Targeting tumor necrosis factor alpha (TNF-a) in diabetic rats could approve avenues for an efficient strategy for diabetic therapy

Karolin K. Abdul-Aziz a, *, Muobarak J. Tuorkey b

a Zoology Department, Division of Physiology, Damanhour University, Egypt
b Medical Biochemistry, Faculty of Science, Damanhour University, Egypt

A B S T R A C T

Background: Several studies held belief that downregulation of TNF-a may be effective for preventing diabetes and its complications. However, it is not known whether TNF-a downregulation in long-term can generate any biological adverse.

Aim: The aim of the present study was to clarify what the impact is for such treatment with specific antibody for TNF-a on the other biological activities after 4 weeks.

Methods: Using western blot, IHC, Elisa, biochemical assays and scanning electron microscope.

Results: Results show that TNF-a, FOXO-1, IL-6 and MPO, when expressed in diabetic rats, collectively induce dramatic changes in diabetic rats. Since, TNF-a is involved in activation of transcription factor FOXO1 along with oxidative stress mediated by neutrophils. On one hand, IL-6 mediates neutrophils activation leading to an augmentation in stress mediators. And FOXO1 is activated in order to eliminate these oxidative mediators, on the other hand. Data show also that the prominent defect in mucosal IgA and IL-2 secretions may be the leading reasons for digestive atrophy. Finally, Akt-1 inhibits the cleavage of caspase 3, so, it could prevent the incidence of apoptosis.

Conclusion: Findings of this study reveal how TNF-a can be mechanistically coupled to greater diabetic complications potential.
Keywords:
Tumor necrosis factor a (TNF-a)
Protein kinase B (Akt)
Forkhead box O-1 (FOXO-1)
Myeloperoxidase (MPO)
Mucosa-associated lymphoid tissues (MALT)

References


ABSTRACT
The purpose of this review is to summarize the pertinent literature published in the present era regarding ulcerogenic effectors, and all available therapeutic concepts in this regard including; different physiological/pathological changes in response to H. pylori infection, nonsteroidal anti-inflammatory drugs (NSAID), bile acids, nitric oxide, copper complexes, acid pump inhibitors, histamine blockers, curcuminoids, cytokines and/or growth factors and finally probiotics. Because of the partial understanding of gastric ulcer pathogenesis three major hypotheses were strongly speculated and widely documented. Firstly, the hyperacidity hypothesis entailing the disturbance of the gastric acid, histamine, gastrin and somatostatin. Secondly, the eicosanoid imbalance hypothesis exploiting changes in the microcirculation through the vasoconstrictor eicosanoids such as TXA2 and vasodilator cytoprotectant eicosanoids such as PGE2. Thirdly, the infective hypothesis implementing H. pylori as the major pathogenic effector for the gastroduodenal ulceration. In fact, all of the previous effectors could be involved and possibly employing inflammatory/antiinflammatory, oxidative stress and/or angiogenic disturbance.
Keywords:

Oxidative stress, Non-steroidal anti-inflammatory drugs, entero- chromaffin-like (ECL) cells, Proton pump inhibitors (PPIs), Probiotics.

Publish in: Research Journal of Pharmaceutical, Biological and Chemical Sciences

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Bioelectrical Impedance as a Diagnostic Factor in the Clinical Practice and Prognostic Factor for Survival in Cancer Patients: Prediction, Accuracy and Reliability

Muobarak J Tuorkey*
Biology Department, Division of Physiology and Medical Biochemistry, Faculty of Science, Damanhour University, Egypt

Abstract

Bioimpedance analysis could provide a clear figure about changes in cells and tissues based on frequency-dependent changes, due to their electrical resistances for the applied electrical current. This review explains the physical principle of bioimpedance. And to monitor the progression of radiation induced tissue injury, particularly in radiotherapies. The interaction of radiation with the biological tissues and a prediction for their earlier and later alterations due to exposure are discussed. As well as, an overview for tissue identification by bioelectrical impedance analysis (BIA) is proposed. Bioimpedance analysis “applications and its limitations” in the health care, clinical practice and prognosis of overall survival in cancer patients are discussed.

Keywords: Phase Angle (PhA); Body Mass Index (BMI); Fat-Free Mass (FFM); Extracellular Fluid (ECF); Intracellular Fluid (ICF); Estimated Blood Loss (EBL)

Publish in:
Biosensors & Bioelectronics
Tuorkey, J Biosens Bioelectron 2012, 3:4
http://dx.doi.org/10.4172/2155-6210.1000121
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