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Immunosenescence and Vaccination

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Abstract

The problems associated with the ageing immune system and vaccination were discussed recently at an international workshop at the Jenner Institute for Vaccine Research, Compton, UK, 6–7 October, 2005. This is a commentary on that session. The meeting included discussions on T and B cell differentiation and ageing, as well as dendritic cell and neutrophil data, with the emphasis on T cell immunosenescence, perceived as the most important hindrance to satisfactory responses to vaccines in the elderly. The main questions to be addressed in this context are the reasons for dysfunctionality of T cells in the elderly and what to do to improve T cell function. Several of the major reasons for poor T cell responses in the elderly were discussed; however, many important questions remain: The next meeting at the Jenner Institute may already be able to provide some of the answers to these questions, which have serious implications for public health issues in increasingly elderly populations.

Introduction

An international workshop on Immunosenescence and Vaccination was organised at the Jenner Institute for Vaccine Research, Compton, UK, 6–7 October, 2005 by Peter Beverley (Jenner Institute), Arne Akbar (University College, London, UK) and Don Palmer (Royal Veterinary College, London, UK). The most profound clinical impact of age on the immune system concerns the response of the elderly to vaccination. The meeting included discussions on T and B cell differentiation and ageing, as well as dendritic cell and neutrophil data, with the emphasis on T cell immunosenescence, perceived as the most important hindrance to satisfactory responses to vaccines in the elderly. The main questions to be addressed in this context are the reasons for dysfunctionality of T cells in the elderly and what to do to improve T cell function.

What is the problem?

Several of the major reasons for poor T cell responses in the elderly were discussed: is it a problem of stem cells with decreased potential for differentiation into naïve T cells? Is it a problem of the thymic environment not supporting this differentiation sufficiently any more? Is it a problem of naïve T cell ageing in the periphery and/or memory cell ageing and loss? The answers as they are emerging of course suggest that all these problems, and more, apply, as outlined below.

Diana Wallace and Peter Beverley [1] at the Jenner Institute, together with Derek Macallan in London, measured T cell turnover during human ageing by assessing the rate of uptake of deuterated glucose into DNA and its dilution with cell division. In healthy young people, CD45RO+ cells divide more rapidly than CD45RA+ cells in both the CD4 and CD8 subsets, whereby CD45RO+ cells have a 26 day half-life, with 2.7% of cells dividing every day,