<<2014考研英语题源阅读100篇>>

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章节摘录

版权页: immune system over-reactive to random allergens to the thought that chemicals in swimming pools are responsible. What these ideas have in common is the suggestion that some environmental change which accompanies economicdevelopment is the cause. A group of researchers led by Shadmehr Demehri of Washington University, in StLouis, believe these explanations are looking in the wrong place. Asthma is not, they think, caused directly byenvironmental factors. Rather, the link is indirect. The direct cause is a chemical distress signal produced in skinthat is damaged by another hazard of modem life: eczema. Eczema is also on the rise in the industrialised world, in the same sorts of countries where asthma is a problem. As they describe in Public Library of Science Biology, Dr. Demehri and his colleagues now believe they know whatcauses this link. The culprit is thymic stromal lymphopoietin (TSLP), a signalling molecule secreted by damaged skin cellswhich elicits a strong immune response from the body to fight off invaders. Dr. Demehri and his team hypothesisedthat eczema-induced TSLP enters the bloodstream and, when it arrives at the lungs, sensitises them so that theyreact to allergens that would not previously have bothered them. In other words, they become asthmatic. They tested their hypothesis in a series of experiments on mice. First, using genetic engineering, they createdmice prone to the kind of skin defects found in eczema. These mice were, as they hoped, susceptible to asthma. Then they used additional engineering to delete the gene for the receptor molecule which picks up TSLP in thelungs. These mice no longer developed asthma. Thirdly, they engineered mice to produce high levels of TSLP intheir skin in the absence of other skin problems. These mice also developed asthma. Taken together these experiments indicate -- at least in mice -- that skin damage creates susceptibility to asthmaby releasing TSLP. If that proves true in people, too, it suggests several ways asthma might be prevented. One isto take eczema seriously, and treat it early. The usual treatment is to apply steroids to the damaged skin, but there is evidence that some parents reject this treatment for their children. If a link between eczema and asthma wereproperly established, that reluctance would probably diminish. In the longer term, it might be possible to devisedrugs that inhibit the production of TSLP or interfere with TSLP-receptor molecules in the lungs. Better still, though, would be to work out what aspect of modern life causes eczema.

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