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Metformin - An Antidiabetic Drug With Anti-aging, Cardioprotective And Potential Anti-cancer Properties

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ABSTRACT

Introduction: This review aims to examine the role of metformin, a widely used oral antihyperglycemic agent, and its emerging potential cardioprotective, anticancer, and anti-aging effects.

Materials and Methods: A comprehensive review of the literature was conducted using the PubMed and Google Scholar databases using the following keywords: “metformin”, “anti-aging”, “cardioprotective”, ”anti-cancer”, “AMPK”, “diabetes”, “anti-inflammatory”, “cancer”, “mTOR”

Summary: Metformin, originally developed to treat type 2 diabetes, has been widely used due to its effectiveness in lowering blood glucose without causing hypoglycemia, and it exhibits a strong safety profile with additional benefits like weight loss and improved cardiovascular health. Beyond diabetes management, metformin shows promising anti-aging effects by improving mitochondrial function, regulating protein homeostasis, reducing inflammation, and modulating the microbiome. Furthermore, metformin has potential protective roles against

dementia, skin aging, cardiovascular diseases, and various cancers through mechanisms involving AMPK activation and immune modulation.

Conclusions: Metformin demonstrates a wide range of beneficial effects beyond glucose control, including anti-aging, anti-inflammatory, cardiovascular protective, and potential anticancer properties. Through its cellular and metabolic mechanisms, metformin shows promise not only as a treatment for type 2 diabetes but also in the prevention and management of various age-related diseases. However, more high-quality clinical studies are needed to confirm these findings.

Keywords: “metformin”, “anti-aging”, “cardioprotective”, ”anti-cancer”, “AMPK”, “diabetes”, “anti-inflammatory”, “cancer”, “mTOR”

Introduction

Metformin, a drug used to manage diabetes, received FDA approval in 1994 for the treatment of type 2 diabetes [1]. Metformin belongs to the biguanide class of drugs, which originally come from herbal sources, and it has been commonly used to manage diabetes since the 1950s. Two other biguanides were removed from medical use because they led to lactic acidosis. Metformin was temporarily withdrawn from the US market over similar safety concerns but was later shown to be safe and effective at reducing blood glucose levels [2]. It is available in both immediate- and extended-release forms and is frequently used in combination with other diabetes medications [1]. Metformin is an oral antihyperglycemic agent that effectively reduces both fasting and postprandial plasma glucose concentrations in patients with type 2 diabetes

mellitus. Its mechanism of action involves improving insulin sensitivity, inhibiting hepatic gluconeogenesis, and decreasing intestinal glucose absorption. Unlike other classes of oral hypoglycemic drugs, metformin does not induce hypoglycemia or hyperinsulinemia [3]. Metformin acts through several mechanisms across various tissues. It suppresses glucose production in the liver, enhances insulin-dependent glucose uptake in muscle tissue, and promotes both uptake and metabolism of glucose in the intestines. After being absorbed in the gut, about 50-60% of the orally administered drug is bioavailable, and it travels through the portal vein to accumulate primarily in the liver.

Additionally, studies in mice and healthy humans have shown that metformin may aid in weight loss and reduce appetite by influencing levels of growth differentiation factor-15 and affecting appetite control centers in the hypothalamus [4]. Following absorption, metformin exhibits an onset of action at approximately 1.5 hours. It has a plasma elimination half-life of 2 to 3 hours, with a duration of effect ranging from 6 to 10 hours [3]. The most frequently reported side effects of metformin are gastrointestinal, including diarrhea, nausea, and abdominal discomfort. These symptoms are generally mild, temporary, and related to the dosage, but they can affect up to half of the patients using the drug. Approximately 5% of people are unable to tolerate metformin even at low doses. A much less common but more serious side effect of biguanide treatment is lactic acidosis [5]. Metformin is generally not recommended for patients who have risk factors for lactic acidosis, such as liver problems, heart failure, or chronic kidney disease (CKD). However, continued clinical experience suggests that metformin is unsafe only for a limited group of patients with severe impairments in liver, heart, or kidney function [2]. A substantial body of experimental and clinical evidence has established that metformin exerts pleiotropic effects. Furthermore, numerous investigations - including meta-analyses and large-scale population studies - have confirmed its favorable safety profile and tolerability, as well as its potential cardioprotective effects. Recent findings also indicate that, beyond its role in improving glucose homeostasis, metformin may contribute to a reduced risk of oncogenesis, a lower incidence of neurodegenerative disorders, and an extension of lifespan [6].

Materials and Methods

A comprehensive literature search was conducted to gather relevant studies investigating the effects of metformin beyond glucose control, including its anti-aging, cardioprotective, anti-inflammatory, and anticancer properties. The databases PubMed and Google Scholar were used to identify peer-reviewed articles published up to 2025. The following keywords and their

combinations were used: “metformin”, “anti-aging”, “cardioprotective”, “anti-cancer”, “AMPK”, “diabetes”, “anti-inflammatory”, “cancer”, and “mTOR”.

Anti-age effects

The effect on mitochondrial function

Mitochondria play a vital role in energy production and cell health. Damage to mitochondrial DNA leads to lower energy output, increased stress, and cell death, accelerating aging and functional decline. Metformin improves mitochondrial function by partially inhibiting complex I, which raises the AMP/ATP ratio and activates AMPK, helping restore energy balance. In models of Parkinson’s disease and astrocyte aging, metformin delays senescence through the Mfn2-cGAS pathway, reducing neurodegeneration. Additionally, AMPK regulates histone H3K79 methylation, which increases the expression of SIRT3, a key mitochondrial deacetylase. This promotes mitochondrial biogenesis and helps delay cellular aging, reducing age-related vascular dysfunction [7]. An increase in AMPK activity may also account for metformin’s protective impact on endothelial function by activating endothelial nitric oxide synthase (eNOS), which helps to offset the harmful effects of a diabetic environment on cardiovascular health. Additionally, AMPK suppresses mTOR signaling, and this mechanism might play a role in lowering the risk of certain cancers linked to metformin use [8].

Regulation of protein homeostasis

Metformin extends lifespan primarily by regulating protein homeostasis. It reduces progerin expression - a mutant form of lamin A linked to premature aging - by inhibiting mTORC1 signaling, which suppresses key translation regulators like p70S6K, rpS6, and 4E-BP1. It also promotes progerin clearance via AMPK/mTOR/ULK1-driven autophagy, delaying cellular senescence. Additionally, metformin boosts protective proteins such as GPx7, which defends cells from oxidative stress and supports longevity. However, its anti-aging effects vary with age, being more pronounced in younger individuals. A clinical trial in people over 70 showed that metformin alters gene expression in a tissue-specific manner and helps mitigate age-related metabolic decline, possibly by modulating regulators like mTORC1, MYC, and TNF [9].

Lysosomes

Lysosomes are key regulators of cellular signaling and metabolism. Metformin activates AMPK and inhibits mTORC1 through processes involving the lysosome, possibly via the lysosomal V-ATPase. It also affects metal ion homeostasis - especially copper, zinc, and iron - due to its metal-binding properties and accumulation in mitochondria. This may create competition between mitochondria and lysosomes, linking metformin's mitochondrial actions to lysosomal regulation. These interactions could explain metformin's anti-inflammatory and potential longevity-promoting effects, though more research is needed to fully understand these mechanisms [10].

The Anti-Inflammatory Effects

Metformin not only improves metabolic health but also regulates key immune functions. It lowers levels of pro-inflammatory cytokines, reduces mortality risk in older individuals with diabetes, and suppresses inflammatory signaling pathways in liver cells. Its anti-inflammatory effects involve blocking NF-κB activation and preventing monocytes from turning into macrophages through AMPK-dependent mechanisms. Additionally, by promoting weight loss and enhancing insulin sensitivity, metformin helps reduce systemic inflammation indirectly [11].

The Microbiome

Studies have shown that the microbiota is strongly linked to many age-related diseases such as type 2 diabetes, obesity, and cancer. In some organisms, metformin may slow aging and related disorders by modulating the microbiome. Human research indicates that metformin increases bacteria that produce short-chain fatty acids, which help with weight loss and reduce inflammation in people with diabetes. Additionally, changes in the microbiota after metformin use may explain its blood sugar-lowering effects and associated side effects [10].

Dementia

Research indicates that metformin use in type 2 diabetes patients is linked to a significantly reduced risk of dementia, especially with use longer than two years. This reduction followed a dose-response trend and remained consistent across analyses, regardless of the year of patient enrollment. Though the exact mechanisms are not fully understood, metformin's beneficial effects may stem from its ability to lower blood glucose by activating the LKB1/AMPK pathway and inhibiting liver gluconeogenesis. This pathway also appears to have neuroprotective properties. Additionally, metformin improves insulin resistance - common in dementia patients - by enhancing insulin signaling. One trial even detected metformin in cerebrospinal fluid and noted cognitive improvements [12].

Skin Aging

Metformin might have a protective effect against skin aging, which is the most typical sign of aging, this potential role has not been thoroughly studied. Experimental research indicates that metformin may have anti-aging properties for the skin. The suggested molecular mechanisms behind metformin's skin anti-aging effects primarily involve the reduction of nuclear factor kappa B (NF-κB) (p65) activity. Additionally, metformin seems to suppress the release of pro-inflammatory cytokines triggered by ultraviolet B (UVB) exposure [13]. Apoptosis removes senescent cells, decreasing their presence in the body and consequently raising the number of non-senescent cells, which helps slow down the aging of skin caused by photoaging [14].

Cardiovascular diseases

Current clinical studies on metformin's heart-protective effects mainly focus on coronary heart disease, heart failure, heart attack, and pulmonary hypertension. Metformin lowers coronary heart disease risk by improving endothelial function and slows early coronary plaque buildup in prediabetic men. It also alters serum lipids, reducing cardiovascular event risk [15]. Metformin, administered either before or after treatment, has been shown to decrease infarct size and improve survival rates after a myocardial infarction [16]. Metformin, through the activation of AMP-activated protein kinase (AMPK), attenuates alpha-dicarbonyl-induced modifications of apolipoprotein residues, thereby mitigating dysfunction of high-

density lipoproteins (HDL) and limiting structural alterations of low-density lipoproteins (LDL). As a result, improved HDL function enhances cholesterol transport and contributes to a lower risk of cardiovascular disease [17] .Additionally, metformin decreases myocardial oxygen use and lowers left ventricular mass, blood pressure, and oxidative stress in coronary artery disease patients [15]. Metformin's protective effects against atherosclerosis involve activating AMPK, reducing inflammation in key vascular cells, and lowering oxidative stress [18]. A randomized controlled trial conducted in non-diabetic patients with coronary artery disease demonstrated that metformin significantly attenuates left ventricular hypertrophy (LVH), a robust prognostic indicator in coronary artery disease. The findings of this study revealed that metformin markedly decreased left ventricular mass indexed to height, absolute left ventricular mass, body weight, and oxidative stress parameters [17].

Cancer

Metformin enhances anticancer immunity by reshaping the tumor immune microenvironment. In esophageal squamous cell carcinoma, it boosts CD8+ T cell infiltration and reduces CD163+ tumor-promoting macrophages by activating AMPK and inhibiting STAT3, which alters cytokine production (increased TNF, decreased IL-10). In head and neck cancers, metformin enhances NK cell activity and cytotoxicity via an AMPK-independent, mTORC1–STAT1-mediated suppression of CXCL1. In liver cancer linked to NASH, metformin improves CD8+ T cell response to anti-PD1 therapy through metabolic reprogramming, increasing T cell mobility and therapy effectiveness. Additionally, using metformin-loaded macrophage-derived microparticles to reprogram M2 macrophages to an M1-like state improves the tumor microenvironment. This shift boosts CD8+ T cell infiltration, reduces suppressive immune cells, and enhances anti-PD1 therapy efficacy [11]. Metformin has been demonstrated to reduce cell proliferation and colony formation across multiple breast cancer cell lines, regardless of the status of estrogen receptor, progesterone receptor, HER-2, or p53. Additionally, it has been found to inhibit the growth of other types of cancer cell lines, including those from colon, prostate, endometrial, ovarian, lung, and brain cancers. These in vitro results align with animal studies using mouse models for different cancers like breast and lung, where metformin administered at doses similar to those used in humans led to decreased tumor growth. Moreover, metformin reduced tumor formation by 25% in mice with a heterozygous mutation in the tumor suppressor gene PTEN, which predisposes them to tumors in various organs. It is important to note, however, that the metformin dose used in this study was more than ten times higher than

the typical clinical dose [19]. Moreover, when used alongside chemotherapy, metformin has demonstrated strong potential in treating leukemia. Its antitumor effects appear to be particularly enhanced in acute myeloid leukemia (AML) when combined with cytarabine (ara-C), likely due to the suppression of the mTORC1/P70S6K signaling pathway [20]. Obesity and type 2 diabetes (T2DM) increase the risk of colonorectal cancer (CRC), partly due to high insulin and insulin-like growth factor levels that promote cancer cell growth. Metformin, the first-line treatment for T2DM, has shown potential as a chemopreventive agent against CRC. Epidemiological studies indicate that diabetic patients taking metformin have a lower risk of developing CRC and improved survival rates after diagnosis. Additionally, metformin may enhance the effects of chemotherapy and immunotherapy, though results are mixed and more research is needed to clarify its mechanisms. Animal studies support metformin's ability to inhibit tumor growth and reduce the development of polyps by activating AMPK and suppressing pathways involved in cell proliferation. It also appears to improve outcomes when combined with standard chemotherapy drugs. Metformin may help counteract the negative effects of obesity on CRC by modulating metabolism and inflammation [21].

Conclusion

Metformin is a key drug for managing type 2 diabetes. It lowers blood glucose by improving insulin sensitivity, reducing liver glucose production, and decreasing intestinal absorption, without causing hypoglycemia. Its safety profile is well established, with precautions mainly for patients with significant liver, kidney, or heart impairments. Beyond diabetes, metformin exerts multiple beneficial effects. It improves mitochondrial function, activates AMPK, and promotes cellular energy balance, which helps delay aging and vascular dysfunction. It regulates protein homeostasis by inhibiting mTORC1 and enhancing autophagy, reducing cellular senescence. Metformin also lowers inflammation, positively modulates the gut microbiome, and is linked to reduced risk of dementia. Cardiovascular benefits include improved endothelial function, reduced left ventricular hypertrophy, and lower oxidative stress, contributing to decreased heart disease risk. In cancer, metformin enhances immune response against tumors, inhibits cancer cell growth, and is associated with reduced incidence and better outcomes in various cancers.

In summary, metformin is a multifaceted drug with effects that extend far beyond glucose control, offering promising potential for anti-aging, cardiovascular protection, neuroprotection,

and cancer prevention. Continued research is essential to fully unlock its broad therapeutic value.

Disclosure

Author's contribution

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The authors deny any conflict of interest.

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