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Perspektívák

Gyermekkori védőoltások és az atópiás betegségek kialakulása



C Grüber

Levelezés:




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Kérjen engedélyeket

Bremner és munkatársai kommentárja a dolgozathoz (lásd 567. oldal)

Az atópia fokozott kockázatának kitett gyermekek szülei gyakran aggódnak a korai immunizálás miatt. A vakcina antigénjeivel vagy magukkal a szennyező anyagokkal szembeni ritka allergiás reakciókkal kapcsolatos aggodalmak mellett (Grüber és munkatársai ¹), fennáll az a félelem, hogy az immunizálás elősegítheti az atópiás betegségek kialakulását, ami e gyermekek késleltetett vagy hiányos beoltásához vezethet. Az immunizálás és az atópiás betegség kapcsolatáról szóló egyes jelentések szították ezt a félelmet.

Ezen túlmenően, az allergiás megbetegedések növekvő előfordulása számos ipari országban a higiéniai normák javulásával jár együtt. Úgy gondolják, hogy a mikrobiális ingerek hiánya késlelteti a magzati Th2 ferde immunrendszerből az iskolás gyermekek Th1 kiegyensúlyozottabb immunrendszere felé történő érését, és így a gyermekeket fogékonyabbá teszi a Th2-függő allergiás betegségekre. Ebben az összefüggésben a kora gyermekkori védőoltásokat az atópia kialakulásának elősegítőjének tekintették, akár közvetlenül Th2-típusú immunválaszt kiváltó szerek beadásával, akár közvetetten olyan fertőzések megelőzésével, amelyek egyébként preferenciális Th1-típusú immunválaszt indukálnának, és így elferdítené a citokin egyensúlyt az atópiától. ²

What is the currently available evidence for an atopy promoting effect of early childhood immunisations? An IgE response to vaccine antigens is commonly detectable in the sera of vaccinated children. About 50% of infants have detectable IgE against diphtheria/tetanus after primary vaccination,³ and after booster vaccination later in life more than 90% of vaccinees have detectable IgE against the vaccine antigens.⁴ The IgE response to vaccine antigens seems to be more pronounced among atopic individuals,^{3,5} but the correlation of IgE and protective IgG against the vaccine antigens is poor.^{6,7} IgE formation against ...

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Competing interests: none declared

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Perspectives

Childhood immunisations and the development of atopic disease



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Commentary on the paper by Bremner *et al* (see page 567)

Parents of children at heightened risk for atopy are frequently concerned about early immunisations. Apart from concerns about rare allergic reactions to the vaccine antigens or contaminants themselves (reviewed in Grüber and colleagues¹), there exists a fear that immunisations may promote the development of atopic disease, leading to delayed or incomplete vaccination of these children. Some reports about an association of immunisation and atopic disease have fuelled this fear.

Moreover, the rising prevalence of allergic diseases in many industrialised countries has been associated with improvement in hygiene standards. It is thought that a lack of microbial stimuli delays the maturation from the fetal Th2 skewed immune system towards the more Th1 balanced immune system of the school child, and thus renders children more susceptible to Th2 dependent allergic disease. In this context, early childhood vaccinations have been viewed as a promoter of atopy development, either directly by the administration of agents which induce a Th2-type immune response or indirectly by the prevention of infections which otherwise would induce a preferential Th1-type immune response, and would thus skew the cytokine balance away from atopy.²

What is the currently available evidence for an atopy promoting effect of early childhood immunisations? An IgE response to vaccine antigens is commonly detectable in the sera of vaccinated children. About 50% of infants have detectable IgE against diphtheria/tetanus after primary vaccination,³ and after booster vaccination later in life more than 90% of vaccinees have detectable IgE against the vaccine antigens.⁴ The IgE response to vaccine antigens seems to be more pronounced among atopic individuals,^{3,5} but the correlation of IgE and protective IgG against the vaccine antigens is poor.^{6,7} IgE formation against ...

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