The findings can serve a valuable educational and informative support for a community of specialists. The positive dynamics of the invention work results on the titanium nickelide application in medicine in Russia and abroad has been analyzed.

The article is admitted to the International Scientific Conference " Innovation technologies in medicine"; Spain (Costa Brava), 8-15th July, 2007г.; came to the editorial office on 09.11.07.

INFECTIONS AND ALLERGY

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The immune response to antigenic stimulation represents the outcome of an integrated network of cells and mediators as well as of complex genetic-environmental interactions. Accordingly, it is quite reasonable to expect that immune responses to allergens and to bacterial, viral or parasitic antigens can influence each other.

The Thl/Th2 paradigm certainly represents an important reading key for a better understanding of the links between infections and allergy, where many bacterial and viral antigens preferentially induce a Thl-type response whereas parasites and allergens preferentially elicit a Th2-type response. However, explaining epidemiological data of associations between allergic and infectious diseases on the basis of the Thl/Th2 paradigm only is quite simplistic because of the many confounding factors influencing the final clinical status of subjects in epidemiological surveys. In fact, individual immune responses in vivo to allergens and infectious agents may vary also depending on the type and dose of antigenic stimulation as well as on the time and site of immune experiences.

While the relationships between parasitic infections and allergy are still controversial, a negative association between some bacterial or viral infections and allergic diseases has been reported by several studies. These negative associations as well as data emerging from studies of cohorts of allergic subjects (socioeconomic status, size and birth order effect) as well as from studies in population samples with different lifestyle provide support to the "hygiene hypothesis" (Strachan, 1989) suggesting that the reduced exposure to infectious agents during early infancy might represent a major factor for a prevalent Th2 polarization and the observed increasing prevalence of allergic diseases.

However, extensive evidence has been accumulated both in prospective and retrospective studies indicating, on the contrary, that some respiratory viral infections in childhood (e.g. RSV infections) are associated with a higher prevalence of allergy and asthma later in life, although it is not clear whether viral infections favour sensitisation or whether allergic subjects who experience respiratory viral infections develop asthma more frequently than non atopics. Certainly, rhinovirus infections have been proven to be a major cause of exacerbations of wheezing and of hyper responsiveness of nasal and bronchial mucosa in both rhinitic and asthmatic patients. On the other hand, allergy can favour infections. In fact, ICAM-1 — a major receptor for human rhinovirus — is overexpressed in allergic inflammation, even in sub-clinical forms.

Our recent epidemiological study of exposure to food borne or orofecal microbes versus airborne viruses in relation to atopy and allergic asthma, might suggest a possible interpretation of the controversial issue whether infections favour or protect from allergy as well as of the inconsistencies impinging on the hygiene hypothesis. In fact, in out study food borne and orofecal exposure to microbes but not respiratory viral infections are associated with a lower prevalence of sensitisation and allergic diseases. Accordingly, the composition of the gut microflora or a high turnover of microbial products stimulating gastro-intestinal path, rather than infections diseases, might have a relevant role in protecting from atopy. Should this interpretation prove correct, mimicking a microbial education of the immune system might represent a new fascinating strategy to prevent allergic diseases and to revert the epidemic trend of atopy and allergic asthma.

The article is admitted to the International Scientific Conference "Present-day problems of experimental and clinical medicine", Thailand (Phuket), 19-27th December, 2007, came to the editorial office on 09.11.07.

ROLE XANTHINEOXIDASE IN PATHOGENESIS OF THE GOUT AND ARTHRITISES

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Gout is a disease barely investigated at the present time. We know its cause, uric acid, we know the enzyme that converts purines to uric acid, xanthineoxidase, and we have an inhibitor, allopurinol, which acts as an effective preventative agent. However, there are a series of unanswered questions outstanding. The first is why is the relationship between the plasma uric acid and the development of gout so poor? For instance, in chronic renal failure uric acid levels are very significantly elevated, but gout a relatively uncommon problem. Secondly, why is gout a joint disease and why does uric acid not precipitate everywhere else? The solubility of uric acid in synovial fluid is no greater than that of plasma. Perhaps the clearance of uric acid from the joint is the problem, but there are other membrane systems where one