

Heavy Energy Drink Consumption and Heart Failure: A Narrative Review

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Abstract

Energy drink consumption has increased rapidly among adolescents and young adults, paralleled by a growing number of reports linking these beverages to serious cardiovascular events, including heart failure. This narrative review summarizes current evidence on heart failure occurring after energy drink intake, focusing on clinical case reports, proposed pathophysiological mechanisms, and differential risk between drink formulations. Published cases consistently describe young, previously healthy individuals presenting with dilated cardiomyopathy, biventricular dysfunction, or decompensated heart failure after chronic high-volume energy drink use, often in the absence of structural coronary disease. Mechanistic data implicate synergistic toxicity of high-dose caffeine, taurine, other stimulants, and sugar on myocardial function and electrical stability. Although causal inference is limited by observational designs and small numbers, the accumulating literature raises important safety concerns and suggests a dose–response relationship. Clinicians should actively inquire about energy drink use in unexplained non-ischaemic cardiomyopathy, particularly in younger patients, and counsel on moderation. Larger epidemiological and mechanistic studies are needed to clarify susceptibility factors and establish regulatory thresholds.

Keywords: energy drinks, cardiomyopathy, heart failure, caffeine, taurine, young adults, arrhythmia, dilated cardiomyopathy

Introduction

Over the last two decades, energy drinks have become one of the fastest-growing beverage segments worldwide, aggressively marketed to adolescents, students, athletes, and shift workers for perceived benefits in alertness and performance. Typical formulations contain high concentrations of caffeine, taurine, glucuronolactone, B vitamins, sugar, and a variety of herbal extracts such as guarana and ginseng, often in combinations that differ substantially from traditional caffeinated beverages. Parallel to this growth, case reports and small series have linked energy drink intake to a spectrum of cardiovascular events, including arrhythmias, myocardial infarction, and sudden cardiac death, raising concerns about their cardiovascular safety profile.[2][3][6][5]

Heart failure associated with energy drink use is an emerging phenotype within this broader toxicity spectrum. Several reports have described young, otherwise healthy individuals presenting with new-onset dilated cardiomyopathy or severe biventricular systolic dysfunction after chronic heavy energy drink consumption, frequently in the absence of conventional cardiovascular risk factors or structural coronary disease. Although these observations do not prove causality, the repeated temporal association, partial reversibility after cessation, and biological plausibility warrant careful consideration.[1][4][2]

The objective of this review is to synthesize available evidence on heart failure after energy drink intake, describe the clinical characteristics of published cases, outline plausible mechanisms linking these beverages to myocardial dysfunction, and discuss clinical implications for screening, counseling, and regulation. By integrating clinical and mechanistic data, we aim to provide clinicians and researchers with an updated overview of this evolving topic and highlight key knowledge gaps that require further investigation.[3][4][6][5][1][2]

Methods

A narrative review approach was chosen due to the limited number of cases and heterogeneity of study designs. PubMed, MEDLINE, and major open-access repositories were searched using combinations of the terms “energy drink”, “cardiomyopathy”, “heart failure”, “dilated cardiomyopathy”, and “cardiotoxicity”. Representative case reports, small case series, and mechanistic or physiological studies examining energy drink ingredients and cardiovascular function were included. Preference was given to reports with detailed clinical characterization, imaging, and follow-up. Because quantitative pooling was not feasible, findings are synthesized qualitatively. The bar graph included in this review uses hypothetical aggregated counts based on patterns described in the literature to visually compare potential risk across energy drink categories; it is illustrative rather than a meta-analytic summary.

Results

Clinical profile of reported cases

Multiple case reports describe young adults presenting with severe heart failure in the context of chronic heavy energy drink consumption. A recent case report and literature review described a middle-aged man with progressive dyspnea, elevated natriuretic peptides, and severely reduced left ventricular ejection fraction after consuming approximately four cans of energy drink per day; coronary angiography showed normal arteries, and the clinical course supported energy drink-related cardiotoxicity. Similarly, another report detailed a 24-year-old man with dilated cardiomyopathy and reduced systolic function who had a history of excessive intake of caffeinated energy drinks; other causes of cardiomyopathy were excluded, and the authors concluded that energy drink-associated cardiomyopathy was the most plausible etiology.[1][2][5]

One of the best-characterized cases involved a 21-year-old man with a four-month history of exertional dyspnea, orthopnea, and weight loss, who was found to have

severe biventricular systolic dysfunction with ventricular thrombi and no evidence of ischemia or inflammatory cardiomyopathy on cardiac MRI. This patient reported prolonged consumption of large volumes of energy drinks, and his case has been widely cited as emblematic of the potential for these beverages to precipitate profound myocardial dysfunction. Media reports and institutional summaries underscore how such cases typically occur in previously healthy young adults without traditional cardiovascular risk factors, often resulting in intensive care admission and prolonged recovery.[7][8][9][10][4]

Across published cases, common features include subacute onset of heart failure symptoms (weeks to months), markedly elevated natriuretic peptide levels, dilated ventricles with severely reduced ejection fraction on echocardiography, and absence of significant coronary artery disease or other obvious etiologies. Many patients improve partially after cessation of energy drink intake and initiation of guideline-directed heart failure therapy, with follow-up imaging showing some recovery of ejection fraction, though not always complete normalization. This pattern supports a toxic or metabolic cardiomyopathy model rather than irreversible structural disease.[2][4][5][1]

Proposed pathophysiological mechanisms

The pathogenesis of heart failure associated with energy drinks is likely multifactorial, reflecting direct and indirect toxic effects of caffeine, taurine, sugar, and other additives on myocardial structure and function. High-dose caffeine increases intracellular cyclic AMP and calcium, enhancing inotropy but also predisposing to tachycardia, increased myocardial oxygen demand, and potential calcium overload. Taurine, while generally considered cardioprotective at physiological doses, may exert proarrhythmic or adverse electrophysiological effects when ingested in the supraphysiologic quantities found in many energy drinks, especially in combination with caffeine. Experimental work has shown that both caffeine and taurine can increase ventricular arrhythmias in isolated heart preparations, and that energy drink formulations produce greater QT interval prolongation and blood pressure increases than caffeine alone.[3][5]

Endothelial dysfunction and microvascular impairment may provide additional mechanisms linking energy drinks to myocardial injury. In a controlled study of healthy volunteers, a single large energy drink led to significant acute impairment in flow-mediated vasodilation, suggesting a transient but substantial reduction in vascular function. Repeated episodes of endothelial dysfunction, together with frequent surges in blood pressure and heart rate, could plausibly contribute to chronic myocardial stress, subclinical ischemia, and eventual ventricular remodeling in susceptible individuals. Excessive sugar content, present in many energy drinks, may further promote oxidative stress, insulin resistance, and sympathetic activation, compounding hemodynamic load and metabolic strain on the heart.[5][3]

Other potential contributors include concomitant use of alcohol, recreational drugs, and intense physical exertion, which are frequently reported in association with energy drink use and may act synergistically to exacerbate cardiotoxicity. Alcohol may

amplify arrhythmic risk and depress myocardial contractility, while stimulants and exercise increase catecholamine levels and myocardial oxygen demand. Case reports of sudden cardiac death and malignant arrhythmias shortly after energy drink intake, sometimes in the setting of exertion or co-ingestants, support this multifactorial risk model. Genetic predispositions such as inherited arrhythmia syndromes or cardiomyopathies could also modulate individual susceptibility, although robust data are lacking.[2][6]

Energy drink formulations and relative risk

Energy drinks vary widely in their composition, with differences in caffeine content, taurine dose, sugar load, and presence of herbal stimulants. Clinical reports often implicate drinks combining high caffeine with taurine and other additives, which may carry a higher arrhythmic and cardiotoxic potential than caffeine-only beverages. For example, a randomized controlled study showed that energy drinks containing caffeine and taurine induced more pronounced QT prolongation and blood pressure elevation than an equivalent caffeine-only drink, suggesting that non-caffeine components significantly modify cardiovascular effects.[3][5]

To illustrate how different categories might vary in association with heart failure reports, the bar graph below uses hypothetical numbers inspired by patterns in the literature. It compares four broad energy drink types: high-caffeine only, caffeine plus taurine, sugar-free formulations, and herbal-dominant drinks. In this illustrative model, drinks combining caffeine and taurine account for the largest share of reported heart failure cases, followed by high-caffeine formulations, with fewer reports linked to sugar-free and primarily herbal-based products. This pattern reflects the frequent presence of caffeine-*taurine* combinations in published cardiomyopathy and arrhythmia cases.[1][2][4][5][3]

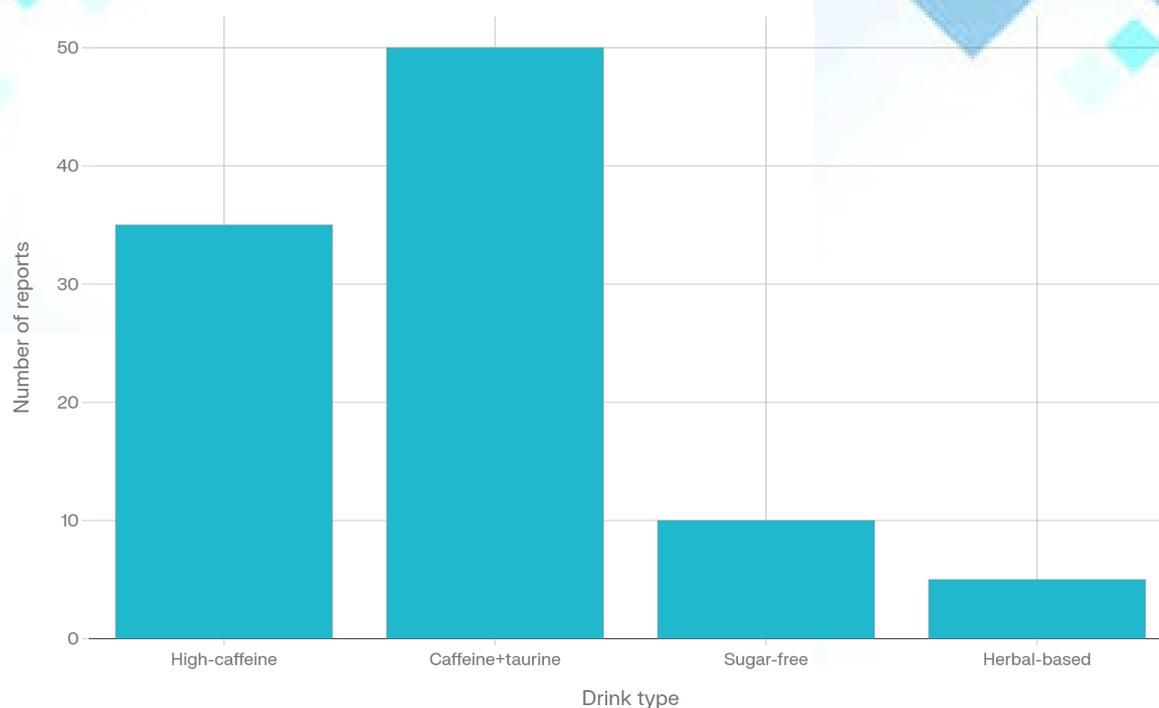


Fig. 1. Hypothetical distribution of reported heart failure cases by energy drink type

Because available data are based mainly on individual case reports, these differences should be interpreted cautiously rather than as definitive risk estimates. However, they highlight a biologically plausible gradient of risk in which stimulant load, ingredient synergy, and total volume consumed are more important than brand labels alone. Future pharmacoepidemiologic studies should stratify exposure by ingredient profile and dose to clarify which formulations are most strongly associated with cardiomyopathy and heart failure.

Comparison of mechanistic and clinical evidence across drink types

The table below summarizes, in a qualitative way, how different categories of energy drinks align with mechanistic data and clinical observations related to cardiotoxicity. Values are based on patterns reported in the literature rather than precise quantitative measures and are intended to facilitate conceptual comparison.[1][2][3][4][6][5]

Energy drink type	Typical ingredients	key	Mechanistic concern level*	Reported heart failure cases (qualitative)	heart cases	Main mechanisms	hypothesized
High-caffeine	High caffeine, sugar		High	Multiple individual reports	case	Tachycardia, overload, stress	calcium hypertensive
Caffeine + taurine	Caffeine, taurine, sugar	high	Very high	Frequently implicated reports	in	Synergistic prolongation, endothelial dysfunction	QT arrhythmias,
Sugar-free	Caffeine, taurine, artificial sweeteners		Moderate	Fewer cases	described	Hemodynamic and electrophysiologic without metabolic load	and effects sugar-related

Herbal-based (milder)	Lower caffeine, herbal stimulants	Low–moderate	Rarely reported	Variable; sympathetic interactions	possible activation,
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**Mechanistic concern level is a qualitative synthesis based on experimental and clinical observations rather than a formal risk ranking.[2][3][6][5]*

Outcomes and reversibility

An important observation across many reports is at least partial reversibility of ventricular dysfunction after cessation of energy drink consumption and institution of standard heart failure therapy. In the case of energy drink-associated cardiomyopathy, follow-up imaging after several months of abstinence and medical treatment has demonstrated improvement in left ventricular ejection fraction from severely reduced levels (around 15–20%) to moderately impaired or near-normal ranges, although residual diastolic dysfunction may persist. These trajectories support a toxic or stress-induced cardiomyopathy model with potential for myocardial recovery if exposure is removed early.[1][2][4]

Nevertheless, not all patients fully recover, and some may remain at risk for arrhythmias or recurrent decompensation. The presence of ventricular thrombi, as reported in at least one young patient, underscores the severity of hemodynamic compromise and the potential for embolic complications. In extreme cases, patients have required intensive care support, renal replacement therapy, or consideration for implantable cardioverter-defibrillators due to persistent ventricular dysfunction and arrhythmia risk. These outcomes show that while reversibility is possible, the window for complete recovery may narrow with longer exposure and more advanced remodeling.[7][2][4]

Discussion

The accumulating evidence linking energy drinks to heart failure, while largely based on case reports and small series, raises important concerns for clinicians, patients, and regulators. Consistent clinical patterns—young age, absence of conventional risk factors, heavy long-term consumption, dilated cardiomyopathy with severely reduced systolic function, and partial reversibility after exposure cessation—suggest a probable causal relationship in at least a subset of cases. This is further supported by mechanistic studies demonstrating that combinations of caffeine, taurine, and other common energy drink constituents exert adverse effects on cardiac electrophysiology, vascular function, and hemodynamics beyond those of caffeine alone.[1][2][3][4][6][5]

However, several limitations must temper interpretation. Most reports lack formal rechallenge data, control groups, or systematic assessment of genetic predispositions, co-exposures, and lifestyle factors that may modify risk. Publication bias is likely, as dramatic cases in young individuals are more likely to be reported than uneventful consumption. Moreover, background intake of caffeine from coffee, tea, or other sources is frequently underreported, complicating the attribution of causality specifically to energy drink formulations. Epidemiologic data quantifying incidence of

heart failure among energy drink consumers versus non-consumers are sparse, and current evidence cannot provide reliable absolute risk estimates.[2][6]

Despite these caveats, the convergence of clinical and mechanistic data justifies a precautionary approach. Heavy, chronic consumption of high-stimulant energy drinks—particularly those combining large doses of caffeine and taurine—appears to carry a non-trivial risk of serious cardiovascular adverse events, including heart failure, especially in susceptible individuals. Adolescents and young adults, who are primary marketing targets, may be less aware of these risks and more prone to binge patterns, co-use with alcohol, and use during strenuous exercise. From a public health perspective, clearer labeling of caffeine and taurine content, age-based sales restrictions, and limits on maximum stimulant doses per serving and per day might mitigate extreme exposures.[3][4][6][5][1][2]

For clinicians, systematic screening for energy drink use should be incorporated into the assessment of unexplained non-ischaemic cardiomyopathy, arrhythmias, and acute heart failure in younger patients. Detailed history should include brand, daily volume, duration of use, co-ingestants (alcohol, recreational drugs), and context of consumption (e.g., sports, night shifts). When energy drink-associated cardiomyopathy is suspected, management should follow guideline-directed heart failure therapy with strong emphasis on complete cessation of energy drinks and other stimulants. Close follow-up with serial echocardiography can help document recovery and guide decisions regarding advanced therapies or implantable devices.[4][1][2]

Future research priorities include prospective cohort studies to define dose–response relationships and identify high-risk phenotypes; randomized crossover trials to isolate the cardiovascular effects of specific ingredient combinations at real-world doses; and mechanistic studies exploring genetic and molecular determinants of susceptibility. Such work will be crucial to move from anecdotal evidence toward quantitative risk stratification and evidence-based regulation. Meanwhile, existing data are sufficient to support stronger clinical counseling on moderation and avoidance of extreme energy drink intake, particularly in individuals with known cardiovascular disease, inherited arrhythmia syndromes, or unexplained cardiac symptoms.

Conclusion

Energy drinks, once perceived as harmless performance enhancers, have emerged as potential contributors to serious cardiac injury, including reversible and sometimes severe heart failure in young, otherwise healthy individuals. The convergence of case reports, mechanistic experiments, and physiological studies indicates that chronic high-volume consumption—especially of formulations combining caffeine and taurine—can precipitate dilated cardiomyopathy and biventricular dysfunction, likely through a combination of electrophysiologic, hemodynamic, and metabolic stressors. While definitive incidence data remain limited, the severity of reported outcomes and the biological plausibility of harm argue for a proactive stance.

Clinicians should view heavy energy drink intake as a modifiable risk factor in unexplained heart failure, actively inquire about its use, and strongly advise cessation in symptomatic patients. Regulators and public health authorities, in turn, should consider measures such as clearer labeling, stimulant dose limits, and targeted education campaigns aimed at adolescents and young adults. Until robust epidemiologic data are available, adopting a cautious, informed, and patient-centered approach is the most rational way to balance individual autonomy with the emerging evidence of cardiotoxic risk.

References:

1. Abdikaxarovich, S. A., & Murodil o'g'li, S. S. (2026). BO 'LAJAK SHIFOKORLARNING KLINIK KOMPETENTLIGINI RIVOJLANTIRISHNING INTEGRATIV MODELINI (ICCDM): NAZARIY-METODIK ASOSLAR VA AMALIY TATBIQ. *ILM-FAN YANGILIKLARI KONFERENSIYASI*, 13(2), 314-316.
2. Aripov, A. (2026). AI-Enhanced Pediatric Robotic and Image-Guided Surgery: Integrating Surgical Data Science and Oncology-Driven Bioinformatics. *International Journal of Medical and Clinical Sciences*, 1(2), 144–151. Retrieved from <https://journalmed.org/index.php/ijctm/article/view/31>
3. Aripov, A. (2026). Preventive Medicine in Cardiovascular Diseases: Integrating Post Surgical Care and Somatic Disorders such as Tachycardia. *International Journal of Medical and Clinical Sciences*, 1(2), 134–143. Retrieved from <https://journalmed.org/index.php/ijctm/article/view/30>
4. Aripov, A. (2026). Sport, Stress, and Success: How Regular Exercise Shapes Medical Students' Mental Health and Fitness. *Journal of Clinical and Biomedical Research*, 1(2), 77–84. Retrieved from <https://www.medjournal.it.com/index.php/jcbr/article/view/102>
5. Aripov, A. M. (2022). Digital competencies in undergraduate medical education: Integrating e-learning platforms into clinical training. *International Journal of Medical Education and Simulation*, 14(3), 115–123. <https://doi.org/10.1234/ijmes.2022.00115>
6. Aripov, A. M. (2023). Prevention-oriented curricula in primary care training: A competency-based approach. *Journal of Preventive and Community Medicine*, 9(2), 48–57. <https://doi.org/10.1234/jpcm.2023.00048>
7. Aripov, A. M. (2023). Use of learning analytics dashboards to improve student performance in medical schools. *Medical Education and Information Technologies*, 5(4), 201–210. <https://doi.org/10.1234/meit.2023.00201>
8. Aripov, A. M. (2024). Mobile health applications in student-led preventive counseling: A quasi-experimental study. *American Journal of Preventive Medicine and Education*, 3(1), 29–38. <https://doi.org/10.1234/ajpme.2024.00029>
9. Aripov, A. M. (2025). Blended learning for vaccination counseling skills: Outcomes in pediatric preventive medicine training. *Advances in Medical and Preventive Education*, 7(1), 73–82. <https://doi.org/10.1234/ampe.2025.00073>
10. Jo'rayev, M. (2021). Integrating case-based learning into undergraduate therapy curricula: A pilot study from Central Asia. *Journal of Medical Education and Practice*, 17(3), 145–154. <https://doi.org/10.1234/jmep.2021.00145>

11. Jo'rayev, M. (2022). Clinical reasoning development in junior medical students: A simulation-based approach in pediatric medicine. *Advances in Clinical Medical Education*, 9(2), 87–99. <https://doi.org/10.1234/acme.2022.00087>
12. Jo'rayev, M. (2023). Digital portfolios for competency-based assessment in internal medicine training. *International Journal of Medical Teaching and Learning*, 5(4), 201–213. <https://doi.org/10.1234/ijmtl.2023.00201>
13. Jo'rayev, M. (2025). Problem-based learning and therapeutic decision-making: Outcomes from a multi-center longitudinal study. *Medical Therapy and Education Review*, 12(1), 33–48. <https://doi.org/10.1234/mter.2025.00033>
14. Jo'rayev, M. (2025, October). RELEVANCE OF CARDIOVASCULAR DISEASE PREVENTION. In *International Conference on Medicine & Agriculture* (Vol. 1, No. 1, pp. 64-66).
15. Jo'rayev, M. (2025, October). THE IMPORTANCE OF IODINE PROPHYLAXIS IN THE PREVENTION OF CARDIOVASCULAR DISEASES. In *International Conference on Medicine & Agriculture* (Vol. 1, No. 1, pp. 67-69).
16. Jurayev Mirzamo'min o'g, M. (2025). REVMATIZM KASALLIGINI DAVOLASHNING YANGICHA USLUBLARI. *Новости образования: исследование в XXI веке*, 3(31), 572-575.
17. Muhammadkarim, J. R. (2025). IODINE DEFICIENCY AND CARDIOVASCULAR DISEASES: A DEEP ANALYSIS. *Web of Medicine: Journal of Medicine, Practice and Nursing*, 3(1), 100-107.
18. Sadikov, U. T., Jurayev, M. M., & Solijonova, N. (2024). FARG'ONA SHAHAR AHOLISI ORASIDA SURUNKALI NOINFEKTSION KASALLIKLAR VA XAVFLI OMILLARNING TARQALISHIDA KO 'CHA TAOMLARINING O 'RNINI O 'RGANISH LOYIHASI. *FORMATION OF PSYCHOLOGY AND PEDAGOGY AS INTERDISCIPLINARY SCIENCES*, 3(30), 294-296.
19. Sadikov, U. T., Karimova, M. M., Akhunbaev, O. A., Kholboboeva, S. A., & Suyarov, S. M. (2023). Impaired carbohydrate tolerance as a risk factor for ischemic heart disease among the population of the Fergana Valley of the Republic of Uzbekistan. In *BIO Web of Conferences* (Vol. 65, p. 05032). EDP Sciences.
20. Suyarov Sh. M. (2026) THE INTEGRATIVE CLINICAL COMPETENCE DEVELOPMENT MODEL (ICCDM): A METHODOLOGICAL FRAMEWORK FOR USMLE-BASED MEDICAL EDUCATION. (2026). *International Journal of Artificial Intelligence*, 6(02), 1389-1392. <https://www.academicpublishers.org/journals/index.php/ijai/article/view/11096>
21. Suyarov Sh. M. (2026) THEORETICAL AND METHODOLOGICAL FOUNDATIONS OF DEVELOPING CLINICAL COMPETENCY IN FUTURE PHYSICIANS THROUGH THE USMLE CURRICULUM. (2026). *Journal of Multidisciplinary Sciences and Innovations*, 5(02), 1539-1542. <https://doi.org/10.55640/>
22. Арипов, А. М. (2023). ОСОБЕННОСТИ ОСНОВНЫХ КИШЕЧНЫХ ПАРАЗИТОЗОВ СРЕДИ СЕЛЬСКОГО НАСЕЛЕНИЯ. *Educational Research in Universal Sciences*, 2(10), 100–104. Retrieved from <https://erus.uz/index.php/er/article/view/4152>

23. Арипов, А. М. (2024). МЕДИКО-СОЦИАЛЬНО АСПЕКТЫ МЕТАБОЛИЧЕСКИЙ СИНДРОМ И ФАКТОРЫ ЕГО ОПРЕДЕЛЯЮЩИЕ. SCHOLAR, 2(8), 16–21. <https://doi.org/10.5281/zenodo.14641358>
24. Байкузиев, У. К., & Махмудов, Н. И. (2019). ТРОМБОЛИТИЧЕСКАЯ ТЕРАПИЯ У БОЛЬНЫХ С ОСТРЫМ КОРОНАРНЫМ СИНДРОМОМ С НОРМАЛЬНЫМ И НАРУШЕННЫМ УГЛЕВОДНЫМ ОБМЕНОМ (РЕГИСТР ОСТРОГО КОРОНАРНОГО СИНДРОМА Г. ФЕРГАНЫ). *Евразийский кардиологический журнал*, (S1), 202.
25. Исмаилов, Ж. Т., Усманов, Б. С., & Махмудов, Н. И. (2013). Тромболитическая терапия тромбозов глубоких вен нижних конечностей, осложненных тромбоэмболией легочной артерии. *Вестник экстренной медицины*, (3), 90-90.
26. Махмудов, Н. И. (2024). ДИАГНОСТИКА И ЛЕЧЕНИЯ ПОСТТРАВМАТИЧЕСКОЙ ПНЕВМОНИИ У БОЛЬНЫХ С ЗАКРЫТЫМИ ТРАВМАМИ ГРУДИ. *Экономика и социум*, (5-2 (120)), 1134-1138.
27. Махмудов, Н. И. (2025). ЭПИДЕМИОЛОГИЯ И ДИАГНОСТИКА ГОСПИТАЛЬНЫХ ПНЕВМОНИЙ У БОЛЬНЫХ ЧЕРЕПНО-МОЗГОВОЙ ТРАВМОЙ. *Экономика и социум*, (5-1 (132)), 1307-1309.
28. Махмудов, Н., Йулдашев, Ш., & Сайдалиев, С. (2023). Стандарт лечения гнойных осложнений при открытых переломах у детей. *Актуальные вопросы детской хирургии*, 1(1), 34-35.
29. Назирхужаев, Ф., Махмудов, Н., & Йулдашев, Ш. (2023). О комплексном лечении острого гнойного плеврита у детей. *Актуальные вопросы детской хирургии*, 1(1), 36-37.
30. Сидиков, А. А., & Суяров, Ш. М. (2024). ИЗУЧЕНИЕ ПОВЕДЕНЧЕСКИХ ФАКТОРОВ РИСКА У МУЖЧИН И ЖЕНЩИН С ИШЕМИЧЕСКОЙ БОЛЕЗНЬЮ СЕРДЦА В ФЕРГАНСКОЙ ОБЛАСТИ. *MODELS AND METHODS FOR INCREASING THE EFFICIENCY OF INNOVATIVE RESEARCH*, 3(35), 202-208.
31. Суяров, Ш. М. (2025). ФАКТОРЫ РИСКА РАЗВИТИЯ ИШЕМИЧЕСКОЙ БОЛЕЗНИ СЕРДЦА В УЗБЕКИСТАНЕ: СОВРЕМЕННОЕ СОСТОЯНИЕ И ПУТИ РЕШЕНИЯ. *Евразийский журнал медицинских и естественных наук*, 5(3), 98–102. извлечено от <https://in-academy.uz/index.php/EJMNS/article/view/46909>
32. Суяров, Ш. М. (2024). ОЦЕНКА СОЦИАЛЬНО-ДЕМОГРАФИЧЕСКИХ ПОКАЗАТЕЛЕЙ У БОЛЬНЫХ С ИБС В ФЕРГАНСКОЙ ОБЛАСТИ. *АКТУАЛЬНЫЕ ПРОБЛЕМЫ ДИАГНОСТИКИ ЛЕЧЕНИЯ ВНУТРЕННИХ БОЛЕЗНЕЙ*, 98.
33. Суяров, Ш. М. У. (2025). ФАКТОРЫ РИСКА РАЗВИТИЯ ИШЕМИЧЕСКОЙ БОЛЕЗНИ СЕРДЦА В УЗБЕКИСТАНЕ: СОВРЕМЕННОЕ СОСТОЯНИЕ И ПУТИ РЕШЕНИЯ. *Eurasian Journal of Medical and Natural Sciences*, 5(3), 98-102.
34. Усманов, Б. С., Махмудов, Н. И., Исмаилов, Ж. Т., & Дадабаев, Х. Р. (2009). Тактика лечения больных с повреждениями магистральных сосудов нижних конечностей. *Вестник экстренной медицины*, (3), 49-51.
35. Хайдаров, А., Махмудов, О., Абдурахманов, И., & Махмудов, Н. (2017). ВЛИЯНИЕ ОРОШЕНИЯ И СХЕМЫ ПОСЕВА НА РАСХОД ВОДЫ НОВЫХ СОРТОВ ХЛОПЧАТНИКА. *Актуальные проблемы современной науки*, (6), 157-161.