour form might help in reducing such post-harvest losses. Moreover, the prospects of using banana flour for development of food bars and other snacks have been articulated in several previous studies. [1, 3]

Semolina is the hard part of coarsely-ground durum wheat, high in gluten and protein contents. The high gluten content present helps in binding of the raw materials during cooking of the energy bar. Semolina is also rich in iron; 100 g of semolina provides about 4.36 mg of iron.[9] Since the production of wheat in Uganda is low in addition to by-products of wheat not being well-known by the people,[10] development of energy bars will help increase people's nutritional awareness about semolina and ultimately increase its usage. Uganda being among the highest importers of wheat, encouraging semolina usage can lead to higher rate of wheat production in the country in addition to reducing wheat import costs, which shall eventually boost Uganda's economy.

Sesame (*Sesamun indicum* L.) seeds have for long been used extensively in traditional healing plus cooking purposes due to their nourishing benefits andcurative properties. Sesame is a vital source of phyto-nutrients for example, flavonoids, phenolic compounds, dietary fiber, anti-oxidants, anti-cancer compounds, as well as other health promoting properties. Sesame being a rich source of high quality edible oil and essential amino acids is thus a healthy/nutritious component for the energy bar. Therefore, the present study aimed at (i) development of an energy bar froma composite of unripe banana flour, sesame flour, and semolina flour (ii) carrying out consumer acceptability tests through sensory evaluation of different formulations prepared for the energy bar (iii) carrying out proximate analysis of the most accepted formulation of the energy bar, and (iv) determining the keeping quality of the most accepted energy bar formulation through peroxide value test and test for the free fatty acids content.

MATERIALS AND METHODS

Study design: Experimental study

Settings: Food Science Laboratory, Department of Food Science and Nutrition, Faculty of Agribusiness and Natural Resource Sciences, Islamic University in Uganda (IUIU).

Study duration: 4 months

Data collection procedure

Unripe banana, brown sesame seeds, and wheat grains were purchased from Mbale Central market. After being washed properly, banana fingers were peeled, followed by soaking in water to clean off the sticky sap that appears upon peeling. This was followed by slicing (using a kitchen grater with 3mm thickness) in water that was treated with food-grade citric acid (1 gof citric acid per litre of water) to avoid enzymatic browning. The slices were soaked for 15 min, then spread on a clean sheet of cloth under the sun for 2 days to achieve complete dryness. Dried slices were grinded in an electricstainless steel food grinder, then sieved using a stainless steel sieve, prior to being stored in a polythene bag. Sesame seeds were sorted to get rid of any visible foreign matter. They were washed with clean water to remove dustor any other residual foreign matter, followed by spreading the seeds on a clean sheet of cloth under the sun for 6 hours. Dried sesame seeds were then roasted on a dry pan set on medium heat of the electric coil, for 15 mintilla slightly brown colour and characteristic aroma were evident. Seeds were left to cool prior to being finely ground and sieved. Sesame seeds were then packed in a polythene bag and stored away from light and moisture, awaiting mixing with flour from other raw materials, as shown in Figure 1. After being sorted, wheat grains were conditioned by adding water and keeping them for 24 hours to mellow the inner endosperm. Wheat grains were then dried and grinded into the desired coarse-particle flour. This was followed by sieving and sifting of the wheat particles to allow heavy coarse particles (semolina) remain on the sieve. The composite flour formulations from each raw material, amount of honey, and ghee used in the production of energy bars are presented in Table 1.After cooking, the energy bar matrix was spread into rectangular moulds to be shaped into bars. Bars of 4cm by 12cm were cooled to luke-warm temperature before being cut out using a sharp knife (when the matrix was just warm), then allowed to completely cool to room temperature so they become firm, prior to packaging in air tight polythene packaging material.

Sensory evaluation Test

Sensory evaluation for the different energy bar formulations was carried out from the Food science and Nutrition laboratory, employing the nine-point hedonic scale ranging from extremely like to extremely dislike.[11] A total of 50 randomly selected IUIU students with no food allergies were used as panelists in the evaluation that consisted of 5 sensory attributes namely; color, texture, taste, flavour & overall acceptability. Asensory evaluation form and 5 g of the sample from each formulation were served to each panelist, in a random order. Panelists were also provided with drinking water to rinse their mouth before and between successive sample evaluation, to minimise any residual sample effect. After sensory

evaluation analysis, all the further tests were conducted on only the most accepted/highly ranked formulation.

Proximate analysis

This analysis involved determination of the moisture content, dietary fibre content, total ash, crude fat, carbohydrate content, crude protein, energy value using intenational standard protocols,[12] plus selected minerals (Ca, Fe, and K).

To determine the moisture content, clean crucibles were dried for 30 min in an oven and then cooled from a desiccators before weighing. The crucible weight was taken and 3g of the sample was weighed into a crucible. This was followed by drying the weighed sample(95^{o}Covernight) in the oven and cooling in desiccators. Weight of the crucible plus the dried sample was recorded, after which moisture content was calculated as follows;

$$\%\ Moisture = \frac{W2 - W3}{W2 - W1} \times 100$$

Where;

W_1 = initial weight of empty crucible (g)

W_2 = weight of crucible + sample before drying (g)

W_3 = final weight of crucible + sample after drying (g)

W_2-W_3 = loss in sample weight (g) after drying

To determine ash percentage, the dry ashing method was employed; crucibles were put in a muffle furnace overnight at 550^{o}C so as to ensure that any impurities on the surface burn off. The crucibles were cooled in a dessicator for 30min and weighed. A proportion of 5g sample was weighed into the crucible and put in a furnace, then heated at 550^{o}C overnight. The crucibles were weighed when the sample turned to grey and ash percentage was calculated as below;

$$\text{Ash (\%)} = \frac{\text{(weight of crucible + sample after drying)- (weight of empty crucible)}}{\text{Weight of sample}} \times 100$$

Determination of the crude protein content was done using the microkjeldahl method.About 4 g of the sample was weighed and placed in the Kjeldahl digestion flask. Sulphuric acid (20mL) and 10g of Na_2SO_4catalyst were added and the sample digested in a fume chamber for 60 min at 350 ^{o}C until the digest appeared clear pale green. The digest was left until completely cool, followed by addition of 100mL H_20 and mixing. The mixture was then transferred to a distillation flask and 85mL of saturated NaOH solution added from a measuring distillation unit. This was followed by distillation of the mixture into

25mL of 0.1N HCl containing 10mL of boric acid as an indicator. Distillation was continued till the flask contents 'bumped', and excess acid was titrated with 0.1N NaOHsolution.

% Nitrogen in the sample (N) = 14 x (V/1000) x 0.1 x (W/100)

Where; V = volume of 0.1N HCl added – volume of 0.1N NaOH used, W = weight of the sample

Crude protein = N x 6.25

To determine the crude fat percentage, 3g of the sample was accurately weighed into a thimble lined with a circle of filter paper. This was followed by placing both the thimble and content into a 50mL beaker and dried in a mechanical convection oven for 1 hour at 125 ºC. Crude fat was extracted with petroleum ether (40 °C) as a solvent using the soxhlet apparatus semi-continuous method (Soxhlet System HT, 1043 Extraction Unit). After the extraction process was finished, the fat extract was moved from the extraction flask into an evaporating dish that had been previously weighed; the solvent was evaporated till its odor could not be detected. The dish and its contents was dried in an oven for 30 min at 100 ºC, after which cooled in a desiccator. The dish plus content were then weighed.

Crude fat (ether extract) % = W2-W1 x 100/S

Where;

W1 = evaporating dish, W2 = weight of the evaporating dish plus contents after drying

S = weight of sample.

Determination of the dietary fibre content involved addition of 5g sample to 200mL of 1.25% H_2SO_4, heating for 30 min and filtering with a Buchner funnel. Distilled water was used to wash the residue until there was no more acid present. For 30 min, the residue was boiled in 200millilitres of 1.25% NaOH. This was filtered and repeatedly rinsed with distilled water till it became alkaline-free. This was followed by rinsing once with 10% HCl, twice with ethanol and thrice with petroleum ether. The residue was placed in a crucible and dried at 105ºC in an oven over night. It was cooled in a dessicator before being ignited for 90 min at 550 ^{0}C in a muffle furnace, after which allowed to cool, and reweighed.

$$\text{Total dietary fiber} = \frac{\text{Weight of ash residue}}{\text{Weight of sample}} \times 100$$

Determination of the carbohydrate percentage was done by difference method;[13]

% carbohydrates =100 - (% fat + % moisture + % ash + % protein)

Energy value was calculated from the formula;[14]

Energy value (KJ/100g) = 4 (% carbohydrates + % protein) + (9 x % fat)

Determination of the calcium content involved pipetting an aliquot of the test solution into a volumetric flask followed by addition of 1% $LaCl_3$ (w/v) solution to make a final concentration of 0.1% $LaCl_3$

(w/v).This was followed by diluting the solution with 1N HNO_3. Calcium determination was done with an atomic absorption spectrophotomer, following the manufacturer's instructions. Absorbance of the prepared standards and test solution was then measured against reagent blank.

$$\text{Ca (mg/100g)} = \frac{\text{Conc.} \times \text{dilution} \times 100}{\text{Weight of sample (g)} \times 1000}$$

Where Conc. = concentration of the sample (mg/L)

To determine the iron content present, the sample was exposed to wet digestion. An aliquot portion of the acidified sample was taken, diluted, and thenread from aninductively coupled plasma-optical emission spectrophotometer,at a wavelength of 248.3.

$$\text{Iron (mg/100g sample)} = \frac{\text{Conc.} \times V \times D \times 100}{W \times P \times 1000}$$

Where; Conc. = concentration of sample in mg/L

V= Total volume in mL

D= Dilution factor

W= Weighed sample in g

P= Sample solution taken, in mL

1000= conversion of mL to L

The results were reported as mg Fe/100g sample and as around number.

Potassium content of the sample was determined using a flame photometer. Standard solutions were prepared by dilution of stock solution of potassium. Volumes of 0.5, 1, 2, 4, 6, and 8 were diluted in 100mL flasks. The photometer was heated for 10 min then distilled water was fed into the instrument. The most concentrated standard solution was aspirated, and the readout was adjusted to 90 using the knobs. Distilled water was aspirated, followed by all the standard solutions. The results were recorded and a calibration curve drawn to calculate the results, using the equation below;

$$y = mx + b$$

Where; y = results from the photometer, x= concentration of the standard solution and M = coefficient

Keeping quality

This analysis involved assessment of the peroxide value plus the free fatty acids content. Peroxide value was determined using the AOCS method.[15] About 0.05g of sample was weighed into a 250 mL glass stoppered Erlenmeyer flask. This was followed by addition of 30mL of acetic acid and the flask swirled until complete dissolution of the sample, then addition of 0.5mL of saturated potassium iodide and the flask swirled for 1 min. This was followed by addition of distilled water and vigorous shaking to liberate

iodine from the chloroform layer. 0.01N sodium thiosulphate was added plusstarch solution (1 mL) as an indicator. Titration was done until the blue-gray colour disappeared and titre values were recorded.

$$\frac{(S-B)\times\ Nthiosulhate\ \times 1000}{weight\ of\ sample}$$

S= titration of sample

B= titration of blank

To determine the free fatty acid content, 0.2g of sample was weighed in glass vial and mixed with 50 mL of the solvent mixture. This was followed by titration with O.1N KOH solution to the end point of phenolphthalein indicator. The free fatty acid content was calculated from the equation;

Free fatty acid content = 56.1 × N × V/M

Where; N= Exact normality, V= volume of KOH and M= mass of the sample (g)

Statistical analysis

Data for the present study was processed using the SAS software, version9.2.Forsensory evaluation results, means were separated by the Duncan's multiple range test (DMRT) with 0.05 as the probability level, using one way analysis of variance.

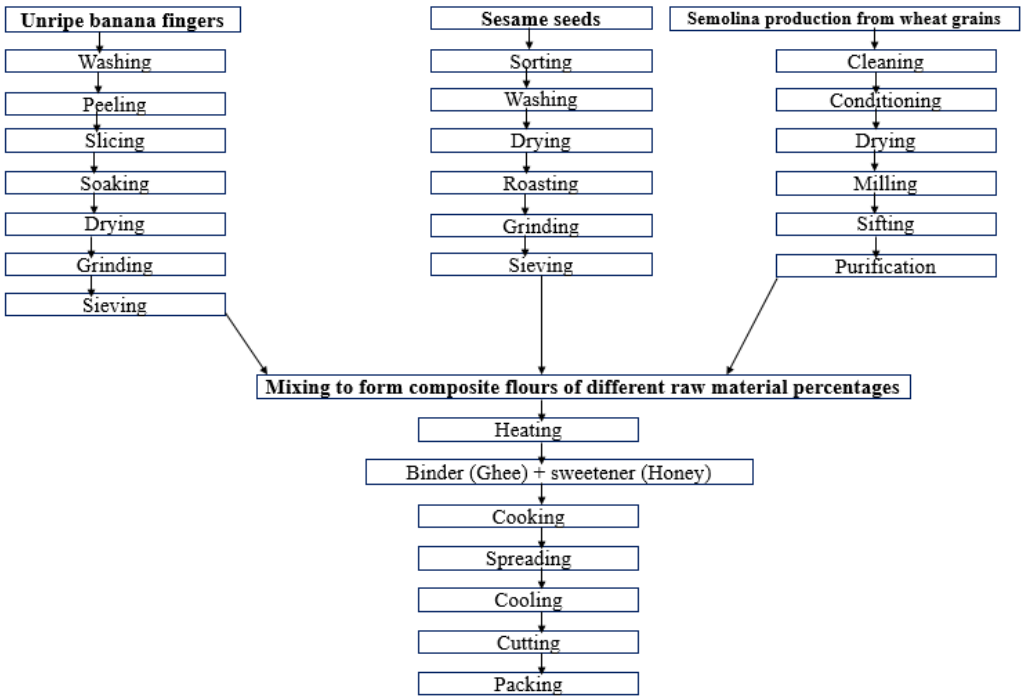

Figure 1: Process flow chart for the energy bar production from unripe banana flour, sesame seeds' flour, and semolina flour.

The composite flour formulations from each raw material, amount of honey, and ghee used in the production of energy bars are presented in Table 1. After cooking, the energy bar matrix was spread into rectangular moulds to be shaped into bars. Bars of 4cm by 12cm were cooled to luke-warm temperature before being cut out using a sharp knife (when the matrix was just warm), then allowed to completely cool to room temperature so they become firm, prior to packaging in air tight polythene packaging material.

Table 1: Composition of the different formulations of the produced energy bars.

Formulation code	Unripe banana flour (%)	Sesame flour (%)	Semolina flour (%)	Honey (g)	Ghee (g)
A	20	20	60	10	10
B	20	10	70	10	10
C	10	10	80	10	10
D	30	30	40	10	10
E	10	30	60	10	10

RESULTS

Sensory evaluation

Results of sensory evaluation of the energy bars developed from different formulations of unripe banana, semolina and sesame composites are presented in Table 2 below;

The most accepted sample of all was D in terms of texture, taste, and overall acceptability but sample E was preffered most in terms of flavour. Table 2 above shows there was no significant difference in the flavour of the energy bars produced by formulation A, B, C and D but there was a significant difference in the flavour of sample E. Results indicate that there was no significant difference in the colour of all the formulations. Generally, the least accepted sample for most sensory attributes was sample A.

Proximate composition and keeping quality of the most accepted energy bar formulation

Results of proximate analysis and keeping quality indicators from the most accepted energy bar formulation of unripe banana, semolina and sesame composites are presented in Table 3 below;

The energy bar's most accepted formulation contained 5.72% moisture which is considered a low moisture content. Similarly, this energy bar contained considerably low ash (1.02%) and protein (5.39%).The crude fat content and dietary fiber in the most accepted energy bar was 2.94% and4.33%respectively.This energy bar provided a high percentage of carbohydrates and energy value i.e. 72.98% and 1440 KJ/100g (360 calories) respectively.Mineral analysis revealed 110.51mg/100g of calcium, 38.407 mg/100g of iron,

and399.291 mg/100g of potassium. Indices for the keeping quality of the most accepted energy bar showed a peroxide value of 1.87 and a free fatty acids content of 0.33.

Table 2: Sensory evaluation results of the energy bars developed from unripe banana, semolina and sesame composites.

Formulation	Texture	Taste	Colour	Flavour	Overall acceptability
A	5.19±0.69^{c}	5.44±0.68^{c}	5.94±0.57^{a}	6.81±0.19^{b}	5.01±0.52^{c}
B	6.38±0.66^{b}	6.25±0.81^{b}	5.57±0.98^{a}	6.74±0.31^{b}	5.71±0.59^{bc}
C	5.86±0.47^{bc}	6.44±0.03^{b}	5.88±0.93^{a}	6.86±0.97^{b}	6.30±0.93^{b}
D	7.50±0.89^{a}	7.38±0.50^{a}	5.90±0.05^{a}	6.69±0.99^{b}	7.56±0.55^{a}
E	6.98±0.98^{ab}	6.89±0.48^{ab}	5.70±0.99^{a}	7.85±0.42^{a}	6.13±0.89^{b}

Values in the table are Mean±Standard deviation.Values with the same superscript in the same column are not significantly different at p≤0.05 using the DMRT.

Table 3 : Proximate composition and keeping quality results for the most accepted energy bar formulation.

Nutrients	Most accepted energy bar (Formulation D)
Moisture content (%)	5.72±0.44
Total ash (%)	1.02±0.03
Crude protein (%)	5.39±1.2
Crude fat (%)	2.94±0.01
Dietary fiber content (%)	4.33±0.26
Carbohydrate content (%)	72.98±10.63
Energy value (KJ/100g)	1440
Calcium content(mg/100g sample)	110.51±82.67
Iron content (mg/100g sample)	38.407±10.37
Potassium content (mg/100g sample)	399.291±254.31
Peroxide value	1.87±0.27
Free fatty acids content	0.33±0.02

Except the Energy value, all other values in the table are Mean±Standard deviation.

DISCUSSION

Texture is a key component which influences a food's sensory quality, in addition toits visual appearance, aroma, plus taste.[16] Texture evaluation is a dynamic intricate process which involves visual perceptions of the product surface, behaviour of the product in reaction to previous handling, and intergration of

sensations in the mouth felt during mastication and subsequent swallowing; all these are combined by the human brain and a unique sensation is brought up.[17] From the present study, texture of formulation D was the most preffered. This could be due to the fact that low quantity of semolina was used in formulation D compared to other formulations, as previously shown in Table 1. Semolina has a granular texture and therefore when it was mixed with the smooth unripe banana plus sesame flours, it dissolved into them forming a neither very soft nor very hard, less chewy texture.

Colour is one of the key quality attribute of a food raw material or product, which greatly influences consumer acceptance.[18] It provides information regarding the chemical composition, appropriateness for processing, plus shelf life. Findings of the current study indicated that no significant difference existed in colour of all the samples. The reason could be the presence of honey in an equal quantity throughout all the formulations (Table 1), because honey has an inherent dark-brown rich color which dominated in all the formulations. The other reason stems from accelerated millard reaction between honey and proteins present in all the formulations which led to production of brown melanins in all the energy bar formulations.[19] Other factors that impacted the colour of the developed energy bars include caramelisation and roasting of sesame seeds.

There are four primary categories of tastes that the tongue's receptors (buds) can identify i.e. sweet, sour, bitter and salty.[18] In contrast to the common knowledge that distinct tongue regions correspond to different tastes, some regions are more sensitive than others.[20] In the present study, the taste of formulation D was preffered most, followed by E. This could be due to the presence of higher quantities of roasted sesame flour in both formulations (as presented in Table 1). Several studies indicate that roasted sesame seeds add a unique nutty taste to many food products.[21]

Flavour of a food is greatly influenced by the chemical senses of taste plus smell.[18] These sensations can be changed by adding natural or artificial flavorants. Even with breakthroughs in instrumental analysis, sensory tests are the sole way to measure the flavor impressions which humans experience.[22] The most preffered flavour in the present study was of formulation E, and this could be attributed to the substantial quantity of sesame flour present in this formulation. This tallies with observations in a previous study[5]that sesame seeds not only develop a pleasant flavor during processing but also enhance the taste of food or make it more acceptable for consumption. Sesame contains flavour compounds like 2-acetyl-1-pyrroline (roasty), 2-furfurylthiol (coffee-like), 2-phenylethylthiol (rubbery), 4-hydroxy-2,5-dimethyl-3(2H)-furanone (caramel-like) and 2-furfuryl alcohol which are significant factors influencing the crushed sesame material's overall flavor.[19]

Overall acceptability is based on multiple organoleptic quality parameters and indicates the cumulative perception plus acceptance by panelists or consumers.[18] The overall acceptability of formulation D was preferred most. This could be linked to the composition ratio of unripe banana flour to sesame flour to semolina flour in formulation D which was almost balanced, hence a high mean overall acceptability score of 7.56±0.55and thus contributed to a better taste, texture and overall acceptability.

The energy bar's most accepted formulation contained 5.72% moisture which is considered a low moisture content, and is attributed to loss of moisture during heating and cooking unit operations of the composite processing. Moisture content has an important influence on the shelflife of the product; the lower the moisture content, the better the food's shelf life. Therefore, the low moisture content of the energy bar indicates a good storage stability, as most pathogenic and spoilage bacteria, plus some molds and yeasts, thrive in an atmosphere with high moisture content.[5] The ash content in the most accepted sample was 1.02%. Ash content isindicative of the approximate quantity of minerals in any food sample, [7, 18] and thus, the considerably low ash content in the present study implies that the formulated energy bar was generally not a good source of minerals.However, present results are different from previous findings,[2, 7]which reported that green banana flour plus snack bars made from them had a high ash and mineral concentration. This anomaly from present findings is because the present snack bar was a composite of flour from three different sources i.e. unripe banana, semolina, and sesame. The other reason for the anomaly could be the differences in varieties of green banana and the nutritional profile of the soil in different geographical regions, as observed in a previous study.[7]

The protein content in this energy bar was 5.39%. The reason for this considerably low value could be high heat processing which may have caused protein denaturation or millard reactions may have taken place in which the proteins reacted with carbohydrates and thus a reduction in the former's detectable quantity. In a related study which developed and compared energy bars from maize flour with those from unripe banana flour;[23] the bar with the least amount of protein and lipid contents was the one made with unripe banana flour. Similarly, another study showed a very low protein content from banana flour (2.40%).[2] Therefore, unripe banana flour used in the current study is responsible for the low protein content in the most accepted energy bar formulation.

The crude fat content in the most accepted energy bar was 2.94%, and this was most-likely contributed by the added ghee during cooking, plus some fat naturally inherent in sesame flour. This energy bar had a considerably high amount of dietary fiber (4.33%) and this was because of the high quantity of unripe banana flour in its formulation, which is in congruence with the emphasis of previous authors,[1, 3, 16]that unripe banana is a good source of dietary fibre.

Similarly, this energy bar provided a high quantity of carbohydrates and energy value i.e. 72.98% and 1440 KJ/100g (360 calories) respectively. As expected, carbohydrates are the main component of the energy bar in comparison with other major nutrients present in the energy bar product. This is because the ingredients used in its production contain high amounts of carbohydrates i.e. a generally high amount of unripe banana and semolina flour in the formulation, as these are rich sources of starch[3, 16] In the same vein, the previous work of other authors[2, 23]showed that unripe banana starch if added to different foods results into a product with high carbohydrate and starch content.This implies that the developed energy bar from the present study can be used to quickly fuel or replenish the body with glucose.

Mineral analysis revealed 110.51mg/100g of calcium. This could be attributed to the quantity of sesame added to the formulation (40%), because sesame contains 780 mg of Ca per 100 g whereas unripe banana and semolina provide lesser amounts of Ca. Furthermore, sesame has been reported to be an excellent source of Ca in several previous related studies.[5] The iron content was 38.407 mg per 100g, and thus, the developed energy bar can be used for iron supplementation in individuals that have high iron demands such as pregnant women.

The most accepted formulation contained 399.291 mg/100g of pottasium and this was mainly contributed by the unripe banana flour, as emphasized in similar experiments that green banana flour is a rich potassium source.[3, 7, 16] The potassium content in the most accepted formulation of the present study (399.291 mg/100 g) was close to the potassium content obtained by a previous researcher[7]i.e. 410 mg/100 g was the primary component found in each of the 15 unripe banana varieties under study. The developed energy bar can be used as a snack and is therefore an ideal food product for mantainance of bone integity in the most vulnerable age groups (adolescents, adults and the elderly), particularly among low income earners, if consumed regularly. To this end, some authorsrecommend that potassium supplementation through food ishelpful for people who have excessive potassium excretion through bodily fluids, especially athletes.[24] Potassium, an electrolyte that aids in controlling fluid balance, neuronal transmission, and acid-base balance, is abundant in bananas.[2]

Indices for the keeping quality of the most accepted energy bar showed a peroxide value of 1.87 and a free fatty acids content of 0.33.Peroxide value is assessed in shelf life studies to detect the initiation stage of the autoxidation process;[25] lipid oxidation is a common and an undesired chemical alteration that can affect a products's flavor, aroma, nutritional value and incertain situations, even the texture.[26] Theobserved peroxide value and free fatty acids content in the current study are attributed to the quantity of ghee that was added into the formulation (10g)plus fat naturally present in sesame. The low quantity of free fatty acidsindicates that the energy bar has good keeping quality and can thus be kept for a

considerably long period without deterioration (keeping other factors constant). Low fat content arrests rancidity and off-flavour production during storage[18]and thus extended shelf-life of the product.

CONCLUSION

This study developed a calories-rich energy bar from unripe banana flour, sesame seeds flour, and semolina flour,which can be quickly utilised to fuel plus replenish the body in periods of high energy demandor as an energy-dense snack for people who dont have enough timefor meal preparation. The study revealed that the developed energy bar had a high carbohydrate content and energy value. The study also showed potential of reducing post harvest losses in unripe bananas, by converting them into flour and further product development. Development of such products can increase the availability of cheap high-energy food sources.

RECOMMENDATIONS: There is need to further investigate the bioavailability of nutrients in the most accepted formulation of the energy bar. Further studies should be carried out on the storage stability of the energy bar,using preservatives. The most accepted formulation may be used for development of other food products such as cookies or other confections.

REFERENCES

1. Ekafitri R, Desnilasari D, Mayasti N, Kristanti D, Indriati A, editors. The Effect Used Nangka (Musa X Paradisiaca L.) Plantain Flour With A Different Process to The Macro-Micronutrient and Sensory of A Snack Bar. BIO Web of Conferences; 2023: EDP Sciences.

2. Abdel-Salam FF, Ibrahim RM, Ali M. Formulation and evaluation of high energy-protein bars as a nutritional supplement for sports athletics. Am J Food Technol. 2022;10:53-65

3. Ekafitri R, Kurniawan YR, Desnilasari D, Surahman DN, Indriati A. Shelf-life assessment of energy banana bar using acceleration method with critical moisture content approach. Food Sci Technol. 2020;41:163-8

4. UN World Food Programme. (2024). https://www.wfp.org › specialized-nutritious-food. Accessed on November 14 2024.

5. ABBAS K. Energy bar microenterprise for improved food security and farmers'incomes in nakasongola district: Makerere university; 2009.

6. Goryńska-Goldmann E, Gazdecki M, Rejman K, Łaba S, Kobus-Cisowska J, Szczepański K. Magnitude, causes and scope for reducing food losses in the baking and confectionery industry—a multi-method approach. Agriculture. 2021;11(10):1-20

7. Dotto J, Matemu AO, Ndakidemi PA. Nutrient composition and selected physicochemical properties of fifteen Mchare cooking bananas: A study conducted in northern Tanzania. Scientific African. 2019;6:e00150

8. Naziri D, Mayanja S, Namanda S, Nalunga A, Abass A, Wanda K, et al., editors. Postharvest losses along the cooking banana, potato and cassava fresh value chains in Uganda. 1st All Africa Postharvest Congress; 2017: Organizing Committee of 1st All Africa Postharvest Congress and Exhibition.

9. Nduka EF. Evaluation of the Physicochemical and Sensory Properties of Yam-Based Semolina Fortified with Orange-Fleshed Sweet Potato and Beetroot Flours. Afr J Agricul Sci Food Res. 2024;14(1):21-38

10. Chemayek B, Baguma C, Wasukira A, Walimbwa K, Woniala B, Kakhasa E, et al. Increasing wheat production and productivity through science-based knowledge and innovations for a competitive wheat manufacturing value chain in Uganda. 2023

11. Peryam DR, Pilgrim FJ. Hedonic scale method of measuring food preferences. Food technology. 1957(11):9-14

12. AOAC. (2002). Official methods of analysis. Association of Analytical Chemists. Washington DC.

13. Mullan WMA. Labelling-determination of energy content of food. (Accessed from http://www.dairyscience.info/energy_lebel.asp 18/04/2024). 2006

14. AOAC. (2019). Official method of analysis chemists. In: Horwitz, W. (Ed.), Maryland, USA 950.46, 21st edn.

15. AOCS. (1990). Official methods and recommended practice of American Oil Chemical Society, 4th edn. Champaign, Illinois.

16. Sarifudin A, Afifah N, Indrianti N, Desnilasari D, Kristanti D, Ratnawati L, et al. Optimization of banana bar formulation to provide a nourishing snack for toddlers using response surface methodology. Food SciTechnol. 2020;41(suppl 1):21-8

17. Liu D, Deng Y, Sha L, Abul Hashem M, Gai S. Impact of oral processing on texture attributes and taste perception. J Food Sci Technol. 2017;54(8):2585-93.https://doi.org/10.1007/s13197-017-2661-1

18. Khadijah B, Abbas K. Development, physico-chemical properties and proximate composition of maize based-soymilk. Pioneer J Bio Med Res. 2023;1(3):35-44

19. Salunkhe DK, Chavan JK, Adsule RN, Kadam SS. World oilseeds: Chemistry, Technology and Utilization. 1992:371-95

20. Spence C. The tongue map and the spatial modulation of taste perception. Curr Res Food Sci. 2022;5:598-610.https://doi.org/10.1016/j.crfs.2022.02.004

21. Mostashari P, Mousavi Khaneghah A. Sesame seeds: a nutrient-rich superfood. Foods. 2024;13(8):1-53

22. Civille GV, Carr BT, Osdoba KE. Sensory evaluation techniques: CRC press; 2024.

23. Utrilla-Coello RG, Agama-Acevedo E, Osorio-Díaz P, Tovar J, Bello-Pérez LA. Composition and starch digestibility of whole grain bars containing maize or unripe banana flours. Starch-Stärke. 2011;63(7):416-23

24. D'Elia L, Barba G, Cappuccio FP, Strazzullo P. Potassium intake, stroke, and cardiovascular disease: a meta-analysis of prospective studies. J Am Coll Cardiol. 2011;57(10):1210-9

25. Garg A, Sharma R, Dey P, Kumar A. Food auto-oxidation: An overview. Antioxidants Effects in Health. 2022:43-68

26. Shahidi F, Hossain A. Role of Lipids in Food Flavor Generation. Molecules. 2022;27(15).https://doi.org/10.3390/molecules27155014

EMOTIONAL REGULATION AMONG PARENTS OF AUTISTIC CHILDREN: A DESCRIPTIVE STUDY IN PAKISTANI POPULATION

Umber Nawaz[1], Asma Zafar[2], Muhammad Yahya Qureshi[1]

1. Post Graduate Scholar, Applied Psychology, Virtual University, Pakistan
2. Lecturer, Psychology, Virtual University, Pakistan

ARTICLE INFO

ORIGINAL ARTICLE

Corresponding author:
Dr. Umber Nawaz, Department of Psychology, Virtual University
Email: dr.umber21@gmail.com

Vol: 3 | Issue: 1
ISSN Print: 2960-2580
ISSN Online: 2960-2599

Publisher:
Medical Research and Statistical Consultancy Training Centre (SMC-PRIVATE) Limited

Author's contributions
Umber Nawaz: Idea concertation, data collection, analysis, write up
Asma Zafar: write up and literature review
Muhammad Yahya Qureshi: data collection, analysis, write up

Keywords:
Autism, Autism- Spectrum disorder, Emotional Regulation, Emotional Intelligence, Suppression, Re-appraisal

ABSTRACT

Background: Emotional Regulation may help to prevent parents of children with disabilities from psychological anguish and to support their child's healthy development. **Objective:** To identify the variables that influence parents of Autistic children to employ both adaptive and maladaptive ER techniques. **Methodology:** It was a cross-sectional survey design in nature because the research collected the data from the respondents at a single point of time, the researcher adopted non-probability purposive sampling technique due to being an unknown percentage of Autistic population in Pakistan. The sample size was 57. By adding 20% of drop out the estimated sample size was 70. Parents with at-least 1 autistic child, Age range was 20 years to 50 years, Divorced and Widowed was excluded. Data was collected on a structured questionnaire IIPB protocol. The questionnaire was already pilot tested and had been used in almost 60 countries across the world. **Results:** Among 70 parents, out of which 35(50%) were Fathers and 35(50%) were Mothers. The Mean Age of participants was 34.5±5.69. Emotional Regulation in parents was found out to be 39.24±16.79. The minimum emotional regulation was 7.00 and maximum score was 66.00. One of the sub-types of Emotional regulation, Reappraisal had a mean score of 23.40±10.35 with minimum score 4.00 and maximum score was 40.00. Suppression was another sub-domain of emotional regulation with mean score of 15.84±7.13 minimum score was 2.00 and maximum score was 27.00. **Conclusion:** It was concluded that reappraisal and emotional regulation was more frequently employed by both parents.

INTRODUCTION

Emotions/ emotional manifestations result in behavior, whenever intrinsic or extrinsic processes are altered, this process is known as emotion regulation (ER). [1] These methods are described in a variety of ER approaches, such as suppression and reappraisal. [2] Adaptive emotion regulation (ER) techniques may

help parents manage the stress that comes with raising a kid with a disability. ER may help to prevent parents of children with disabilities from psychological anguish and to support their child's healthy development. [3]

Autistic/ Autistic Spectrum Disorders (ASD) is defined by recurrent and limiting behaviors, as well as the social and communication impairments, which vary in intensity among individuals. [4]In a study done in 2022 by Zeidan and colleagues, illustrated that the number of participants in the samples, which ranged from 465 to 50 million, was fairly substantial, and with the median of 100/ 10,000, occurrence ranged 1.09/ 10,000 to 436/10,000. Similar to the previous study, the majority of research studies were conducted in America and Northern-Europe, but there are more research studies from places that weren't previously well-represented, such as Africa as well as the Middle East. [5-7]

According to Morris et al., parental ER is regarded as a straight technique by which parents may affect their children's socio-emotional functioning. Parental ER has been linked to a number of developmental outcomes in children who are generally developing, particularly psychopathological disorders and socio-emotional functioning. [8] However, it is unclear how parents of children with autism spectrum disorders emergency room visits might affect the main symptoms of their children. Autistic children's parents may endure extreme stress due to their children's autistic symptoms, emphasizing the importance of emotional regulation. [9]

Parents of such adolescents struggle more to manage many stressful situations in day-to-day life. Parental demands rise, they are put under greater financial burden to pay for necessary medical bills, they must devote more time to the child, and this all adds to the psychological stress and weight on parents.[10]

Mothers continue to bear an overabundance of the responsibility for raising a disable child, making them more susceptible to stress linked to child care and more likely to exhibit depressive, anxious, health issues, social isolation, and low self-esteem. The emotional makeup of parents may also be influenced by the genetic etiology of ASD [11]

To the best of the researcher's knowledge, the experience of fatigued parents with autistic children has never been examined from their point of view. There hasn't been much research done in Pakistan on the stress that parents of autistic children experience. As a result, it is challenging to determine how much parenting an autistic child affects parents. In order to better understand how parents of autistic children regulate their emotions, the current study will give voice to worn-out parents from the perspective of the Pakistani population.[12]

MATERIAL AND METHODS

The study was descriptive, cross-sectional survey. The data was collected from the respondents at a single

point of time without manipulating any variable/variables in existing phenomenon. Non-probability purposive sampling technique due to being an unknown percentage of Autistic population in Pakistan. Sample size was estimated through the following formula:

n = z 2 (E)(1-E)/c2

Where:

z = standard normal deviation set at 95% confidence level[13]

E = Acceptable error

C= confidence interval (confidence interval (0.95). The sample size was 57. By adding 20% of drop out the estimated sample size was 70

Inclusion / Exclusion Criteria

- Parents of both gender were included [14]
- Parents with at-least 1 autistic child. [14]
- Age range was 20 years to 50 years. [14]
- Divorced and Widowed was excluded. [14]
- Age of the Child/Children range between 4 year- 26 years were included. [14]

IIPB protocol was used as a tool for data collection comprise statements regarding emotional regulation strategies and comprise seven points Likert scale e.g. strongly agree, agree, somewhat agree, neutral, somewhat agree, disagree, and strongly disagree.

RESULTS

Among total 70 parents, out of which 35(50%) were Fathers and 35(50%) were Mothers. 56 (80%) parents had 1 autistic child, 9 (12.9%) had 2 autistic children, 3 (4.3%) parents had 3 autistic children and 2 (2.9%) had 4 autistic children. The Mean Age of participants was 34.5±5.69. The minimum age was 21 years and the Maximum age was 49 years. The Parents with autistic children minimum 1 and maximum 4 kids, however, mean and SD count of autistic children were 1.30±.688. Emotional Regulation in parents was found out to be 39.24±16.79. The minimum emotional regulation was 7.00 and maximum score was 66.00.

One of the sub-types of Emotional regulation, Reappraisal had a mean score of 23.40±10.35 with minimum score 4.00 and maximum score was 40.00. Suppression was another sub-domain of emotional regulation with mean score of 15.84±7.13 minimum score was 2.00 and maximum score was 27.00. Pearson Chi-Square of gender and emotional regulation, p-value ≥ .005 showed that there was a difference between the mean values strongly significant. 7 parents said that they never need emotional

regulation, while 4 parents said that they need it rarely. Whereas, 7 parents said that they required emotional regulation occasionally. Although, 18 parents required emotional regulation sometimes. Similarly, 15 parents needed emotional regulation often. There were other 10 parents who wants emotional regulation most of the time and only 9 parents need emotional regulation always.

Table-1: Frequency distribution of gender and Total No. of Autistic Children

		Frequency	*Percent (%)*
Gender	***Fathers***	*35*	*50.0*
	Mothers	*35*	*50.0*
Total No. of Autistic Children	***1***	*56*	*80*
	2	*9*	*12.9*
	3	*3*	*4.3*
	4	*2*	*2.9*

Table-2: Descriptive statistics of age (years), total No. of autistic children, emotional regulation, reappraisal and suppression

	Mean	*SD*	*Min*	*Max*
Age (Years)	***34.50***	***5.70***	***21.0***	***49.0***
Total No. of Autistic Children	*1.30*	*0.688*	*1*	*4*
Emotional Regulation	*39.24*	*16.79*	*7.00*	*66.00*
Reappraisal	*23.40*	*10.35*	*4.00*	*40.00*
Suppression	*15.84*	*7.13*	*2.00*	*27.00*

Table -3: Comparison of Emotional Regulation and Gender

		Emotional Regulation							***Total***
		Never	***Rarely***	***Occasionally***	***Sometimes***	***Often***	***Most of the times***	***Always***	
Gender	***Fathers***	*6*	*2*	*0*	*5*	*8*	*8*	*6*	*35*
	Mothers	*1*	*2*	*7*	*13*	*7*	*2*	*3*	*35*
Total		*7*	*4*	*7*	*18*	*15*	*10*	*9*	*70*

p-value = 0.005

DISCUSSION

The current study was to evaluate the need of emotional regulation adopted by the parents of autistic children due to depression, stress and emotional exhaustion. There was a sample of 70 parents out of which 35 were fathers and 35 were mothers.

This research study showed that mean age of participants came out to be 34.5±5.69. The min-age was 21 years while the max-age was 49 years. The previous study done in 2020 explained that Mother age (years) 37.5±7.2 and Father age (years) 42.7±8.7.[15]

In the current study emotional Regulation in parents was found out to be 39.24±16.79. The minimum emotional regulation was 7.00 and maximum score was 66.00. One of the sub-types of Emotional regulation, Reappraisal had a mean score of 23.40±10.35 with minimum score 4.00 and maximum score was 40.00. Suppression was another sub-domain of emotional regulation with mean score of 15.84±7.13 minimum score was 2.00 and maximum score was 27.00. 11 parents had very low reappraisal approach, 17 parents had low reappraisal, 24 had moderate level of reappraisal and 18 parents had high level of reappraisal. A previous study showed that mother's emotional regulation's showed that mothers with ASD kids used positive reappraisal less frequently than those mothers of kids with intellectually disabled ($q = 4.03$, $p = .02$, Cohen's $d = 0.68$).[15]

This existing study showed that nevertheless, 18 out of 35 parents occasionally used emotional regulation techniques. Similarly, 15 parents want emotional regulation often. There were other 10 parents who want emotional regulation most of the time and only 9 parents need emotional regulation always.

CONCLUSIONS

It was concluded that reappraisal as a sub-type of emotional regulation was more frequently employed by both parents. This study also suggests that as the age of the parents increases the reappraisal, suppression and emotional regulation strategies were habitually implemented by parents of autistic children.

STRENGTHS AND LIMITATIONS

1. This study adds to the existing global knowledge to present a latest holistic worldwide view on epistemological, social, and pragmatic stances. This helps in devising effective caring strategies for parents' relevant distress.
2. The major difficulties/ hurdles in accessing parents of autistic children as well as in data collection were; some parents were shy, some had complex of being less/ uneducated, some were unavailable due to their prolonged professional /other engagements, and few hesitated / avoided sharing their opinions, as they perceived that having an autistic child is a social stigma. Most of the autistic child(ren) were accompanied by caregiver (either some relative or maid) who were not the appropriate respondents for the study.
3. There was no mechanism to identify or minimize the effect of so many other moderating, mediating or confounding stressors that the respondents may have been facing in other areas of their lives (e.g.,

work stress, extended family conflicts, neighbors' conflicts, and diversity and variance of beliefs, religious impact, community intervention programs and other key life events).

4. The fact that the current study was cross-sectional and correlation, which prevents us from drawing inferences about the causal pathways connecting psychological discomfort, emotion regulation, and parenting, is one of its limitations.

IMPLICATIONS AND SUGGESTIONS

1. Testing a longitudinal, mediational model of the relationship between psychological distress, emotion control, and parenting in many populations, particularly clinically at-risk populations, may be beneficial for future research.
2. The relationship between parental emotion control and their capacity to understand the thoughts and reasons behind their children's behavior, as well as how these affective and cognitive processes may jointly contribute to sensitive parenting behavior, may be examined in future research.

REFERENCES

1. Majeed R. The "puzzle" of emotional plasticity. Philosophical Psychology. 2022;35(4):546-68.

2. Lippert MW, Sommer K, Flasinski T, Schomberg J, Pflug V, Christiansen H, et al. Bochum Assessment of Avoidance-based Emotion Regulation for Children (BAER-C): Development and evaluation of a new instrument measuring anticipatory avoidance-based emotion regulation in anxiety eliciting situations. Plos one. 2023;18(1):e0279658.

3. Keleynikov M, Benatov J, Cohen N. Emotion Regulation among Parents Raising a Child with Disability: A Systematic Review and Conceptual Model. Journal of Child and Family Studies. 2023:1-18.

4. Lord C, Elsabbagh M, Baird G, Veenstra-Vanderweele J. Autism spectrum disorder. The lancet. 2018;392(10146):508-20.

5. Al-Mamri W, Idris AB, Dakak S, Al-Shekaili M, Al-Harthi Z, Alnaamani AM, et al. Revisiting the prevalence of autism spectrum disorder among Omani children: a multicentre study. Sultan Qaboos University Medical Journal. 2019;19(4):e305.

6. Alshaban F, Aldosari M, Al-Shammari H, El-Hag S, Ghazal I, Tolefat M, et al. Prevalence and correlates of autism spectrum disorder in Qatar: a national study. Journal of Child Psychology and Psychiatry. 2019;60(12):1254-68.

7. Chinawa JM, Manyike PC, Aniwada EC, Chinawa AT, Obu HA, Odetunde OI, et al. Prevalence and socioeconomic correlates of autism among children attending primary and secondary schools in south east Nigeria. African Health Sciences. 2016;16(4):936-42.

8. Han ZR, Shaffer A. The relation of parental emotion dysregulation to children's psychopathology symptoms: The moderating role of child emotion dysregulation. Child Psychiatry & Human Development. 2013;44:591-601.

9. Le Vigouroux S, Charbonnier E, Scola C. Profiles and age-related differences in the expression of the three parental burnout dimensions. European Journal of Developmental Psychology. 2022;19(6):885-904.

10. Vogel D, Falter-Wagner CM, Schoofs T, Krämer K, Kupke C, Vogeley K. Interrupted time experience in autism spectrum disorder: empirical evidence from content analysis. Journal of autism and developmental disorders. 2019;49:22-33.

11. Zhao S, Chen WJ, Dhar SU, Eble TN, Kwok OM, Chen LS. Pursuing genetic testing for children with autism spectrum disorders: What do parents think? Journal of Genetic Counseling. 2021;30(2):370-82.

12. Tarbox CM, Silverman EA, Chastain AN, Little A, Bermudez TL, Tarbox J. Taking ACTion: 18 simple strategies for supporting children with autism during the COVID-19 pandemic. Behavior Analysis in Practice. 2021;14(4):1099-127.

13. Schultheis AM, Mayes LC, Rutherford HJ. Associations between emotion regulation and parental reflective functioning. Journal of child and family studies. 2019;28:1094-104.

14. Ardıç A, Olçay S. Investigation of the relationship between the burnout level of parents of children with autism spectrum disorder (asd) and asd symptom level and family needs by regression analysis. Egitim ve Bilim. 2021;46(206).

15. Megreya AM, Al-Attiyah AA, Moustafa AA, Hassanein EEJRiASD. Cognitive emotion regulation strategies, anxiety, and depression in mothers of children with or without neurodevelopmental disorders. 2020;76:101600.

COMPARISON OF HEMATOLOGICAL PARAMETERS OF JAK2 POSITIVE AND JAK2 NEGATIVE PATIENTS OF POLYCYTHEMIA VERA: A MINI-REVIEW

Adnan Khan[1], Afshan Nosheen[2], Fahad Ur Rehman[3], Muhammad Maaz Arif[4]

1. Dept. of Hematology, University of Health Sciences, Lahore
2. Dept. of Biochemistry, Superior University, Lahore
3. Dept. of Health Sciences, Khyber Medical University, Kohat
4. Contech International Health Consultants, Lahore

ARTICLE INFO

REVIEW ARTICLE

Corresponding author:
Muhammad Maaz Arif: Contech International Health Consultants, Lahore
Email: maazarifbutt@gmail.com

Vol: 3 | Issue:1
ISSN Print: 2960-2580
ISSN Online: 2960-2599

Publisher:
Medical Research and Statistical Consultancy Training Centre (SMC-PRIVATE) Limited

Author's contributions
Adnan Khan: Idea and Main write up, **Afshan Nosheen:** Literature search, **Fahad Ur Rehman:** Literature search and writeup
Muhammad Maaz Arif: Literature search, review and writeup

Keywords: Polycythemia Vera; Janus Kinase 2; Erythropoietin; Polymerase Chain Reaction; Mutation

ABSTRACT

Polycythemia vera (PV) is a malignant disorder with a different frequency based upon age and population. The present review aimed to determine the frequency of Janus Kinase-2 (JAK2) positive and JAK2 negative patients with PV and compare the hematological parameters. Operating polymerase chain reaction (PCR), studies found that mutations in the patients of JAK-2 V617F gene with low serum erythropoietin (EPO) and bone marrow depicted hypercellularity with increased red cell mass. PV is a subtype of myeloproliferative neoplasms (MPN), causing hyperviscosity and thrombotic complications. It's symptoms include fatigue, headache and pruritus. It's incidence ranges from 0.02 to 2.8 per 100,000. The JAK-STAT pathway mainly encompasses normal blood cell production and function, but irregularities can lead to disease conditions.

INTRODUCTION

Polycythemia Vera (PV), a hematopoietic stem cell clonal disorder marked by uncontrolled proliferation of granulocytes, erythrocytes and megakaryocytes, that follows hyperviscosity in the blood and thrombotic complications. The World Health Organization (WHO) defines PV as a type of chronic myeloproliferative disease [1]. Janus kinase 2 gene (JAK2-V617F) mutation affects part of JAK kinase known as pseudo kinase [2]. Firstly, it was reported in 1892 [3]. Further, polycythemia is split into two categories: primary polycythemia (Polycythemia vera), which is a multipotent hematopoietic stem cell

disorder; and secondary polycythemia, called erythrocytosis, which occurs due to the accumulation of the erythropoietin hormone [4]. Fatigue and itching, microvascular symptoms (such as migraines, dizziness, headaches, paresthesia, and unusual chest pain), and characteristics like splenomegaly, hyperviscosity, leukocytosis, thrombocytosis, thrombotic, and bleeding problems are among the clinical symptoms of PV. Acute myeloid leukemia or secondary myelofibrosis could develop from PV [5].

In 1903 and 1892, William Osler and Vasquez, respectively, showed rheumatism and stroke as clinical features of disease. Another scientist, Thomas Person, identified (famous for the phlebotomy technique) the use of leeches and bloodletting, which was a successful therapy for decades. A scientist from Framingham studied haemoglobin concentration and cerebral infraction, according to his study and stroke. 1972, man Hb ≥ 15 and female Hb ≥ 14 had twice as many cerebral infarctions [6].

This mini-review article explores the literature on the comparison between JAK2 positive and JAK2 negative patients of polycythemia vera, conducted at Khyber Medical University, Kohat, Pakistan, over a four-month period from February to May 2020. The studies on PV diagnosed cases and hematological parameters analysis were sourced from Google Scholar databases published between 1990-2023 in English language. The keywords used included “polycythemia”, “polycythemia vera”, “JAK2”, “hematological parameters” and “patients”.

Polycythemia can lead to several complications, each contributing to serious health risks. One of the potential complications is leukemia progression, with around 5% of cases progressing to acute myeloid leukemia (AML), a difficult-to-treat condition. The initiation of AML has been linked to certain medications like radioactive phosphorus, pipobroman, and chlorambucil [7]. Complications include recurrent nosebleeds and gastrointestinal bleeding, often due to iron deficiency anemia may complicate interpretation, particularly when assessing changes in bone marrow [8]. Thrombosis, a risk due to increased blood viscosity, increases the chances of venous and arterial clots, that may lead to to issues like cerebral ischemic infarctions and digital infarctions, and Budd-Chiari syndrome [9]. Additionally, polycythemia, can reduce oxygen delivery to tissues, due to compromised lung function that affects oxygen transfer, even with a high red blood cell count [5, 7]. Heart-related issues are prevalent including hypertension, heart failure, and an increased risk of stroke, which arise due to heightened blood viscosity and the additional strain placed on the heart [5]. These complications enhance the significance of closely monitoring and managing polycythemia to prevent further health risks.

The annual incidence of PV in European countries ranged from 0.4 to 2.8 per 100,000 people [10]. PV patients have approximately 90% mutations in JAK2V617F [11]. Recent studies from European countries depicts that polycythemia is the most common myeloproliferative disorder [12]. PV is a rare condition in

children, with a yearly incidence ranging from 0.02 to 2.8/100000 patients, increasing with age. The highest prevalence is between 70 and 79 years, with 1.6-fold higher mortality rates. JAK2, a cytoplasmic tyrosine kinase, has four groups in humans: JAK1, JAK2, JAK3, and TYK2.15. Its molecular weight is 120-140 kDa and irregularities in the JAK-STAT pathway can lead to disease conditions [13]. In 2007, V617F-negative patients with polycythemia vera showed multiple mutations in JAK 2's 12 axon, revealing that JAK2 12 axon 12 controls the erythropoietin channel [14]. About 80% of PV patients exhibit symptoms, including headaches, pruritus, fatigue, dyspnea, dizziness, visual changes, weight loss, and increased sweetness. The primary cause of morbidity and death in PV adult patients is thrombosis and bleeding [15].

The World Health Organization defines diagnostic criteria for erythroid colony formation, including two major criteria: high hemoglobin levels in men and women, the presence of a JAK2 mutation, and three minor criteria: bone marrow biopsy, low serum erythropoietin levels, and endogenous erythroid colony formation. Patients fulfilling the 2 major criteria and at least 1 minor criterion should be confirmed as diagnosed cases of polycythemia vera (PV) [16].

Cytogenetics is particularly useful in the diagnosis of myeloid disorders. It gives way or path in several pathogenic studies [17]. Since 2005, over 20 JAK2 gene mutations have been identified, with V617F being the most prevalent, accounting for 97% of PV patients, making it a crucial diagnostic criterion. [18].

In relation to comparative analysis of JAK2 positive and JAK2 negative patients**,** Akram et al.'s study found that hematological parameters of mutation-carrying individuals do not significantly differ from other PV patients. The 15-year survival rate for V617F positive patients is 76%, compared to 94% for wild type V617 patients. The clinical characteristics of PV patients are shown in Table 1 [18].

Table 1: Characteristics of Polycythemia Vera patients, including the detection of JAK2 V617F and exon 12 mutations.

Patient characteristics	**Patients n=24 (%)**	
Gender	Males	16 (66.7%)
	Females	8 (33.3%)
Median age (years)	57	
Platelet count (x10^9/L)	100-450	02 (8.3%)
	>450	22 (91.7)
Mean Platelet count (x10^9/L)	552±253	
Total leukocyte count (x10^9/L)	4-11	05 (20.8%)
	>11	19 (79.2%)
Mean TLC (x10^9/L)	17.6± 9.1	
Mean Hematocrit (%)	51.4±5.2	
Hemoglobin level in peripheral blood	>16g/dl	11 (45.8%)
Mean corpuscular Volume (MCV)	<77fl	18 (75.0%)
Mean MCV	77.2±13.0 fl	

Mean corpuscular Hemoglobin (MCH)	<27pg	20 (83.4%)
Mean MCH	25.6±3.9 pg	

Silver et al.'s study revealed that patients with PV diagnosis exhibit age-related hypercellularity due to pan-myelosis, characterized by a high number of megakaryocytic patients with cytologic pleomorphism and mild atypia [16]

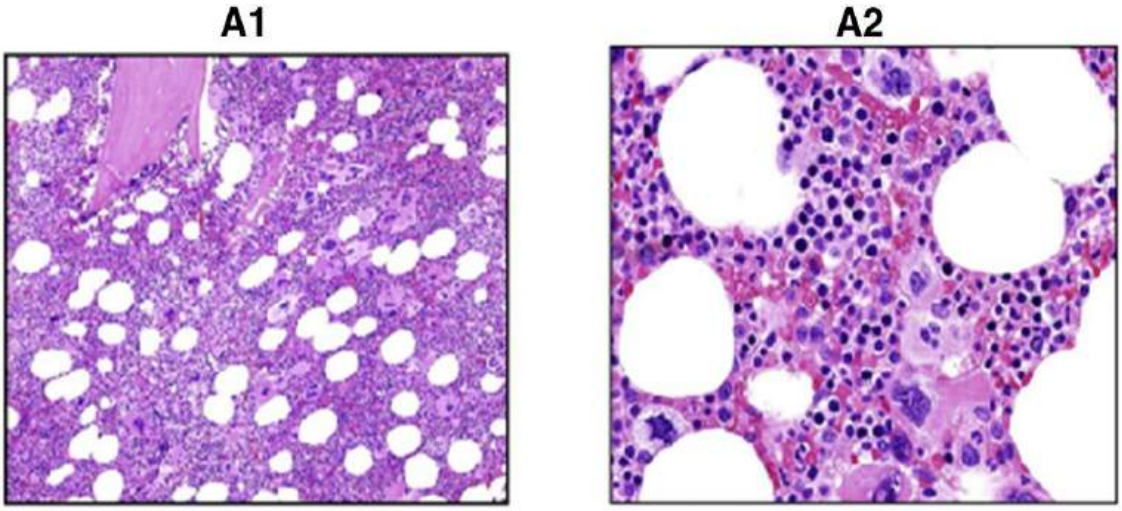

Figure 1: The hematoxylin and eosin sections of JAK2V617F positive study cases display typical PV morphology. (A1) The photo shows a hypercellular marrow with panmyelosis, with a 3200-magnification view (A2) The photo shows a 3400-magnification image of the same case, revealing pleomorphic megakaryocytes in loose clusters [16]

The study also showed that 28 patients diagnosed with PV found that only 7 met all the criteria for PV, including JAK2 positivity, increased red blood cell (RCM), hemoglobin, hematocrit, low serum EPO, and bone marrow. Only 10 and 20 met the elevated hemoglobin and hematocrit criteria, respectively (Table 2) [16].

Table 2: The diagnostic findings for the diagnosis of MPNs (n = 30)

Parameters						Number of patients	Final diagnosis
JAK2	RCM	HGB	HCT	BM biopsy result	EPO		
+	+	+	+	+	+	7	Polycythemia vera
+	+	–	+	+	+	7	Polycythemia vera
+	+	–	–	+	+	4	Polycythemia vera
+	+	–	+	+	–	2	Polycythemia vera
+	+	–	–	+	–	3	Polycythemia vera

+	+	+	+	+	*	3	Polycythemia vera
+	+	–	+	+	*	1	Polycythemia vera
+	+	–	–	+	*	1	Polycythemia vera
+	–	–	+	–†	+	1	Essential thrombocytopenia
+	–	–	–	–‡	–	1	Primary myelofibrosis

* EPO obtained 3 months after diagnosis

† (+ve for ET)| ‡ (+ve for PMF)| + (+ve for PV)| - (-ve for PV)

In another study done by Karkucak et al., it was found that 60% of patients (89 out of 148) diagnosed with polycythemia vera (PV) and essential thrombocytopenia (ET) had JAK2-V617F (+) mutations. JAK2 (+) patients had higher WBC (12.9×109/L) and Hb count (15.56 g/dL) and lower platelet count (683×10^9/L) than JAK2 (-) patients. Statistical significance tests showed that the group with the (+) mutation had significantly higher levels of WBCs, Hb and splenomegaly ($p = 0.021$, $p = 0.010$, $p = 0.014$). The differences between JAK2 positive and negative hematological parameters are displayed below (Table 3). [16]

Table 3: Characteristics stratified by JAK2-V617F mutation status patients of PV and ET [1]

Parameters	**JAK2-V617F (+) (n = 89)**	**JAK2-V617F (-) (n = 59)**
Gender (Male/Female)	36/53	26/33
Age	62.17 ± 12.85	50.03 ± 14.28
WBC ($\times10^9$/L)	12.9 ± 5.6	11.1 ± 3.8
Hb (g/dL)	15.56 ± 3.01	14.22 ± 3.07
Platelet ($\times10^9$/L)	683(124–2763)	968(155–2900)

A study done by Vera showed that reducing cardiovascular (CV) risk factors including hypertension, smoking, diabetes, hypercholesterolemia, and obesity can lead to better management among PV patients [19].

Treatments should be focused on normalizing Hb, Hct, and leukocytosis as well as lowering cardiovascular risk factors like obesity, diabetes, hypertension, dyslipidemia, and smoking in order to lower the risk of thrombotic events. Above all, hypertension and its treatment play a major part. In fact, recent research indicates that angiotensin converting enzyme inhibitors (ACEIs) may also help lower erythrocytosis [20]. According to Berlin et al., the initial treatment of PV is accomplished via phlebotomy, myelosuppression using radioactive phosphorus ^{32}P or other myelosuppressive chemotherapeutic drugs, hydroxyurea and interferon [15]. Since decades ago, polycythemia vera (PV) has been treated using

interferons (IFNs). Clinical trials evaluating IFN in patients with PV showed significant hematological and molecular response rates, suggesting that IFN may have disease-modifying effects. Ropeginterferon alfa-2b, or ROPEG, is an isoform-specific monopegylated IFN that differs from earlier IFNs in terms of dose frequency and tolerability. Due to its enhanced pharmacokinetic and pharmacodynamic qualities, ROPEG can be administered on a monthly basis throughout the maintenance phase and at longer intervals of every two weeks [21].

Therapeutic phlebotomy is a popular method to reduce hematocrit levels and thromboembolic risk. However, increasing hepcidin and using antisense oligonucleotides against Tmprss6 mRNA can also achieve similar results. Tmprss6-ASO, administered less frequently, may improve compliance and reduce side effects at injection sites [22]. Passamonti highlighted aspirin as the primary antiplatelet drug in PV treatment, but withdrawing it if bleeding occurs is advised, especially in patients over 60 years old, severe thrombosis, or splenomegaly. The first-line treatment for cytoreductive therapy in PV is hydroxyurea [23].

Patients with PV requiring cytoreductive therapy must use oral antimetabolites to inhibit ribonucleoside reductase, preventing DNA synthesis [24].

CONCLUSION

PV is a subtype of MPN, a hematopoietic stem cell disorder causing hyperviscosity and thrombotic complications. Its frequency varies regionally and age-wise, with 5% diagnosed annually in patients under 40. The highest incidence is between 70 and 79 years old. PV's clinical features include headache, pruritus, and fatigue. Morbidity and death in adult patients are due to thrombosis and bleeding. Diagnosis is based on JAK2-positive patients with low EPO levels, increased RCM, and hypercellularity. Aspirin is commonly used in PV antiplatelet therapy.

REFERENCES

1. Karkucak M, Yakut T, Ozkocaman V, Ozkalemkas F, Ali R, Bayram M, et al. Evaluation of the JAK2-V617F gene mutation in Turkish patients with essential thrombocythemia and polycythemia vera. Molecular biology reports. 2012;39:8663-7.

2. Johansson P, editor Epidemiology of the myeloproliferative disorders polycythemia vera and essential thrombocythemia. Seminars in thrombosis and hemostasis; 2006: Copyright© 2006 by Thieme Medical Publishers, Inc., 333 Seventh Avenue, New ….

3. Spivak JL. Polycythemia vera: myths, mechanisms, and management. Blood, The Journal of the American Society of Hematology. 2002;100(13):4272-90.

4. Hoffbrand V, Higgs DR, Keeling DM, Mehta AB. Postgraduate haematology: John Wiley & Sons; 2016.

5. Benevolo G, Marchetti M, Melchio R, Beggiato E, Sartori C, Biolé CA, et al. Diagnosis and management of cardiovascular risk in patients with polycythemia vera. Vascular Health and Risk Management. 2023:765-78.

6. Prchal JT. Philadelphia Chromosome–Negative Myeloproliferative Disorders: An Historical Perspective. ASH Education Program Book. 2008;2008(1):68-.

7. Satheesh S. Insights into the Pathophysiology and Therapeutic Targets of Consequences Induced by Polycythemia in COPD. Biosciences Biotechnology Research Asia. 2023;20(4):1225-35.

8. Nicol C, Lacut K, Pan-Petesch B, Lippert E, Ianotto J-C. Hemorrhage in essential thrombocythemia or polycythemia vera: epidemiology, location, risk factors, and lessons learned from the literature. Thrombosis and Haemostasis. 2021;121(05):553-64.

9. Tefferi A, Vannucchi AM, Barbui T. Polycythemia vera: historical oversights, diagnostic details, and therapeutic views. Leukemia. 2021;35(12):3339-51.

10. Moulard O, Mehta J, Fryzek J, Olivares R, Iqbal U, Mesa RA. Epidemiology of myelofibrosis, essential thrombocythemia, and polycythemia vera in the E uropean U nion. European journal of haematology. 2014;92(4):289-97.

11. Limsuwanachot N, Rerkamnuaychoke B, Chuncharunee S, Pauwilai T, Singdong R, Rujirachaivej P, et al. Clinical and hematological relevance of JAK2 V617F and CALR mutations in BCR-ABL-negative ET patients. Hematology. 2017;22(10):599-606.

12. Prchal JT. Polycythemia vera and other primary polycythemias. Current opinion in hematology. 2005;12(2):112-6.

13. Ibrahim IK, Hassan R, Ali EW, Omer A. Polycythaemia vera among Sudanese patients with special emphasis on JAK2 mutations. Asian Pacific Journal of Cancer Prevention: APJCP. 2019;20(1):41.

14. Scott MW. The severed snake: Matrilineages, making place, and a Melanesian Christianity in Southeast Solomon Islands: Carolina Academic Press; 2007.

15. Berlin NI. Polycythemia vera: diagnosis and treatment 2002. Expert review of anticancer therapy. 2002;2(3):330.

16. Silver RT, Chow W, Orazi A, Arles SP, Goldsmith SJ. Evaluation of WHO criteria for diagnosis of polycythemia vera: a prospective analysis. Blood, The Journal of the American Society of Hematology. 2013;122(11):1881-6.

17. de Thé H, Chomienne C, Lanotte M, Degos L, Dejean A. The t (15; 17) translocation of acute promyelocytic leukaemia fuses the retinoic acid receptor α gene to a novel transcribed locus. Nature. 1990;347(6293):558-61.

18. Current W. Evaluation of WHO criteria for diagnosis of polycythemia vera: a prospective analysis. 2013.

19. Vera P. Leukocytosis as a major thrombotic risk factor in patients with.

20. Benevolo G, Vassallo F, Urbino I, Giai V. Polycythemia vera (PV): update on emerging treatment options. Therapeutics and clinical risk management. 2021:209-21.

21. Krecak I, Skelin M, Verstovsek S. Evaluating ropeginterferon alfa-2b for the treatment of adults with polycythemia vera. Expert review of hematology. 2023;16(5):305-16.

22. Casu C, Liu A, De Rosa G, Low A, Suzuki A, Sinha S, et al. Tmprss6-ASO as a tool for the treatment of polycythemia vera mice. PLoS One. 2021;16(12):e0251995.

23. Fox S, Griffin L, Harris DR. Polycythemia vera: rapid evidence review. American family physician. 2021;103(11):680-7.

24. Passamonti F. How I treat polycythemia vera. Blood, The Journal of the American Society of Hematology. 2012;120(2):275-84.

MECHANISTIC PATHWAYS LINKING POSTTRAUMATIC STRESS DISORDER, SLEEP DYSREGULATION, AND CARDIOVASCULAR DISEASE: AN INTEGRATIVE REVIEW

Muhammad Ajmal Dina[1], Anam Arshed[2], Khalid Pervaiz[3], Arifa Jabeen[2], Mahnoor Khan[4], Duaa Fatima Rana[5], Aqsa Iqbal[6]

1. Department of Biostatistics and Epidemiology, Shahid Sadoughi University of Medical Sciences, Yazd, Iran
2. Rahbar Medical and Dental College, Lahore Pakistan
3. Nishtar medical University Multan, Pakistan
4. Quaid-e-Azam Medical and Dental College, Bahawalpur, Pakistan
5. Sharif Medical and Dental College, Lahore Pakistan
6. Punjab Rangers Teaching Hospital Lahore Pakistan

ARTICLE INFO

REVIEW ARTICLE

Muhammad Ajmal Dina,
Department of Biostatistics and Epidemiology, Shahid Sadoughi University of Medical Sciences, Yazd, Iran.
Email: ajmaljhl@gmail.com

Vol: 3 | Issue:1
ISSN Print: 2960-2580
ISSN Online: 2960-2599

Publisher:
Medical Research and Statistical Consultancy Training Centre (SMC-PRIVATE) Limited

Author's contributions
Muhammad Ajmal Dina: Main idea, literature synthesis, **Anam Arshed:** Main writeup, **Khalid Pervaiz:** Corrected and added tables data, **Arifa Jabeen:** Data synthesis and drafting, **Mahnoor Khan:** Drafting, **Duaa Fatima Rana:** discussion, **Aqsa Iqbal:** Drafting and reference management

Keywords: Post-Traumatic Stress Disorder; Cardiovascular Diseases; Sleep Disorders; inflammation; intervention

ABSTRACT

Background: Posttraumatic stress disorder (PTSD) is increasingly recognized as a significant predictor of cardiovascular disease (CVD), with emerging evidence highlighting the role of sleep dysregulation as a key mediator in this relationship. PTSD is associated with physiological dysregulation, including hypothalamic-pituitary-adrenal (HPA) axis dysfunction and chronic inflammation, alongside behavioral risk factors such as poor sleep quality, which may exacerbate CVD risk. **Objective**: This integrative review aims to synthesize existing evidence on the mechanistic pathways linking PTSD, sleep disturbances, and CVD, with a focus on identifying sleep as a modifiable intervention target to mitigate cardiovascular risk in trauma-exposed populations. **Methodology**: A systematic search was conducted across PubMed, PsycINFO, and Web of Science databases, focusing on studies published between 2000 and 2023. Keywords included "PTSD," "sleep disturbances," "cardiovascular disease," and related terms. Study selection followed PRISMA guidelines, with inclusion criteria encompassing peer-reviewed articles examining PTSD, sleep, and CVD outcomes or biomarkers. Data extraction captured study design, population, PTSD/sleep assessment tools, CVD endpoints, and mechanistic findings. **Result**: The review reveals consistent evidence that individuals with PTSD exhibit elevated CVD risk (27–59% higher incidence), driven by sleep disturbances such as insomnia (70–90% prevalence), obstructive sleep apnea (20–40%), and nightmares (50–70%). These disturbances contribute to CVD via sympathetic overactivation, endothelial dysfunction, and systemic inflammation. Interventions targeting sleep (e.g., cognitive behavioral therapy for insomnia, continuous positive airway pressure) show promise in improving both sleep and cardiovascular outcomes. **Conclusion**: Sleep dysregulation is a critical, modifiable pathway in the PTSD-CVD relationship. Integrating sleep-focused interventions into PTSD care may reduce cardiovascular morbidity. Future research should prioritize longitudinal studies and precision-based strategies to clarify causal mechanisms and optimize clinical outcomes for trauma survivors.

INTRODUCTION

Cardiovascular disease (CVD) remains the most reason of mortality worldwide[1], responsible for more deaths annually than all cancers and chronic lower respiratory diseases combined. In the USA the economic burden of CVD is estimated to cost $363.4 billion, annually, comprising $216 billion in direct medical expenses and $147.4 billion in lost productivity due to premature mortality[2]. While the major public health impact, up to 80% of CVD cases may be preventable with behavioral modifications, underscoring the significance of targeting modifiable risk factors in prevention strategies, the critical role of psychological health in cardiovascular outcomes, as underlined in a recent Scientific Statement by the American Heart Association[3]. In psychological conditions, posttraumatic stress disorder (PTSD) has gotten attention for its robust relationship with amplified incidence of cardiovascular events[4], as well as myocardial infarction, stroke, and heart failure[5]. Meta-analytic results from nine prospective studies uncovered that individuals with PTSD face a 61% elevated risk of coronary heart disease or related mortality compared to those without the disorder[6]. The observational position of PTSD is a potentially modifiable risk factor for CVD, and deserves deep exploration of the mechanisms hidden in this association[7]. PTSD is a trauma-related psychiatric condition[8], that advances in the subset of individuals following exposure to traumatic events[9]. Lifetime trauma is remarkably common[10], affecting 50 to 89 % of population, PTSD prevalence is estimated at 13 % for women, 6.2 % for men [11]. That disorder can be branded by invasive traumatic memory[12], prevention behavior and insistent changes in cognition, mood, causing major basic deficiency[13]. PTSD is connected to many physical health comorbidities, its link with CVD is most and well-documented across diverse populations[14], with veterans and community samples[15], association remains even after adjusting the traditional CVD risk factors[14, 16], proposing exclusive pathways connecting PTSD to cardiovascular pathophysiology[17].

Possible mechanisms among PTSD to CVD include both physiological dysregulation with health behaviors[16]. Physiologically, PTSD is linked with chronic dysfunction of the hypothalamic-pituitary-adrenal axis and with autonomic nervous system[18]. It can be added to a sustained sympathetic arousal with decreased stress response recovery[19]. Systemic inflammation observed in PTSD populations may increase atherosclerotic processes and endothelial dysfunction, aggregate susceptibility to hypertension with other CVD signs[16]. Individuals with PTSD showed high rates of smoking, substance use, physical inactivity with poor medical adherence, all recognized contributors to cardiovascular risk [20].

Sleep showed a multidimensional construct with duration, continuity, and the presence of specific disorders such as insomnia and obstructive sleep apnea (OSA) [21]. The evidence connects sleep disturbances as independent risk factors for CVD [22], but the role in the PTSD to CVD pathway remains inadequately observed [23]. Like short sleep duration and poor sleep quality have been associated with hypertension, arrhythmias, and elevated inflammatory markers [24], and OSA is linked to amplified cardiovascular morbidity with intermittent hypoxia and sympathetic activation [25]. Mostly, PTSD normally co-occurs with sleep disorders [26], upto 90% of patients reporting clinically significant insomnia and 40–90% exhibiting OSA symptoms [27]. The overlay raises questions about sleep disturbances aggravate cardiovascular risk in trauma exposed individuals and targeted sleep interventions could mitigate the risk[28].

PTSD is linked to higher cardiovascular disease (CVD) risk. Sleep problems are common in PTSD and worsen CVD outcomes. This review explores how poor sleep connects PTSD to heart disease. We examine stress hormones, inflammation, and nervous system effects. Studies show insomnia and sleep apnea raise CVD risk in PTSD patients. Treatments like therapy and CPAP may improve both sleep and heart health. Understanding this link can help develop better interventions. Sleep-focused care could

reduce CVD in trauma survivors. More research is needed on long-term effects and tailored treatments. This work bridges mental health and heart disease prevention.

MATERIALS AND METHODS

Data and Study selection

The data extraction was performed using a standardized template capturing, Study characteristics, Author, year, sample size, population like veterans, community, study design. Key variables PTSD assessment tools, sleep measures, CVD outcomes like hypertension, myocardial infarction. Mechanistic findings Biomarkers, pathways, and intervention outcomes.

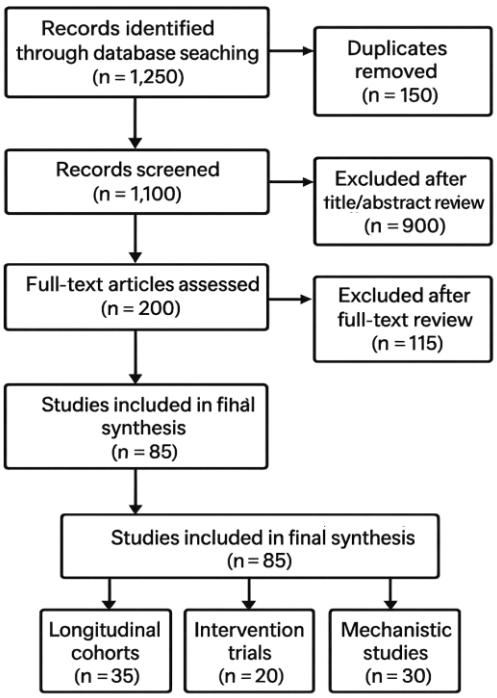

Fig-1: PRISMA Flow Diagram

Quality and data Synthesis

Study quality was evaluated using, Newcastle-Ottawa Scale (NOS) for observational studies [29], assessing selection, comparability, and outcome or exposure measurement. A narrative-thematic approach was employed to integrate evidence and key themes were identified. Physiological pathways, HPA axis dysregulation, autonomic dysfunction, inflammation. Behavioral pathways: Health risk behaviors like smoking and sleep-specific mechanisms. Tables 1- 4 were developed to summarize study findings, mechanistic pathways, and interventions, with patterns and contradictions discussed in the Results section.

Ethical Considerations:

This review synthesized existing published data, ethical approval was not required.

Strengths and Limitations

The review's strength on its integration of multidisciplinary evidence across diverse populations and study designs. The heterogeneity in PTSD measurement tools with dependence on observational data limits causal inferences. Upcoming updates could incorporate emerging RCTs or biomarker-driven studies to strengthen mechanistic insights. The methodology ensures transparency, reproducibility, and

alignment with the review's aim to elucidate PTSD-sleep-CVD pathways and inform targeted interventions.

Results

The table shows that many studies examined the association between posttraumatic stress disorder (PTSD) and cardiovascular disease (CVD) risk, highlighting consistent evidence of elevated CVD risk among individuals with PTSD. Longitudinal and prospective cohort studies report PTSD-linked increases in CVD incidence ranging from 27% to 59%, with amplified risks of myocardial infarction, stroke, heart failure, and hypertension. Meta-analyses consolidate these findings, demonstrating a 55 to 59% amplified CVD risk across diverse populations[30]. Mechanistic insights of the studies propose association with endothelial dysfunction and arterial stiffness, although others link PTSD to atherosclerosis and metabolic syndrome.

Table 1: Summary of key studies on PTSD and CVD risk

Sample Size	Study Design	Findings	Limitation	References
4,462	Longitudinal	PTSD linked with double-fold amplified risk of CVD; stronger in younger adults.	Reliance on self-reported CVD, potential confounding by depression.	Boscarino JA. Psychosom Med. 2008;70(6):668-76.
1,946	Cohort study	PTSD symptoms expected higher incidence of coronary heart disease (CHD).	Mostly male sample, limited generalizability.	Kubzansky LD, et al. Arch Gen Psychiatry. 2007;64(1):109-16.
562	Cross-sectional	PTSD associated with endothelial dysfunction, raised arterial stiffness.	Low sample size, cross-sectional design limits causal inference.	Vaccarino V, et al. JAMA Cardiol. 2019;4(5):437-45.
1,000	Longitudinal	PTSD associated with 53% high risk of incident CVD over 3 years.	Self-reported PTSD symptoms, potential recall bias.	Edmondson D, et al. Am J Cardiol. 2013;112(2):178-82.
49,978	Meta-analysis	PTSD associated with 55% increased risk of CVD across 11 studies.	Heterogeneity in study designs and PTSD assessment methods.	Sumner JA, et al. Psychol Med. 2015;45(7):1477-88.
2,424	Prospective cohort	PTSD expected high risk of myocardial infarction (MI) and stroke.	Limited diversity in sample, residual confounding possible.	Roy SS, et al. J Am Heart Assoc. 2018;7(15):e008065.
1,647	Longitudinal	PTSD symptoms associated with increased carotid atherosclerosis.	Lack of clinical CVD outcomes, low sample size.	Cohen BE, et al. Psychosom Med. 2019;81(1):42-50.
287	Cross-sectional	PTSD linked with high prevalence of hypertension, dyslipidemia.	Cross-sectional design, cannot establish causality.	Ahmadi N, et al. J Affect Disord. 2011;134(1-3):453-8.

Sample Size	Study Design	Findings	Limitation	References
3,093	Prospective cohort	PTSD linked to 27% high risk of heart failure over 7 years.	Mostly male veterans.	Wentworth BA, et al. Am J Cardiol. 2013;112(1):29-33.
2,519	Longitudinal	PTSD linked with 41% increased risk of CVD in a veteran population.	Limited to veterans, potential confounding by combat exposure.	Scherrer JF, et al. J Am Heart Assoc. 2019;8(11):e011133.
1,220	Cross-sectional	PTSD linked with high prevalence of metabolic syndrome and CVD risk.	Cross-sectional design, self-reported CVD outcomes.	Dong M, et al. Psychosom Med. 2014;76(8):628-36.
32,826	Prospective cohort	PTSD symptoms expected high risk of stroke in women.	Focus on women, limited generalizability to men.	Kubzansky LD, et al. Stroke. 2019;50(11):2999-3005.
1,647	Longitudinal	PTSD related to increased risk of atrial fibrillation.	Low sample size, limited diversity in study population.	Turner JH, et al. J Am Heart Assoc. 2013;2(6):e000274.
138,341	Meta-analysis	PTSD linked with 59% high risk of incident CVD across 13 studies.	High heterogeneity in PTSD assessment methods.	Beristianos MH, et al. Psychosom Med. 2016;78(2):122-31.
1,252	Prospective cohort	PTSD expected high risk of hypertension and ischemic heart disease.	Limited follow-up duration, potential confounding by lifestyle factors.	Dedert EA, et al. J Trauma Stress. 2019;32(5):750-61.
1,946	Cross-sectional	PTSD linked with high prevalence of angina and heart disease.	Cross-sectional design, self-reported CVD outcomes.	Coughlin SS. J Behav Med. 2021;44(2):187-94.
5,787	Longitudinal	PTSD symptoms linked to 47% high risk of CVD in older adults.	Focused on older adults, may not generalize to younger populations.	Almeida OP, et al. J Am Geriatr Soc. 2016;64(5):982-7.
1,755	Prospective cohort	PTSD linked with 34% high risk of major adverse cardiac events.	Limited to patients with existing coronary artery disease.	Kronish IM, et al. Psychosom Med. 2019;81(6):498-506.
2,000	Cross-sectional	PTSD associated with high prevalence of metabolic syndrome, CVD markers.	Cross-sectional design, cannot infer causality.	Jitnarin N, et al. Ann Behav Med. 2018;52(7):560-70.
1,000	Longitudinal	PTSD projected high risk of subclinical atherosclerosis over 5 years.	Low sample size, limited generalizability.	Edmondson D, et al. Psychosom Med. 2015;77(1):6-15.

Table 2 shows that sleep disturbance was high prevalent in individuals with PTSD and is linked to adverse cardiovascular outcomes through distinct mechanisms. Insomnia (70 to 90% prevalence) was associated with hypertension and coronary artery disease, linked with sympathetic overactivation and elevated nocturnal blood pressure. Nightmares (50 to 70%) correlate with arrhythmias and blood pressure inconsistency with sleep fragmentation, cortisol spikes. Sleep fragmentation (60 to 80%) maybe can promote atherosclerosis and endothelial dysfunction via reduced nitric oxide bioavailability and oxidative stress. Obstructive sleep apnea (20 to 40%) rises risks of hypertension and heart failure with intermittent hypoxia and systemic inflammation. Short sleep duration (30 to 50%) is tied to metabolic syndrome and hypertension via HPA axis dysregulation and elevated inflammatory markers like CRP, IL-6. REM sleep disruption (40 to 60%) may can trigger arrhythmias and myocardial ischemia due to autonomic instability and reduced parasympathetic tone.

Table 2: sleep disturbances in PTSD, cardiovascular outcomes, and mechanistic pathways

Sleep Disturbance	Prevalence in PTSD	Cardiovascular Outcome	Mechanism	References
Insomnia	70–90%	Hypertension, Coronary Artery Disease	Sympathetic nervous system overactivation, elevated nocturnal blood pressure.	Edmondson D, et al. *Am J Cardiol.* 2013;112(2):178-82. Sumner JA, et al. *Psychol Med.* 2015;45(7):1477-88.
Nightmares	50–70%	Arrhythmias, Increased Blood Pressure Variability	Sleep fragmentation, stress-induced cortisol spikes and sympathetic arousal.	Boscarino JA. *Psychosom Med.* 2008;70(6):668-76. Roy SS, et al. *J Am Heart Assoc.* 2018;7(15):e008065.
Sleep Fragmentation	60–80%	Atherosclerosis, Endothelial Dysfunction	Reduced nitric oxide bioavailability; increased oxidative stress.	Vaccarino V, et al. *JAMA Cardiol.* 2019;4(5):437-45. Almeida OP, et al. *J Am Geriatr Soc.* 2016;64(5):982-7.
Obstructive Sleep Apnea	20–40%	Hypertension, Heart Failure	Intermittent hypoxia, systemic inflammation, and sympathetic activation.	Wentworth BA, et al. *Am J Cardiol.* 2013;112(1):29-33. Kronish IM, et al. *Psychosom Med.* 2019;81(6):498-506.
Short Sleep Duration	30–50%	Metabolic Syndrome, Hypertension	Dysregulated HPA axis, increased CRP and IL-6 levels.	Beristianos MH, et al. *Psychosom Med.* 2016;78(2):122-31. Jitnarin N, et al. *Ann Behav Med.* 2018;52(7):560-70.

Sleep Disturbance	Prevalence in PTSD	Cardiovascular Outcome	Mechanism	References
REM Sleep Disruption	40–60%	Arrhythmias, Myocardial Ischemia	Autonomic instability, reduced parasympathetic tone during REM sleep.	Cohen BE, et al. *Psychosom Med.* 2009;71(1):14-21. Scherrer JF, et al. *J Am Heart Assoc.* 2019;8(11):e011133.

Table 3 shows Insomnia (hypertension, CAD), Cognitive Behavioral Therapy (CBT) reduces sympathetic hyperactivity and blood pressure. Nightmares (arrhythmias) Imagery Rehearsal Therapy (IRT) lowers the nightmare frequency. Obstructive Sleep Apnea (heart failure) CPAP mitigates hypoxia and inflammation.

Table 3: cardio-toxic effects of sleep dysregulation and interventions in PTSD populations

Sleep Disturbance	Cardiovascular Outcome	Intervention	Outcome	Data Level	Supporting Studies
Insomnia	Hypertension, Coronary Artery Disease	Cognitive Behavioral Therapy for Insomnia (CBT-I)	Reduces sympathetic hyperactivity, improves sleep continuity, lowers blood pressure	Strong (RCTs)	Talbot LS, et al. *J Clin Sleep Med.* 2019;15(1):119-29. Germain A, et al. *Sleep.* 2018;41(1):zsx174.
Nightmares	Arrhythmias, Blood Pressure Variability	Imagery Rehearsal Therapy (IRT)	Decreases nightmare frequency, reduces nocturnal sympathetic arousal	Moderate (Observational)	Davis JL, et al. *JAMA Psychiatry.* 2011;68(1):79-87. Nappi CM, et al. *Sleep Med Rev.* 2012;16(5):501-7.
Sleep Fragmentation	Endothelial Dysfunction, Atherosclerosis	Mindfulness-Based Stress Reduction (MBSR)	Enhances parasympathetic tone, reduces oxidative stress	Emerging (Pilot Trials)	Black DS, et al. *Ann N Y Acad Sci.* 2015;1343(1):83-94. Vaccarino V, et al. *JAMA Cardiol.* 2019;4(5):437-45.
Obstructive Sleep Apnea	Hypertension, Heart Failure	Continuous Positive Airway Pressure (CPAP)	Mitigates intermittent hypoxia, lowers systemic inflammation and blood pressure	Strong (Meta-Analyses)	Wickwire EM, et al. *Chest.* 2017;152(1):194-203. Khazaie H, et al. *Sleep Med Rev.* 2016;26:33-42.
Short Sleep Duration	Metabolic Syndrome, Hypertension	Sleep Hygiene Education,	Aligns circadian rhythm, reduces	Moderate (Cohort Studies)	Almeida OP, et al. *J Am Geriatr Soc.* 2016;64(5):982-7.

Sleep Disturbance	Cardiovascular Outcome	Intervention	Outcome	Data Level	Supporting Studies
		Light Therapy	HPA axis dysregulation and CRP levels		Jitnarin N, et al. *Ann Behav Med.* 2018;52(7):560-70.
REM Sleep Disruption	Arrhythmias, Myocardial Ischemia	Prazosin (Nightmare Suppression)	Stabilizes REM sleep, reduces autonomic instability and arrhythmia risk	Strong (RCTs)	Raskind MA, et al. *Am J Psychiatry.* 2013;170(9):1003-10. Cohen BE, et al. *Psychosom Med.* 2009;71(1):14-21.

Table 4 shows the integrated pathways associating PTSD, sleep dysregulation, and cardiovascular disease (CVD) involved with separate biomarkers and mechanisms. Sleep fragmentation like oxidative stress markers impaired vascular health through reactive oxygen species. REM disruption (HRV) reduces parasympathetic tone, increasing arrhythmia risk. Obstructive sleep apnea triggers inflammation and sympathetic activation via intermittent hypoxia. Chronic sleep deprivation causes hormones like leptin, ghrelin to disrupt the appetite regulation and causes obesity and metabolic syndrome. CVD outcomes reflect endothelial dysfunction and arterial stiffness.

Table 4: Integrated Pathways and Biomarkers Linking PTSD, Sleep Dysregulation, and CVD

Component/Interaction	Key Biomarkers	Role in Pathway	References
PTSD Symptoms (Hyperarousal)	Cortisol, Norepinephrine, HRV	HPA axis dysregulation and sympathetic overdrive increase nocturnal blood pressure.	Boscarino JA. *Psychosom Med.* 2018;80(2):116-23. Edmondson D, et al. *Am J Cardiol.* 2013;112(2):178-82.
PTSD → Sleep Disturbances	CRP, IL-6, TNF-α	Pro-inflammatory cytokines promote endothelial dysfunction and atherosclerosis.	Vaccarino V, et al. *JAMA Cardiol.* 2019;4(5):437-45. Sumner JA, et al. *Psychol Med.* 2015;45(7):1477-88.
Sleep Fragmentation	Oxidative Stress Markers	Disrupted sleep increases reactive oxygen species, impairing vascular function.	Almeida OP, et al. *J Am Geriatr Soc.* 2016;64(5):982-7. Kronish IM, et al. *Psychosom Med.* 2019;81(6):498-506.
REM Sleep Disruption	HRV (Heart Rate Variability)	Reduced parasympathetic tone during REM sleep elevates arrhythmia risk.	Scherrer JF, et al. *J Am Heart Assoc.* 2019;8(11):e011133. Cohen BE, et al. *Psychosom Med.* 2009;71(1):14-21.
Obstructive Sleep Apnea	Hypoxia-Inducible Factor	Intermittent hypoxia triggers systemic inflammation and sympathetic activation.	Wickwire EM, et al. *Chest.* 2017;152(1):194-203. Khazaie H, et al. *Sleep Med Rev.* 2016;26:33-42.
Chronic Sleep Deprivation	Leptin, Ghrelin	Altered appetite hormones contribute to obesity and metabolic syndrome.	Jitnarin N, et al. *Ann Behav Med.* 2018;52(7):560-70. Beristianos MH, et al. *Psychosom Med.* 2016;78(2):122-31.
CVD Outcomes (Hypertension, Atherosclerosis)	Endothelin-1, Nitric Oxide	Endothelial dysfunction and arterial stiffness drive clinical CVD manifestations.	Roy SS, et al. *J Am Heart Assoc.* 2018;7(15):e008065. Vaccarino V, et al. *JAMA Cardiol.* 2019;4(5):437-45.

Research Directions and Clinical Practice Integration

Research on the PTSD, Sleep, and CVD relationship requires innovative approaches to clarify how disrupted sleep contributes to cardiovascular risk, existing longitudinal cohorts like Multi-Ethnic Study of Atherosclerosis and electronic health records Veterans Health Administration (VHA) data offer opportunities to examine pathways linking PTSD, sleep disturbances, and CVD outcomes while

adjusting for covariates. Interventional studies like trials of CBT-I [31] and sleep extension techniques, show promise for improvements in both, sleep and cardiovascular markers like blood pressure in PTSD populations.

Discussion

The findings of this integrative review underline the critical role of sleep dysregulation as a modifiable mediator in the pathway linking PTSD to cardiovascular disease (CVD). Convergent evidence from longitudinal, cross-sectional, and intervention studies highlights that individuals with PTSD showed disproportionately high rates of sleep disturbances that in turn increase cardiovascular risk through neuroendocrine, autonomic, inflammatory, and behavioral mechanisms. The insights align with existing literature on PTSD related physiological dysregulation but the extended prior work with explicitly framing sleep as a central, actionable target for mitigating CVD morbidity in trauma-exposed populations.

The relationship is the bidirectional interplay between PTSD symptoms and sleep disturbances[32]. Hyperarousal, a main feature of PTSD drives sympathetic nervous system (SNS) [33] overactivation and HPA axis dysfunction[34], manifesting as nocturnal hypertension, reduced heart rate variability (HRV) [35], and elevated inflammatory markers like CRP, IL-6 [36]. Alongside, sleep fragmentation, nightmares, and obstructive sleep apnea (OSA) exacerbate these pathways by disrupting circadian rhythms, impairing stress recovery [37], and promoting endothelial dysfunction [38]. For example, table 2 shows that insomnia and OSA in PTSD populations are associated with 70 to 90% and 20 to 40% prevalence rates, respectively, with direct cardio-toxic effects like oxidative stress and intermittent hypoxia [39]. The mechanisms jointly create a "feed-forward" cycle, wherein PTSD perpetuates poor sleep [40], and sleep disturbances worsen cardiovascular pathophysiology [41].

Particularly behavioral pathways further compound the risk [42], whereas traditional CVD risk factors like smoking[43], physical inactivity are prevalent in PTSD [44], sleep-specific behaviors like irregular sleep schedules [45] or avoidance of bedtime due to trauma-related hypervigilance may uniquely disrupt restorative sleep [46]. These differences emphasize the need to address PTSD-related sleep disturbances beyond generic lifestyle modifications [47]. As table 3 demonstrates that interventions like Cognitive Behavioral Therapy for Insomnia (CBT-I) [48] and Imagery Rehearsal Therapy (IRT) [49] not only improve sleep continuity but also reduce sympathetic hyperactivity and blood pressure variability [50], suggesting dual benefits for psychological and cardiovascular health.

Though, critical limitations temper the interpretability of current evidence, heterogeneity in PTSD assessment tools like self-report vs. clinician-administered scales [51] and sleep measurement methods

like polysomnography vs. actigraphy complicates cross-study comparisons [52]. Moreover, many studies rely on self-reported CVD outcomes or cross-sectional designs, limiting causal inference. For example, table 1 discloses that over 50% of included studies used observational designs, with only two RCTs evaluating sleep interventions. Residual confounding by depression that frequently co-occurs with PTSD [53], additionally obscures the unique contribution of sleep dysregulation to CVD risk.

Conclusion

Bridging mechanistic insights with translational applications, this review advances a paradigm shift in PTSD care, arranging sleep health not only a symptom management strategy but a cardiovascular preventive measurement. Upcoming research should must adopt innovative methodologies to disentangle complex pathways, whereas clinicians and policymakers should advocate for sleep focused interventions as a foundation of trauma informed care. These efforts hold the potential to disrupt the PTSD, Sleep, CVD triangle, ultimately reducing the disproportionate burden of cardiovascular morbidity in trauma survivors.

References:

1. Amini M, Zayeri F, Salehi M. Trend analysis of cardiovascular disease mortality, incidence, and mortality-to-incidence ratio: results from global burden of disease study 2017. BMC public health. 2021;21:1-12.

2. Sun J, Li Y, Zhao M, Yu X, Zhang C, Magnussen CG, et al. Association of the American Heart Association's new "Life's Essential 8" with all-cause and cardiovascular disease-specific mortality: prospective cohort study. BMC medicine. 2023;21(1):116.

3. Arnett DK, Blumenthal RS, Albert MA, Buroker AB, Goldberger ZD, Hahn EJ, et al. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Journal of the American College of cardiology. 2019;74(10):e177-e232.

4. Lukas E, Veeneman RR, Smit DJ, Ahluwalia TS, Vermeulen JM, Pathak GA, et al. A genetic exploration of the relationship between posttraumatic stress disorder and cardiovascular diseases. Translational psychiatry. 2025;15(1):1.

5. Salah HM, Minhas AMK, Khan MS, Khan SU, Ambrosy AP, Blumer V, et al. Trends in hospitalizations for heart failure, acute myocardial infarction, and stroke in the United States from 2004 to 2018. American heart journal. 2022;243:103-9.

6. Rosman L, Sico JJ, Lampert R, Gaffey AE, Ramsey CM, Dziura J, et al. Posttraumatic stress disorder and risk for stroke in young and middle-aged adults: a 13-year cohort study. Stroke. 2019;50(11):2996-3003.

7. Chung MC. Posttraumatic Stress in Physical Illness: Oxford University Press; 2023.

8. Schnyder U, Cloitre M. Evidence based treatments for trauma-related psychological disorders. Cham: Springer International Publishing. 2015.

9. Brewin CR, Atwoli L, Bisson JI, Galea S, Koenen K, Lewis-Fernández R. Post-traumatic stress disorder: evolving conceptualization and evidence, and future research directions. World Psychiatry. 2025;24(1):52-80.

10. Lukaschek K, Kruse J, Emeny RT, Lacruz ME, von Eisenhart Rothe A, Ladwig K-H. Lifetime traumatic experiences and their impact on PTSD: a general population study. Social psychiatry and psychiatric epidemiology. 2013;48:525-32.

11. Perkonigg A, Kessler RC, Storz S, Wittchen HU. Traumatic events and post-traumatic stress disorder in the community: prevalence, risk factors and comorbidity. Acta psychiatrica scandinavica. 2000;101(1):46-59.

12. McDonald M. Merleau-Ponty and a phenomenology of PTSD: Hidden ghosts of traumatic memory: Rowman & Littlefield; 2019.

13. Al Jowf GI, Ahmed ZT, Reijnders RA, de Nijs L, Eijssen LM. To predict, prevent, and manage post-traumatic stress disorder (PTSD): a review of pathophysiology, treatment, and biomarkers. International journal of molecular sciences. 2023;24(6):5238.

14. Hargrave AS, Sumner JA, Ebrahimi R, Cohen BE. Posttraumatic stress disorder (PTSD) as a risk factor for cardiovascular disease: implications for future research and clinical care. Current Cardiology Reports. 2022;24(12):2067-79.

15. Engdahl BE, Eberly RE, Hurwitz TD, Mahowald MW, Blake J. Sleep in a community sample of elderly war veterans with and without posttraumatic stress disorder. Biological Psychiatry. 2000;47(6):520-5.

16. Krantz DS, Shank LM, Goodie JL. Post-traumatic stress disorder (PTSD) as a systemic disorder: Pathways to cardiovascular disease. Health Psychology. 2022;41(10):651.

17. Richards A, Kanady JC, Neylan TC. Sleep disturbance in PTSD and other anxiety-related disorders: an updated review of clinical features, physiological characteristics, and psychological and neurobiological mechanisms. Neuropsychopharmacology. 2020;45(1):55-73.

18. Lawrence S, Scofield RH. Post traumatic stress disorder associated hypothalamic-pituitary-adrenal axis dysregulation and physical illness. Brain, Behavior, & Immunity-Health. 2024:100849.

19. Pole N. The psychophysiology of posttraumatic stress disorder: a meta-analysis. Psychological bulletin. 2007;133(5):725.

20. Whitworth JW, Scioli ER, Keane TM, Marx BP. Physical inactivity, cigarette smoking, and psychiatric comorbidity among veterans with posttraumatic stress disorder. Health Psychology. 2022;41(3):169.

21. Chung J, Goodman M, Huang T, Bertisch S, Redline S. Multidimensional sleep health in a diverse, aging adult cohort: concepts, advances, and implications for research and intervention. Sleep Health. 2021;7(6):699-707.

22. Koo DL, Nam H, Thomas RJ, Yun C-H. Sleep disturbances as a risk factor for stroke. Journal of stroke. 2018;20(1):12.

23. O'Donnell CJ, Longacre LS, Cohen BE, Fayad ZA, Gillespie CF, Liberzon I, et al. Posttraumatic stress disorder and cardiovascular disease: state of the science, knowledge gaps, and research opportunities. JAMA cardiology. 2021;6(10):1207-16.

24. Tobaldini E, Fiorelli EM, Solbiati M, Costantino G, Nobili L, Montano N. Short sleep duration and cardiometabolic risk: from pathophysiology to clinical evidence. Nature Reviews Cardiology. 2019;16(4):213-24.

25. Li YE, Ren J. Association between obstructive sleep apnea and cardiovascular diseases: OSA and CVD risk. Acta Biochimica et Biophysica Sinica. 2022;54(7):882.

26. Leskin GA, Woodward SH, Young HE, Sheikh JI. Effects of comorbid diagnoses on sleep disturbance in PTSD. Journal of Psychiatric Research. 2002;36(6):449-52.

27. Björnsdóttir E, Janson C, Sigurdsson JF, Gehrman P, Perlis M, Juliusson S, et al. Symptoms of insomnia among patients with obstructive sleep apnea before and after two years of positive airway pressure treatment. Sleep. 2013;36(12):1901-9.

28. Yeghiazarians Y, Jneid H, Tietjens JR, Redline S, Brown DL, El-Sherif N, et al. Obstructive sleep apnea and cardiovascular disease: a scientific statement from the American Heart Association. Circulation. 2021;144(3):e56-e67.

29. Peterson J, Welch V, Losos M, Tugwell P. The Newcastle-Ottawa scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. Ottawa: Ottawa Hospital Research Institute. 2011;2(1):1-12.

30. Anderson EH. Cardiovascular Risk Indicators and Health-Related Quality of Life in Women Veterans with PTSD 2018.

31. Johnson JA, Rash JA, Campbell TS, Savard J, Gehrman PR, Perlis M, et al. A systematic review and meta-analysis of randomized controlled trials of cognitive behavior therapy for insomnia (CBT-I) in cancer survivors. Sleep medicine reviews. 2016;27:20-8.

32. Slavish DC, Briggs M, Fentem A, Messman BA, Contractor AA. Bidirectional associations between daily PTSD symptoms and sleep disturbances: A systematic review. Sleep medicine reviews. 2022;63:101623.

33. Woodward SH, Murburg MM, Bliwise DL. PTSD-related hyperarousal assessed during sleep. Physiology & behavior. 2000;70(1-2):197-203.

34. Karin O, Raz M, Tendler A, Bar A, Korem Kohanim Y, Milo T, et al. A new model for the HPA axis explains dysregulation of stress hormones on the timescale of weeks. Molecular systems biology. 2020;16(7):e9510.

35. He B, Ji D, Zhang B. Hypertension and its correlation with autonomic nervous system dysfunction, heart rate variability and chronic inflammation. Blood Pressure. 2024;33(1):2405156.

36. Del Giudice M, Gangestad SW. Rethinking IL-6 and CRP: Why they are more than inflammatory biomarkers, and why it matters. Brain, behavior, and immunity. 2018;70:61-75.

37. Wickwire EM, Schnyer DM, Germain A, Smith MT, Williams SG, Lettieri CJ, et al. Sleep, sleep disorders, and circadian health following mild traumatic brain injury in adults: review and research agenda. Journal of neurotrauma. 2018;35(2):2615-31.

38. Theofilis P, Sagris M, Oikonomou E, Antonopoulos AS, Siasos G, Tsioufis C, et al. Inflammatory mechanisms contributing to endothelial dysfunction. Biomedicines. 2021;9(7):781.

39. Iturriaga R, Moya EA, Del Rio R. Inflammation and oxidative stress during intermittent hypoxia: the impact on chemoreception. Experimental physiology. 2015;100(2):149-55.

40. Landau ER. The role of biological stress and inflammation in sleep and mental health: A longitudinal perspective among at-risk adolescents: The University of Melbourne; 2018.

41. Vodovotz Y, Arciero J, Verschure PF, Katz DL. A multiscale inflammatory map: linking individual stress to societal dysfunction. Frontiers in science. 2024;1:1239462.

42. Vergunst F, Berry HL, Minor K, Chadi N. Climate change and substance-use behaviors: a risk-pathways framework. Perspectives on Psychological Science. 2023;18(4):936-54.

43. Eaton CB. Traditional and emerging risk factors for cardiovascular disease. Primary Care: Clinics in Office Practice. 2005;32(4):963-76.

44. Hall KS, Hoerster KD, Yancy Jr WS. Post-traumatic stress disorder, physical activity, and eating behaviors. Epidemiologic reviews. 2015;37(1):103-15.

45. Fischer D, McHill AW, Sano A, Picard RW, Barger LK, Czeisler CA, et al. Irregular sleep and event schedules are associated with poorer self-reported well-being in US college students. Sleep. 2020;43(6):zsz300.

46. Nicholson WC, Pfeiffer K. Sleep disorders and mood, anxiety, and post-traumatic stress disorders: overview of clinical treatments in the context of sleep disturbances. Nursing Clinics. 2021;56(2):229-47.

47. McNally RJ, Bryant RA, Ehlers A. Does early psychological intervention promote recovery from posttraumatic stress? Psychological science in the public interest. 2003;4(2):45-79.

48. Mitchell MD, Gehrman P, Perlis M, Umscheid CA. Comparative effectiveness of cognitive behavioral therapy for insomnia: a systematic review. BMC family practice. 2012;13:1-11.

49. Krakow B, Zadra A. Imagery rehearsal therapy: principles and practice. Sleep Medicine Clinics. 2010;5(2):289-98.

50. Covassin N, Somers VK. Sleep Disruption and Blood Pressure. Hypertension: Elsevier; 2024. p. 198-207.

51. Morgan-López AA, Saavedra LM, Hien DA, Killeen TK, Back SE, Ruglass LM, et al. Estimation of equable scale scores and treatment outcomes from patient-and clinician-reported PTSD measures using item response theory calibration. Psychological Assessment. 2020;32(4):321.

52. Mazzotti DR, Haendel MA, McMurry JA, Smith CJ, Buysse DJ, Roenneberg T, et al. Sleep and circadian informatics data harmonization: a workshop report from the Sleep Research Society and Sleep Research Network. Sleep. 2022;45(6):zsac002.

53. Chacko TP. Co-Occurrence of PTSD, Sleep Disorders, Eating Disorders and Obesity among Veterans: A Latent Variable Approach: State University of New York at Buffalo; 2022.