ионы двухвалентного и одновалентного железа; это может ускорить повреждение клеток и сосудов, что приводит к сосудистым осложнениям СД 2 [1].

Существует предположение, что влияние ингибиторов натрий-глюкозного котранспортера 2-го типа (НГлТ-2) на уровень МК можно объяснить способностью увеличивать экскрецию МК с мочой [1]. Взрослые с СД 2, которым был назначен иНГЛТ-2, имели более низкую частоту возникновения подагры, чем те, кто получал агонист рецепторов глюкагоноподобного пептида-1 [1].

У ингибиторов дипептидилпептидазы-4 (ДПП- 4) также было выявлено влияние на обмен МК. Вилдаглиптин снижал уровень МК у пациентов с СД 2 [1]. Линаглиптин оказывал влияние на пуриновый обмен через двойной механизм, ингибируя активность ДПП-4 и ее связывание с аденозиндезаминазой (АДА) с последующим повышением уровня аденозина и снижением доступности субстратов для КО, а также непосредственно ингибируя активность КО благодаря наличию в химической структуре пуринового кольца, причем не только у пациентов с СД 2, но и у здоровых добровольцев [1].

Перспективным представляется изучение свойств данных препаратов для лечения СД 2, направленных на снижение уровня МК.

Выводы

Полученные в результате анализа данные свидетельствуют о ведущей роли препаратов и НГлТ-2, иДПП- 4 Использующихся для лечения сахарного диабета 2-го типа.

Несомненно, необходимо больше исследований для определения причинно-следственных нарушений пуринового и углеводного обменов. Подтверждение связи подагры и СД2, выявление уровня МК, при котором риск развития СД2 увеличивается, могут способствовать разработке методов первичной профилактики подагры.

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EXPLORING MITOCHONDRIAL DYSFUNCTION AND STRESS RESPONSES IN ENHANCING CANCER PROGNOSIS

Introduction

Mitochondria are integral to cellular metabolism, apoptosis, and calcium homeostasis, playing a crucial role in maintaining cellular health. Their dysfunction has been implicated in various diseases, particularly cancer, where they contribute to tumorigenesis and cancer progression. In our experiments on the effect of incorporated ^{137}Cs on energy metabolism in the

myocardium of white rats, we used eight male rats divided into two groups, fed products contaminated with ^{137}Cs for seven days. Polarography assessed oxygen consumption in myocardial tissue preparations with endogenous substrates and added exogenous succinic and glutamic acids, as well as the uncoupler 2,4-dinitrophenol. Results showed increased respiration rates on endogenous substrates due to oxidative phosphorylation uncoupling, with no significant change in substrate amounts. This disruption in energy production may be linked to altered potassium channel function, potentially explaining cardiovascular pathology from ionizing radiation [1].

Recent studies have highlighted the potential of mitochondrial markers as indicators of cancer prognosis and therapeutic strategies. This article explores the relationship between mitochondrial dysfunction, cellular stress, and oncomarkers in the context of cancer, aiming to elucidate how these factors can enhance cancer prognosis and inform treatment approaches.

Aim

The primary aim of this article is to investigate the role of mitochondrial dysfunction and its relationship with oncomarkers in cancer. By examining how stress affects mitochondrial function and contributes to cancer progression, this research seeks to identify potential biomarkers that can improve cancer diagnosis and treatment outcomes.

Tasks

1. Analyze the relationship between mitochondrial dysfunction and cellular stress in cancer.
2. Identify various types of oncomarkers associated with mitochondrial activity.
3. Evaluate the clinical implications of mitochondrial markers in cancer prognosis and therapy.
4. Discuss the limitations of current oncomarker tests in oncology.

Materials and Methods

This study utilizes a comprehensive literature review approach, focusing on recent findings related to mitochondrial function, oncomarkers, and their roles in cancer biology. Key sources include peer-reviewed articles from journals such as *Signal Transduction and Targeted Therapy*, *Biomedical Journal, and Journal of Biomedical Science* [2–4]. The analysis includes:

- Examination of mitochondrial DNA (mtDNA) mutations.
- Review of metabolic enzymes and proteins involved in mitochondrial biogenesis.
- Assessment of reactive oxygen species (ROS) production as a byproduct of mitochondrial dysfunction.

Obtained Results and Their Discussion

Mitochondrial Dysfunction as a Source of Oncomarkers

Mitochondrial dysfunction leads to several metabolic alterations that are characteristic of cancer cells. One significant phenomenon is the **Warburg effect**, where cancer cells preferentially utilize glycolysis for energy production, even in the presence of oxygen. This metabolic shift results in:

- **Increased ROS Production:** Dysfunctional mitochondria generate higher levels of ROS, contributing to oxidative stress that can damage cellular components and promote tumorigenesis.

- **Release of Mitochondrial Markers:** Cancer cells may release mtDNA, mitochondrial proteins, and other molecules into circulation, which can serve as potential oncomarkers for diagnosis and prognosis.

Types of Oncomarkers Related to Mitochondrial Function

Oncomarkers can be categorized into several types:

- **Proteins:** Enzymes or hormones produced by cancer cells (e.g., PSA for prostate cancer).
- **Genes:** Specific mutations detectable in blood or tumor tissue (e.g., BRCA1/2).
- **DNA/RNA Fragments:** Circulating tumor DNA (ctDNA) that reflects tumor presence.
- **Reactive Oxygen Species:** Elevated levels indicating mitochondrial dysfunction.

Clinical Implications

The identification of mitochondrial markers has significant clinical implications:

- **Prognosis Prediction:** Certain oncomarker levels can indicate the likelihood of recurrence or progression.
- **Therapeutic Targets:** Mitochondria represent promising targets for new therapies aimed at disrupting energy production in cancer cells.
- **Monitoring Treatment Response:** Tracking changes in oncomarker levels during therapy can provide insights into treatment effectiveness.

Limitations of Oncomarkers

Despite their potential, oncomarkers have notable limitations:

- **Lack of Specificity:** Many oncomarkers are not exclusive to cancer; elevated levels can occur in benign conditions.
- **Sensitivity Issues:** Some cancers may not produce detectable levels of oncomarkers until advanced stages.
- **False Positives/Negatives:** Results can lead to misdiagnosis or missed diagnoses.

Conclusion

The interplay between mitochondrial dysfunction, cellular stress, and oncomarkers is complex yet critical for understanding cancer biology. Mitochondrial markers hold promise for enhancing cancer prognosis by providing insights into tumor behavior and treatment responses. However, challenges remain regarding their specificity and sensitivity as diagnostic tools. Continued research is essential to fully realize the clinical utility of mitochondrial-based oncomarkers, potentially leading to improved personalized treatment strategies for cancer patients.

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