

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.

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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 Th1/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 Th1-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 Th1/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 infec-

tions 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 hyperresponsiveness 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 our 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.

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ROLE XANTHINE OXIDASE 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, xanthine oxidase, 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

might imagine there could be difficulties, but no difficulties arise. Most patients with ischaemic limbs with a degree of renal failure experience no problems. An hypothesis is that the joint itself contributes to urate production making it an environment where super saturation can occur if plasma levels are elevated. If such a mechanism existed, the most likely explanation would be that the enzyme, xanthineoxidase, was up regulated somehow within the synovial membrane.

Our understanding of this enzyme has changed dramatically in the past few years. Firstly, it is now known that the conversion of purines to uric acid is only one of the functions of this redox centres enzyme. There are two other redox centres in the enzyme, one site, which is known to reduce oxygen to superoxide and a other site - an iron sulphur centre whose function is less clear. Recent studies have shown that the enzyme can serially reduce nitrates to nitrites, nitrites to nitric oxide and nitric oxide reacts with superoxide very rapidly to produce peroxynitrite. Nitric oxide, superoxide and peroxynitrite are powerful anti-bacterial systems. This raises the question - does the joint have a powerful anti-bacterial system and is the development of gout a reflection of this process?

Our interest in this area was stimulated when we took note of the fact that the levels of xanthineoxidase were very high in breast milk of lactating mammals (Stevens C.R. et al., 2000). A series of studies have now clearly demonstrated that the function of this enzyme is antibacterial and its purpose to protect the neonatal stomach and perhaps the lactating breast.

Now we shall consider infective and reactive arthropathies. Isolating intact organisms from the joint of patients with a bacteremia and septicemia is very difficult and indeed, most bacteraemic and septicemic illnesses are not associated with an infective arthritis. The synovial membrane - is a fragile structure with multiple vessels for the most part supported by fatty tissue, potentially easily traumatised and able to leak. This structure is necessary for the correct physiological function of the joint allowing cartilage, which is avascular, but metabolic active. It is therefore at risk, and the presence of an antibacterial system, especially for the joint, would minimise this.

The evidence is recently found that the synovial micro vessels have an enhanced capacity to generate reactive nitrogen species and preliminary evidence that isolated synovial endothelial cells have an enhanced capacity prepared with other micro vessels to produce xanthineoxidase. It allows to connect these observations, and suitable experiments to do conclusion. In this presentation, we use teleological centred arguments to develop this hypothesis utilizing examples from other species in the animal kingdom.

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DEVELOPMENT FEATURES OF DISCIRCULATORY ENCEPHALOPATHY IN PERSONS SUBJECTED TO IONIZING RADIATION

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Epidemiological surveys showed that in accident consequences liquidators (ACL) in Chernobyl Nuclear Power Plant vascular damages of the central nervous system take one of leading places in disease incidence structure being the main reason of disability, social maladjustment and mortality.

The investigation purpose is to study the features of chronic brain ischemia in persons subjected to radiation effect.

Materials and methods

An examination of 536 men aged from 39 to 60 years old having taken part in Chernobyl disaster clean-up in the period from 1986 to 1988 (the first group) was carried out. 436 men with burdened radiation induced anamnesis made the control group (CG). Both groups were representative in age, physical comorbidity, intensity of encephalitic semiotics for April, 1986. The patients' state evaluation was carried out on one diagnostic algorithm taking into account physical and neurologic state, radiological anamnesis, laboratory-instrumental investigation methods including brain CT, MRT.

The statistical treatment of the material was carried out with the help of the Biostat program. The Student's criteria were used for pair values; the differences were considered to be authentic at $p < 0,05$.

Research results

For the accident moment there were no cerebrovascular insufficiency signs registered in the ACL, in 20 persons (4,2%) of the CG there was semiotics of initial signs of cerebrovascular insufficiency (ISCI) observed. A year after the emergency works the clinical picture of cerebral circulation insufficiency became apparent in 108 liquidators, that is 2,2 times as more than in the CG patients (49 persons). Among them the ISCI in the ACL made 9,9% (53 persons), in CG patients - 5,6% (27 persons); clinical implications of discirculatory encephalopathy (DE) were detected in 10,2% of the ACL and 4,5% of the CG patients accordingly. In the ACL the organic manifestations of encephalitic pathology were represented by the I stage DE (8,9% of the cases) and the II stage DE (1,3% of the cases); in persons having been subjected to the radiation effect - only DE of the I stage (4,5%). For the following 3 years the DE was formed in 16,0% of the ACL: I stage DE - in 9,7% (52 persons), II stage DE - in 6,3% (34 persons); in every third liquidator (35,4%) the ISCI was registered. In patients without radiological anamnesis the DE was detected in 15,6% of the cases (75 persons): I stage DE - in 14,2%, II