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The complexity of nicotine's effect on cutaneous wound healing

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ABSTRACT

Nicotine, mostly known through its association with tobacco, has long been regarded as straightforwardly harmful - addictive and vasoconstrictive. Yet a growing number of studies have begun to complicate that picture, revealing a range of physiological effects that are far less predictable than once assumed. Over the years, multiple contradictory studies have emerged, a few suggesting that nicotine, under certain circumstances, can be beneficial – especially for angiogenesis. In this review, we aim to supply a comprehensive summary of the most significant effects of nicotine on cutaneous wound healing. We focus on key components of this process, including keratinocytes, fibroblasts, endothelial cells, inflammation, and the crucial cellular events of proliferation, differentiation, and migration.

Keywords: nicotine, skin, wound healing, fibroblasts, inflammation, angiogenesis

1. INTRODUCTION

Nicotine is a pyridine alkaloid typically found in tobacco leaves. Nicotine has been well known for centuries, and many researchers describe its receptor as the best-characterised neurotransmitter receptor. However, its impact on the human body remains unknown. This alkaloid has an omnidirectional influence on the human body. Nicotine interacts with nicotinic acetylcholine (nACh) receptors, affecting cell proliferation, differentiation, and migratory capacity. Nicotinic receptors are found in both peripheral and central neurons, as well as in glial cells, platelets, fibroblasts, vascular endothelial cells, bronchial epithelial cells, keratinocytes, neutrophils, lymphocytes, and various organs, including the bowel. Extensive research has shown that nicotine harms the human body, mainly because of its addictive potential and vasoconstrictive effects (Martin et al., 2009; Misery, 2004). Its addictive potential originates from stimulating dopaminergic neurons in the mesolimbic system. Nicotine is an agonist of nicotinic acetylcholine (nACh) receptors containing the α4, α5, α6, β2, and β3 subunits, in various combinations, which are directly responsible for triggering dopamine release from these neurons (Picciotto and Kenny, 2021). Researchers have identified 17 subunits of the nicotinic acetylcholine (nACh) receptor, naming them α1–α10, β1–β4, γ, δ, and ε (Kalamida et al., 2007). Nicotinic acetylcholine (nACh) receptors found in the skin consist of five subunits forming a transmembrane pathway for ions. The exact composition of these

subunits is directly responsible for the function and pharmacological effect of the formed ionic canals (McGehee and Role, 1995; Sgard et al., 2002). In this paper, we will review nicotine's effects on cutaneous wound healing processes.

2. REVIEW METHODS

The authors of this paper reviewed reports, reviews, articles, and scientific literature. We conducted the search using reliable databases such as PubMed, the National Library of Medicine, ScienceDirect, Google Scholar, and the World Health Organization (WHO). To find appropriate source material, we used the following key words: "Nicotine", "Skin wound", "Skin healing", "nicotine's influence on skin", "Cutaneous inflammation", "Nicotine and inflammation". Cited sources cover the years from 1990 to 2021. We established inclusion criteria based on source reliability, clinical relevance, and the prevailing state of medical knowledge. We excluded case studies, non-academic articles, non-medical articles, studies and articles that discussed nicotine in contexts unrelated to the cutaneous healing process or inflammation, and articles written in languages other than English (Figure 1).

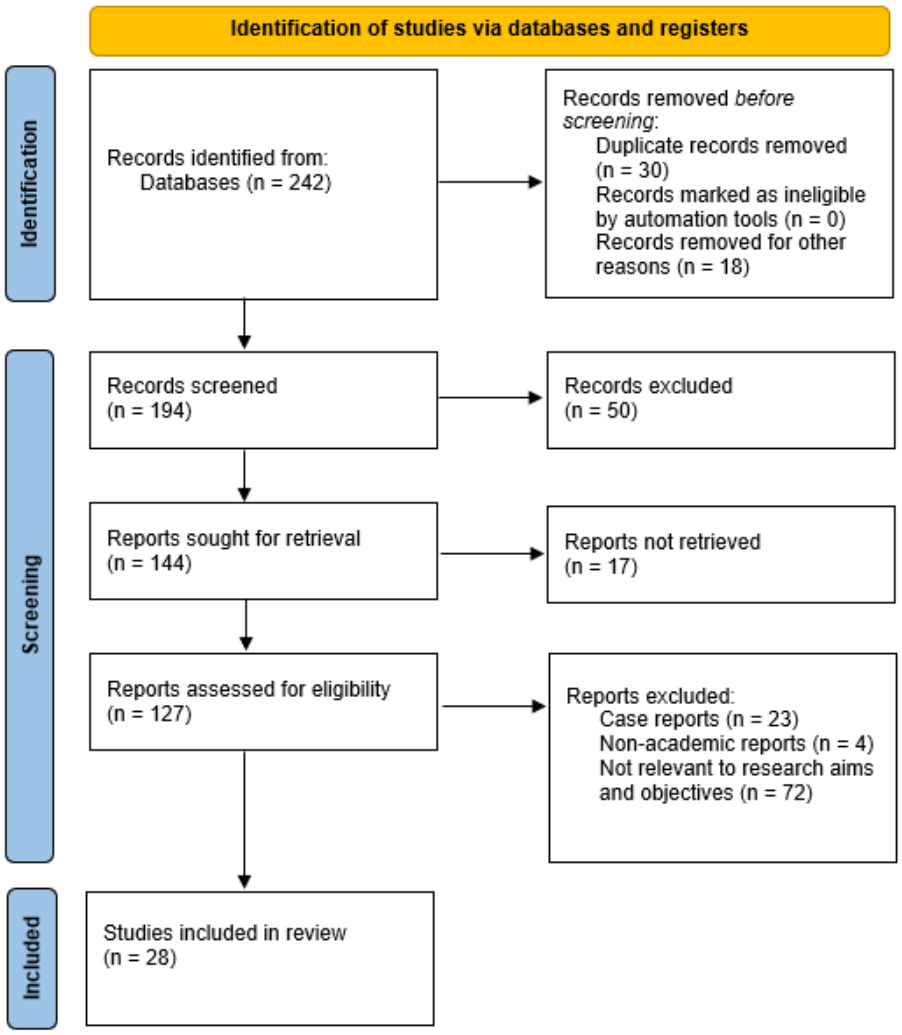

Figure 1: PRISMA chart

3. RESULTS & DISCUSSION

Wound healing process

The first cells that respond to injury are platelets. Their role is to aggregate to stop haemorrhage - the resulting clot releases platelet-derived growth factor (PDGF), which attracts macrophages to the wound site. Macrophages undergo activation and play a vital role in the entire healing process. They remove foreign bodies, dead cells, and debris of damaged tissue. In addition, platelets release increased amounts of platelet-derived growth factor (PDGF) (Diegelmann, 2004), as well as vascular endothelial growth factor (VEGF) and basic

fibroblast growth factor (bFGF) (Martin et al., 2009), which initiates the next stage of the process. The granulation. During granulation, fibroblasts migrate to the wound site. These cells are primarily responsible for rebuilding the extracellular matrix, creating a new and more stable base layer for the wound than the initial clot. The extracellular matrix forms a foundation for the formation of new blood vessels. During this stage, vascular endothelial growth factor (VEGF) stimulates endothelial progenitor cells (EPCs) to initiate angiogenesis. Effective wound closure needs a structured extracellular matrix and blood vessels, which provide oxygen and nutrients. The last stage is known as remodelling. Remodelling occurs as keratinocytes migrate and differentiate, consequently allowing the newly formed tissue to strengthen. Each of these stages is essential for wound healing. Nicotine influences every part of this process in various ways. However, it appears its effects can vary depending on multiple factors.

Keratinocytes

Nicotinic receptors found on keratinocytes, bronchial and vascular endothelial cells consist of α3, α5, β2, and β4 subunits, similarly to those on neurons in sympathetic ganglia. It's speculated that these receptors are responsible not only for the adverse constrictive effects on bronchi and blood vessels but also for controlling cell proliferation and differentiation in tegumental cells (Conti-Fine et al., 2000). Researchers found that the α5, α7, α3, β2, and β4 subunits in keratinocytes are involved in calcium influx, which leads to differentiation. This results in enhanced keratosis (Grando et al., 1996). Furthermore, agonists of α3, α9 subunits of nicotinic acetylcholine (nACh)and M3 muscarinic receptors regulate the expression of cadherins, catenins, and desmoglein 1 and 3, alongside the phosphorylation status of the latter (Nguyen et al., 2003).

Nicotine triggers a calcium ion influx, which stimulates the formation of the cornified envelope by recruiting more cells for this process. It additionally enhances the expression of transglutaminase 1, involucrin, filaggrin, and keratin 10, which is a cytoskeletal protein typically expressed in large quantities in the differentiated epidermal keratinocytes. This process increases the skin's stiffness and improves its structural integrity. At concentrations up to 100 μg/ml, nicotine induces cornification and squamatisation rather than being irritant (Grando, 2001). Nicotine contributes to cell-to-cell and cell-to-substrate adhesion, as well as to lateral migration of cultured keratinocytes. However, by increasing calcium influx, nicotine inhibits the mobility of these cells and slows re-epithelialization (Arredondo et al., 2002). In addition to other receptors like muscarinic M1, also the α3 and α9 subunits promote apoptosis in late-stage differentiated keratinocytes (Nguyen et al., 2001).

Fibroblasts

Human fibroblasts contain the α3, α5, α7, β2, and β4 subunits of the nicotinic acetylcholine (nACh) receptor. Arredondo et al. showed that exposing fibroblasts to 10^{-6} M nicotine for 24 hours increased levels of p21, cyclin D1, Ki-67, Proliferating Cell Nuclear Antigen (PCNA), Bcl-2, and caspase 3, resulting in altered expression of key cell cycle proteins. The researchers chose a concentration of 10−6 m because it is thought to resemble the one in chronic smokers' plasma (Arredondo et al., 2003). Apart from these, Wong et al. showed that nicotine in smoke stimulates the expression of interleukin-8, Protein Kinase B (PKB)/Akt, p53, and the previously mentioned p21 protein, resulting in increased fibroblast survival rather than death (Wong and Martins-Green, 2004). Smoke- and nicotine-exposed cells exhibited reduced mobility (Wong and Martins-Green, 2004). The activation of α1 subunit increases the production of extracellular matrix proteins such as collagen type I α, elastin, and metalloproteinase-1 (Arredondo et al., 2003). Nicotine impairs the ability of fibroblasts which limits their contribution to the wound healing process. Fibroblasts produce higher levels of stress and extracellular proteins, but their ability to migrate is limited. This restriction leads to prolonged healing, fibrosis, and abnormal connective tissue buildup.

Inflammation

Another vital part of the wound healing process is inflammation. Nicotine modulates it in various ways. Its activity is not unambiguously pro- or anti-inflammatory (Table 1). On the one hand, it induces cyclooxygenase-2 (COX-2), which, among other factors, is a reason for tobacco-related periodontal diseases, but also downregulates the anti-inflammatory cytokine IL-10 and, in certain circumstances, growth mediators like vascular endothelial growth factor (VEGF), platelet-derived growth factor (PDGF), transforming growth factor β1 (TGF-β1), and transforming growth factor β1 (TGF-β2). On the other hand, Sopori et al. demonstrated in a mouse model that nicotine inhibits the inflammatory response via the central nervous system (Sopori et al., 1998). Through peripheral sensory neurons, nicotine can act via nicotinic acetylcholine (nACh) receptors, resulting in the release of calcitonin gene-related peptide (CGRP)

(Dussor et al., 2003). The release of CGRP can have a bidirectional effect—both pro-inflammatory and anti-inflammatory—since CGRP blocks antigen presentation.

Furthermore, nicotine directly acts on immune system cells. It increases calcium influx and the activity of protein tyrosine kinase (PTK) in T-cells, effectively impairing their function (Sopori et al., 1998). Nicotine impairs the migratory ability of macrophages and fibroblasts. At the same time, it acts as a chemoattractant for neutrophils and increases their responsiveness to other chemotactic peptides. Other studies show that nicotine inhibits enzyme release and superoxide production by neutrophils and, via the α7 subunit of the nicotinic acetylcholine (nACh) receptor, blocks tumor necrosis factor (TNF) release by macrophages, additionally impairing inflammation and immunological defence (Misery, 2004). In a mouse model, Osborne-Hereford et al. showed that nicotine decreases IL-1β levels by activating the α7 subunit of the nicotinic acetylcholine (nACh) receptor (Osborne-Hereford et al., 2008). Johnson et al. also showed that nicotine stimulates IL-1α concentrations in a culture of keratinocytes (Johnson and Organ, 1997), which is an inflammatory mediator. Eliakim et al. did not observe this effect in their study, which focused on the bowel. Local effects of nicotine in the bowel were different as nicotine decreased levels of inflammatory cytokines (Eliakim and Karmeli, 2003). This finding may help explain nicotine's protective effects on the bowel in inflammatory bowel diseases. It also indicates that the effects of nicotine may differ considerably, even being contradictory, depending on the location.

Table 1. Pro- and Anti-inflammatory actions caused by nicotine

Pro-Inflammatory actions	*Anti-inflammatory actions*
Induces COX-2	Stimulates release of CGRP
Is a chemotactant for neutophils	Increases activity of PTK
Stimulates Il-1α concentrations	Impairs mobility of macrophages and fibroblasts
Down-regulates Il-10	Blocks TNF release by macrophages

Microcirculation and angiogenesis

Nicotine is typically associated with vasoconstriction and limited blood flow, which is necessary for a wound to heal. Nicotinic acetylcholine (nACh) receptors on endothelial cells mediate this effect. In addition, it increases platelet adhesiveness and reduces red blood cell proliferation (Misery, 2004). However, over recent years, many publications have reported contradictory conclusions about the effects of nicotine on microcirculation. Using a mouse model, Heeschen et al., (2001) demonstrated that nicotine supports angiogenesis in both in vitro and in vivo settings. In vitro, it increased endothelial tube formation and growth. In vivo, it stimulates fibrovascular growth. The researchers administered 0.03 μg/kg of nicotine locally or 100 μg/ml orally to mice, achieving plasma nicotine concentrations similar to those found in moderate smokers.

Similarly, Jacobi et al., (2002) evaluated that nicotine enhances wound healing in diabetic mice. Researchers showed that diabetic mice had a significantly impaired wound healing process compared to non-diabetic mice. Treatment of nicotine in 10−8 mg, 10−9 mg doses or 25 ug/kg basic fibroblast growth (bFGF) contributed to faster wound closure. Animals that received 10−8 mg of nicotine had increased vascularity in the area of the wound compared to other groups. Based on this observation, researchers concluded that nicotine may help in wound healing. Notably, by day 14 of follow-up, wound closure did not show significant differences between non-diabetic mice treated with phosphate-buffered saline and those treated with nicotine.

Later, Morimoto et al., (2008) conducted an experiment in which non-diabetic mice received 10−4 mg of topical nicotine, resulting in improved wound healing. Together, these two studies suggest that non-diabetic mice require larger doses of nicotine to achieve faster wound healing. Considerably, diabetic patients can present lower growth factor concentrations and, thus, are more prone to nicotine stimulation (Galiano et al., 2004). In a rat model, Li and Wang (2006) injected 9 μg/kg of nicotine intramuscularly into rodents with prior myocardial infarction. After 21 days, left ventricular capillary density was markedly increased.

Sugimoto et al., (2007) proposed that nicotine's proangiogenic action may extend to vasculogenesis, with endothelial progenitor cells (EPCs) as a key mediating target. To investigate this, the authors cultured EPCs from peripheral blood mononuclear cells drawn from healthy, non-smoking volunteers — crucially, without any direct nicotine exposure to the cells themselves. For 7 days prior to endothelial progenitor cell injection, the scientists ordinated athymic mice with ischemic hindlimbs oral nicotine at 100 ng/ml. The researchers then injected endothelial progenitor cells, into the tail vein, at a dose of 1×10^5 cells per mouse. After the injection, the researchers continued administering nicotine orally in the same manner for four more weeks. Laser Doppler analysis showed substantially enhanced blood perfusion in the previously ischemic hindlimbs, suggesting a proangiogenic effect of nicotine.

Conklin et al., (2002) concluded that cotinine, a nicotine metabolite, may have a greater angiogenic effect than nicotine, as at the same concentration, cotinine caused a 66% increase in vascular endothelial growth factor (VEGF) expression in endothelial cells compared to 52% with nicotine Cotinine's more pronounced effect is because of its longer half-life and plasma concentrations approximately 10 times higher than those of nicotine. Ng et al., (2007) also proved that nicotine is an angiogenic factor. Nicotine provoked dose-dependent migration of human microvascular endothelial cells. The extent of which was equal to 10 ng/ml of basic fibroblast growth (bFGF) but weaker than 10 ng/ml of VEGF.

A molecular analysis, a transcriptional profiling, revealed that all three factors: nicotine, basic fibroblast growth (bFGF) and vascular endothelial growth factor (VEGF) acted correspondingly as proangiogenic factors regulating gene expression. The thioredoxin-interacting protein (TXNIP), which acts as an endogenous inhibitor of thioredoxin, was co-repressed. Thioredoxin is a redox regulator. Silencing thioredoxin with small interfering RNA suppressed all angiogenesis-induced migration. Conversely, silencing thioredoxin-interacting protein (TXNIP) with small interfering RNA induced dose-dependent migration of human microvascular endothelial cells. In contrast, nicotinic acetylcholine (nACh) receptor antagonists inhibited the thioredoxin-stimulating activity of basic fibroblast growth factor (bFGF) and vascular endothelial growth factor (VEGF). Furthermore, nicotinic acetylcholine (nACh) receptor activation is required for dose-dependent migration of human microvascular endothelial cells. A migration of said cells is a key step in angiogenesis. Exemplary studies such as these by Wong and Martins-Green (2004) and Galiano et al., (2004) report on exogenously administered platelet-derived growth factor (PDGF), vascular endothelial growth factor (VEGF) and even basic fibroblast growth factor (bFGF) supporting diabetic wound healing. These conclusions, together with those of Jacobi et al., (2002) and Morimoto et al., (2008) support the existence of beneficial, though not yet fully elucidated, mechanisms underlying nicotine's role in wound healing.

4. CONCLUSION

The many-sided influence of nicotine, summarised in this review, provides a complex picture that points to the need for further examination of the mechanisms by which it affects the human body. The impact of nicotine is significantly determined through factors such as dosage, targeted cells, duration of exposure, whether it is acute or chronic, systemic and organ location, and other factors, some of which remain not fully understood. Notably, nicotine's effects appear to vary considerably with the duration of exposure — whether acute or chronic — and the concentrations at which it is applied. The cited studies collectively demonstrate that nicotine alters cellular activity at the molecular level, modulating gene expression. Its effects are dual, encompassing both pro-inflammatory and anti-inflammatory actions. Innovative local delivery strategies that minimize systemic exposure may therefore yield a viable avenue for utilizing nicotine's beneficial effects on wound healing while decreasing its adverse ones. Many contradictory reports prompt an evaluation of the exact mechanism behind nicotine's actions or even formulating a nicotinic analog deprived of nicotine's negative impact, but at the same time retaining some of its supposedly beneficial influences.

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Authors' Contributions

Jakub Jaworski: Conceptualization, supervising, writing, investigation, literature review
Ewelina Komorowska: Investigation, methodology
Natalia Kriese: literature review
Jakub Szyszkowski: formal analysis, editing
Tomasz Kucharski: Project administration
Brygida Tucka: editing
Zuzanna Zgrzywa: editing
Izabella Zawadzka: literature review
Paulina Wądołowska: methodology, literature review
Bartłomiej Kowalski: Project administration

Informed consent

Not applicable.

Ethical approval

Not applicable. This article does not contain any studies with human participants or animals performed by any of the authors.

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Conflict of interest

The authors declare that they have no conflicts of interest, competing financial interests or personal relationships that could have influenced the work reported in this paper.

Data and materials availability

All data associated with this study will be available based on the reasonable request to corresponding author.

REFERENCES

1. Arredondo J, Hall LL, Ndoye A, Nguyen VT, Chernyavsky AI, Bercovich D, Orr-Urtreger A, Beaudet AL, Grando SA. Central Role of Fibroblast α3 Nicotinic Acetylcholine Receptor in Mediating Cutaneous Effects of Nicotine. Lab Invest 2003;83(2):207-25. doi:10.1097/01.LAB.0000053917.46614.12
2. Arredondo J, Nguyen VT, Chernyavsky AI, Bercovich D, Orr-Urtreger A, Kummer W, Lips K, Vetter DE, Grando SA. Central Role of α7 Nicotinic Receptor in Differentiation of the Stratified Squamous Epithelium. J Cell Biol 2002;159(2):325-36. doi:10.1083/jcb.200206096
3. Conklin BS, Zhao W, Zhong DS, Chen C. Nicotine and Cotinine Up-Regulate Vascular Endothelial Growth Factor Expression in Endothelial Cells. Am J Pathol 2002;160(2):413-8. doi:10.1016/S0002-9440(10)64859-6
4. Conti-Fine BM, Navaneetham D, Lei S, Maus ADJ. Neuronal Nicotinic Receptors in Non-Neuronal Cells: New Mediators of Tobacco Toxicity? Eur J Pharmacol 2000;393(1-3):279-94. doi:10.1016/S0014-2999(00)00036-4
5. Diegelmann RF. Wound Healing: An Overview of Acute, Fibrotic and Delayed Healing. Front Biosci 2004;9:283. doi:10.2741/1184
6. Dussor GO, Leong AS, Gracia NB, Kilo S, Price TJ, Hargreaves KM, Flores CM. Potentiation of Evoked Calcitonin Gene-Related Peptide Release from Oral Mucosa: A Potential Basis for the Pro-Inflammatory Effects of Nicotine. Eur J Neurosci 2003;18(9):2515-26. doi:10.1046/j.1460-9568.2003.02935.x
7. Eliakim R, Karmeli F. Divergent Effects of Nicotine Administration on Cytokine Levels in Rat Small Bowel Mucosa, Colonic Mucosa, and Blood. Isr Med Assoc J 2003;5:178-80
8. Galiano RD, Tepper OM, Pelo CR, Bhatt KA, Callaghan M, Bastidas N, Bunting S, Steinmetz HG, Gurtner GC. Topical Vascular Endothelial Growth Factor Accelerates Diabetic Wound Healing through Increased Angiogenesis and by Mobilizing and Recruiting Bone Marrow-Derived Cells. Am J Pathol 2004;164(6):1935-47. doi:10.1016/S0002-9440(10)63754-6
9. Grando SA, Horton RM, Mauro TM, Kist DA, Lee TX, Dahl MV. Activation of Keratinocyte Nicotinic Cholinergic Receptors Stimulates Calcium Influx and Enhances Cell Differentiation. J Invest Dermatol 1996;107(3):412-8. doi:10.1111/1523-1747.ep12363399
10. Grando SA. Receptor-Mediated Action of Nicotine in Human Skin. Int J Dermatol 2001;40(11):691-3. doi:10.1046/j.1365-4362.2001.01293.x
11. Heeschen C, Jang JJ, Weis M, Pathak A, Kaji S, Hu RS, Tsao PS, Johnson FL, Cooke JP. Nicotine Stimulates Angiogenesis and Promotes Tumor Growth and Atherosclerosis. Nat Med 2001;7(7):833-9. doi:10.1038/89961
12. Jacobi J, Jang JJ, Sundram U, Dayoub H, Fajardo LF, Cooke JP. Nicotine Accelerates Angiogenesis and Wound Healing in Genetically Diabetic Mice. Am J Pathol 2002;161(1):97-104. doi:10.1016/S0002-9440(10)64161-2
13. Johnson GK, Organ CC. Prostaglandin E2 and Interleukin-1 Concentrations in Nicotine-Exposed Oral Keratinocyte Cultures. J Periodontal Res 1997;32(5):447-54. doi:10.1111/j.1600-0765.1997.tb00557.x
14. Kalamida D, Poulas K, Avramopoulou V, Fostieri E, Lagoumintzis G, Lazaridis K, Sideri A, Zouridakis M, Tzartos SJ. Muscle and Neuronal Nicotinic Acetylcholine Receptors.

FEBS J 2007;274(15):3799-845. doi:10.1111/j.1742-4658.2007.05935.x
15. Li XW, Wang H. Non-Neuronal Nicotinic Alpha 7 Receptor, a New Endothelial Target for Revascularization. Life Sci 2006;78(16):1863-70. doi:10.1016/j.lfs.2005.08.031
16. Martin JW, Mousa SS, Shaker O, Mousa SA. The Multiple Faces of Nicotine and Its Implications in Tissue and Wound Repair. Exp Dermatol 2009;18(6):497-505. doi:10.1111/j.1600-0625.2009.00854.x
17. McGehee DS, Role LW. Physiological Diversity of Nicotinic Acetylcholine Receptors Expressed by Vertebrate Neurons. Annu Rev Physiol 1995;57:521-46. doi:10.1146/annurev.ph.57.030195.002513
18. Misery L. Nicotine Effects on Skin: Are They Positive or Negative? Exp Dermatol 2004;13(11):665-70. doi:10.1111/j.0906-6705.2004.00274.x
19. Morimoto N, Takemoto S, Kawazoe T, Suzuki S. Nicotine at a Low Concentration Promotes Wound Healing. J Surg Res 2008;145(2):199-204. doi:10.1016/j.jss.2007.05.031
20. Ng MKC, Wu J, Chang E, Wang B, Katzenberg-Clark R, Ishii-Watabe A, Cooke JP. A Central Role for Nicotinic Cholinergic Regulation of Growth Factor-Induced Endothelial Cell Migration. Arterioscler Thromb Vasc Biol 2007;27(1):106-12. doi:10.1161/01.ATV.0000251517.98396.4a
21. Nguyen VT, Arredondo J, Chernyavsky AI, Kitajima Y, Grando SA. Keratinocyte Acetylcholine Receptors Regulate Cell Adhesion. Life Sci 2003;72(18-19):2081-5. doi:10.1016/S0024-3205(03)00087-0
22. Nguyen VT, Ndoye A, Hall LL, Zia S, Arredondo J, Chernyavsky AI, Kist DA, Zelickson BD, Lawry MA, Grando SA. Programmed Cell Death of Keratinocytes Culminates in Apoptotic Secretion of a Humectant upon Secretagogue Action of Acetylcholine. J Cell Sci 2001;114(6):1189-204. doi:10.1242/jcs.114.6.1189
23. Osborne-Hereford AV, Rogers SW, Gahring LC. Neuronal Nicotinic Alpha7 Receptors Modulate Inflammatory Cytokine Production in the Skin Following Ultraviolet Radiation. J Neuroimmunol 2008;193(1-2):130-9. doi:10.1016/j.jneuroim.2007.10.029
24. Picciotto MR, Kenny PJ. Mechanisms of Nicotine Addiction. Cold Spring Harb Perspect Med 2021;11(5). doi:10.1101/cshperspect.a039610
25. Sgard F, Charpantier E, Bertrand S, Walker N, Caput D, Graham D, Bertrand D, Besnard F. A Novel Human Nicotinic Receptor Subunit, α10, That Confers Functionality to the α9-Subunit. Mol Pharmacol 2002;61(1):150-9. doi:10.1124/mol.61.1.150
26. Sopori ML, Kozak W, Savage SM, Geng Y, Soszynski D, Kluger MJ, Perryman EK, Snow GE. Effect of Nicotine on the Immune System: Possible Regulation of Immune Responses by Central and Peripheral Mechanisms. Psychoneuroendocrinol 1998;23(3):189-204. doi:10.1016/S0306-4530(97)00076-0
27. Sugimoto A, Masuda H, Eguchi M, Iwaguro H, Tanabe T, Asahara T. Nicotine Enlivenment of Blood Flow Recovery Following Endothelial Progenitor Cell Transplantation into Ischemic Hindlimb. Stem Cells Dev 2007;16(4):649-56. doi:10.1089/scd.2007.9991
28. Wong LS, Martins-Green M. Firsthand Cigarette Smoke Alters Fibroblast Migration and Survival: Implications for Impaired Healing. Wound Repair Regen 2004;12(4):471-84. doi:10.1111/j.1067-1927.2004.12403.x