



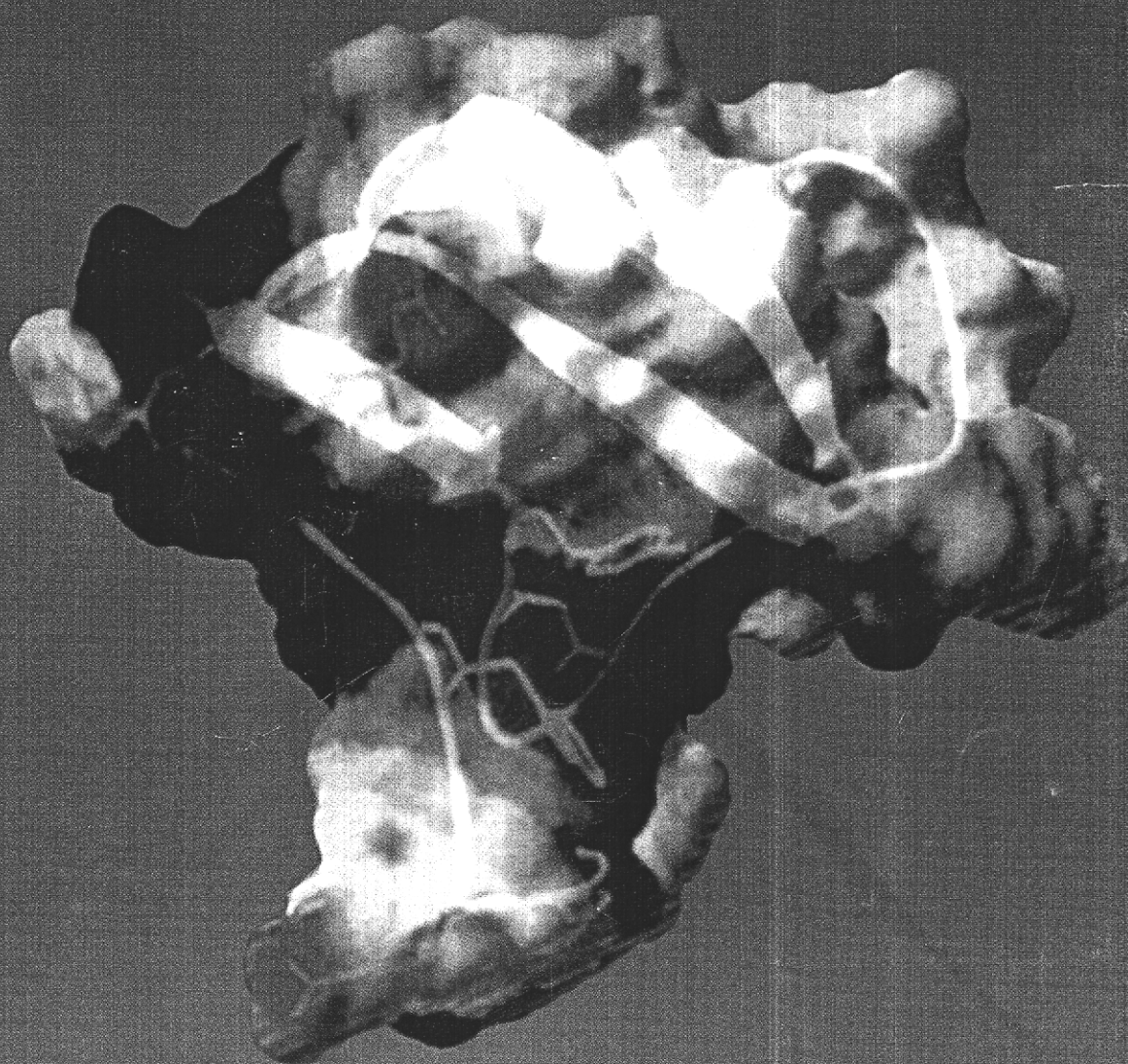
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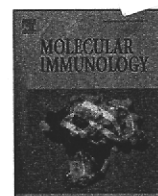
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Review

Inflammation at the interface of Innate and Acquired Immunity

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ABSTRACT

This is the short summary of the presentation at the 2nd Belgrade Meeting on Immunoregulation entitled "Inflammation at the interface of Innate and Acquired Immunity" held recently under the auspice of European Federation of Immunological Societies and organized by Medical School, University of Kragujevac.

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The main theme of the second EFIS/EJI Belgrade Symposium, on Inflammation at the interface of Innate and Acquired Immunity (7–10 September, 2008) meeting was on immunoregulation in autoimmune and inflammatory disease. Below we summarize the new findings reported by the invited and selected participants.

1. Cells, cytokines and their receptors

IL-33, a newly discovered cytokine related to IL-1, turns out to be a ligand for the hitherto orphan ST2 receptor expressed on some Th2 cells and on mast cells. On the latter it joins the Fc-epsilon and chemokine receptors in regulating degranulation (Foo Liew, Glasgow, UK). ST2 and IL-33 both show promise in animal models of atherosclerosis and arthritis, although IL-33 could also presumably enhance disease via its degranulating effect. Liew also described a second novel cytokine, the IL-12-related IL-35. IL-35 expands FoxP3 suppressor cells and suppresses Th17 cell development, and attenuates collagen-induced arthritis. Another IL-1 family member, IL-1F7, binds to the IL-18 receptor alpha chain (IL-18RA) and elicits an anti- rather the pro-inflammatory response expected of a member of the IL-1 family (Charles Dinarello, Denver, USA). The IL-1 ligand family now has 11 members, and at least one inhibitory ligand now recognised as an anti-inflammatory drug – tested in many inflammatory conditions including gout. It is worth noting that IL-1 is the only cytokine known to be able to convert an other-

wise tolerance-inducing regimen into an immune response. Marija Mostarica Stojkovic (Belgrade, Serbia) found evidence of synergy between IL-17- and IFN- γ -producing cells the EAE model. By means of IL-18 gene knockout Nada Pejnovic (London, UK) discovered a role for Th17 cells in atherosclerosis.

TNF blockers are attracting much interest in clinical use. Sergei Nedospasov (Berlin, Germany) exploited an impressive collection of TNF ablation mouse models to better define the mode-of-action of the range of these therapeutic agents currently used in human autoimmune disease. Similarly, Nikola Vujanovic (Pittsburgh, USA) distinguishes between soluble and membrane-bound and addresses their respective roles in inflammation and immunity.

Sergio Romagnani (Florence, Italy) described the extensive use of T-cell clones to define expression of receptors and cytokines in T-cell subsets. A landscape begins to emerge in which the subsets relate to well defined disease entities. Matthias von Herrath (La Jolla, USA) compared the activity of standard T-regs (FoxP3+) and IL-10+ T-regs, finding the later particularly potent in a T1D model. These cells can also have a deleterious effect, in prolonging virus infection. Milica Vukmanovic-Stejic (London, UK) traced the accumulation of T-regs during the Mantoux reaction (recall reaction to intradermal tuberculin) in man. Ivana Stojanovic (Belgrade, Serbia) finds that MIF (migration inhibitory factor) acts upstream of other proinflammatory cytokines in several models of inflammation.

2. Anti-cancer immunity

Olivera Finn (Pittsburgh, USA) added to these receptors a growing list of cancer-related surface macromolecules. These offer possible candidates for anti-cancer vaccines, including just possibly a universal anti-cancer vaccine. Lazar Vujanovic (Pittsburgh, USA) finds

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