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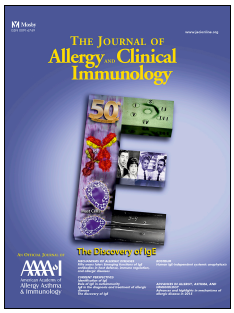

Drug hypersensitivity: we need to do more

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Editorial

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Drug hypersensitivity: we need to do more

Drug hypersensitivity reactions (DHR) are drug induced immune and/or inflammatory reactions. They go beyond classical IgE-mediated reactions like anaphylaxis and comprise many T cell mediated reactions, which altogether are likely more common than IgE reactions. DHR are an important clinical issue and are frequent even if DHR to a single drug may be rare. The clinician is usually confronted with a particular clinical situation and has to elucidate which drug may be involved. This process is difficult in a useful time span – which makes clinical experience a valuable pillar of decision making. However, experience without a sound understanding of the underlying mechanism means to stand on shaky ground. Thus, DHR research is needed, as repeatedly emphasized[1,2].

Sullivan et al[3] uses piperacillin as a model for β-lactam hypersensitivity. They study the nature of the piperacillin specific T-cell response isolated from blood and from positive patch tests of hypersensitive patients and also include T cell clones (TCC) from healthy volunteers. By analyzing a high number of piperacillin specific TCC (474 from blood and 96 from positive skin tests) they found that a majority of the TCC secreted significant amounts of the cytotoxic molecules perforin, granzyme B and FasL, which confirmed the cytotoxic features of drug reacting CD4+ T cells as described previously. Novel is the high secretion of IL22 by piperacillin specific TCC, which could be detected mainly in combination with IFNγ or IL13 in CD4+ as well as in CD8+ TCC. Remarkably, IL17 was totally absent from the observed cytokine secretion profile of piperacillin specific TCC, revealing the Th22 phenotype of such cells.

The role of IL22 in the context of drug hypersensitivity should be further investigated. It is still unclear whether IL22 belongs to the specific characteristics of drug reacting T cells and if IL22 drives drug reacting T cells to affect particularly the skin. IL22 production has also been described in atopic dermatitis or psoriasis[4]. The IL22 receptor is expressed as a heterodimer only on non-immune cells and mainly on epithelial cells. It may act pro-inflammatory by inducing several defensins, chemokines and