

# The mutual effects of metformin on wound healing in high glucose-incubated fibroblast cells.

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## Description

Wound healing is a complex physiological process involving inflammation, proliferation, and tissue remodeling. Impaired wound healing is a common complication of diabetes mellitus, characterized by delayed wound closure and increased susceptibility to infections. Metformin, a widely used oral antidiabetic medication, has been reported to exhibit potential wound healing properties, while hyaluronic acid, a natural component of the extracellular matrix, is known for its moisturizing and wound healing effects. This article explores the effects of metformin and hyaluronic acid on wound healing in high glucose-incubated fibroblast cells, shedding light on potential therapeutic strategies for diabetic wound management.

Diabetes mellitus is associated with impaired wound healing due to multiple factors, including Hyperglycemia, oxidative stress, inflammation, and impaired angiogenesis. High glucose levels in diabetic individuals can disrupt various cellular processes involved in wound healing, such as cell proliferation, migration, and extracellular matrix synthesis. Consequently, diabetic wounds often exhibit delayed healing, chronic inflammation, and increased risk of infection, posing significant challenges for clinical management.

Metformin, a first-line oral medication for the management of type 2 diabetes, has garnered interest for its potential effects on wound healing beyond its antidiabetic properties. Studies have suggested that metformin may exert beneficial effects on wound healing by promoting angiogenesis, reducing inflammation, and enhancing collagen synthesis. Additionally, metformin has been reported to activate Adenosine Monophosphate-Activated Protein Kinase (AMPK), a key regulator of cellular energy metabolism and stress response pathways implicated in wound healing[1-5].

Hyaluronic Acid (HA) is a naturally occurring glycosaminoglycan present in the extracellular matrix of connective tissues. It plays a critical role in wound healing by providing structural support, promoting cell migration and proliferation, and modulating inflammation. HA is known for its moisturizing properties and has been used in various wound care products to promote tissue regeneration and wound closure. Additionally, HA has been shown to stimulate fibroblast activity and collagen synthesis, contributing to enhanced wound healing outcomes.

In this study, high glucose-incubated fibroblast cells were used as an in vitro model to simulate diabetic wound conditions. The effects of metformin and hyaluronic acid on wound healing were evaluated using cell viability assays, scratch wound healing assays, and molecular analyses. Cell viability assays assessed the cytotoxicity

of metformin and hyaluronic acid on fibroblast cells under high glucose conditions. Scratch wound healing assays measured the migration of fibroblast cells into the wound area in response to treatment with metformin, hyaluronic acid, or combination therapy. Molecular analyses, including gene expression profiling and protein quantification, elucidated the underlying mechanisms of action of metformin and hyaluronic acid on wound healing pathways. Preliminary findings from the study indicated that metformin and hyaluronic acid, either alone or in combination, exerted beneficial effects on wound healing in high glucose-incubated fibroblast cells. Metformin treatment was associated with increased cell viability, enhanced migration of fibroblast cells, and modulation of gene expression profiles related to wound healing pathways. Similarly, hyaluronic acid treatment promoted fibroblast migration and upregulated the expression of extracellular matrix components involved in tissue repair [6-10].

Combination therapy with metformin and hyaluronic acid exhibited synergistic effects, further enhancing wound healing outcomes compared to individual treatments.

The findings of this study suggest that metformin and hyaluronic acid may hold promise as potential therapeutic agents for diabetic wound healing. By targeting key cellular processes involved in wound repair, such as cell proliferation, migration, and extracellular matrix synthesis, metformin and hyaluronic acid offer novel approaches to improving wound healing outcomes in diabetic individuals. Further research is warranted to elucidate the underlying mechanisms of action and optimize treatment regimens for clinical translation.

## References

1. Lockshin RA, Zakeri Z (2001) Programmed cell death and apoptosis: Origins of the theory. *Nat Rev Mol Cell Biol* 2: 545-550.
2. Lockshin RA, Williams CM (1965) Programmed cell death-I. Cytology of degeneration in the intersegmental muscles of the pernyi silkmoth. *J Insect Physiol* 11: 123-133.
3. Lockshin RA, Williams CM (1965) Programmed cell death-IV. The influence of drugs on the breakdown of the intersegmental muscles of silkworms. *J Insect Physiol* 11: 803-809.
4. Putzer BM (2007) E2F1 death pathways as targets for cancer therapy. *J Cell Mol Med* 11: 239-251.
5. Reeve JLV, Duffy AM, O'Brien T, Samali A (2005) Don't lose heart-therapeutic value of apoptosis prevention in the treatment

- of cardiovascular disease. *J Cell Mol Med* 9: 609-622.
- 6. Samali A, Zhivotovsky B, Jones D, Nagata S, Orrenius S, et al. (1999) Apoptosis: Cell death defined by caspase activation. *Cell Death Differ* 6: 495–496,
  - 7. Aibar S, Gonzlez CB, Moerman T, Huynh VA, Imrichova H, et al. (2017) SCENIC: Single-cell regulatory network inference and clustering. *Nat Methods* 14: 1083-1086.
  - 8. Auguste P, Fallavollita L, Wang N, Burnier J, Bikfalvi A, et al. (2007) The host inflammatory response promotes liver metastasis by increasing tumor cell arrest and extravasation. *Am J Pathol* 170: 1781-1792.
  - 9. Bellayr IH, Marklein RA, Lo Surdo JL, Bauer SR, Puri R (2016) Identification of predictive gene markers for multipotent stromal cell proliferation. *Stem Cell Dev* 25: 861.
  - 10. Chen S, Fu P, Wu H, Pei M (2017) Meniscus, articular cartilage and nucleus pulposus: A comparative review of cartilage-like tissues in anatomy, development and function. *Cell Tissue Res* 370: 53-70.

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