Special issue for: Endocrine, Metabolic & Immune Disorders - Drug Targets

Pathogenic mechanisms and therapeutic implication in nickel-induced cell damage

Aims & Scope:

Hypersensititation to nickel is the main cause of allergic contact dermatitis (ADC) since this metal is largely present in industrial and consumer products. Quite importantly, nickel is able to bind to Toll-like receptor 4, a specific receptor for lipopolysaccharide of Gram-negative bacteria, thus inducing the transcription of genes encoding for pro-inflammatory cytokines. At dermal sites this event is the major pathogenic mechanism accounting for ADC.

In this special issue, several aspects of nickel-induced pathology will be covered, even including therapeutic attempts to attenuate tissue damage.

From an immunological point of view, in patients affected by nickel-ADC the involvement of both T helper (h)-1 and Th2 cells will be described along with a reduced function of T regulatory cells. In vitro polyphenols are able to modulate immune response in these patients increasing interleukin-10 production and decreasing nitric oxide release.

Another issue will be represented by the capacity of nickel to induce oxidative stress at tissue levels and the property of ascorbic acid to attenuate free radical production in ADC.

Also lungs and kidneys are potential targets of nickel toxicity and the relevant pathogenic mechanisms will be illustrated with the aim to define innovative treatments.

Finally, a severe complication of nickel exposure is its carcinogenic effects mainly base on DNA alteration.

Potential topics include, but are not limited to:

Epidemiology, pathogenenesis and clinical patterns

Mechanisms of nickel allergy

Mechanisms of carcinogenesis

Use of polyphenols for therapeutic purposes

Use of vitamin C as an anti-oxidant

Schedule:

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