1. The Nobel Prize in Physiology or Medicine in 2011 awarded three scientists, Bruce A. Beutler, Jules A. Hoffmann, and Ralph M. Steinman. Please describe their major scientific findings (15%).

2. Please compare the features of innate immunity versus adaptive immunity (15%).

3. Please describe the mechanisms for generating antibody diversity (20%).

4. Describe the requirements for the activation of naive B cells by a T-dependent antigen. By what mechanisms do T cells provide help to B cells in the humoral response? (10%)

5. List the different ways in which viruses can evade the immune system. (10%)

6. Based on your opinion, describe an important unresolved question in the interaction between host and immunity, and what will be the possible approaches to solve it. (10%)

7. From the following abstract, please describe what you know about:
(a) inflammatory response;
(b) the mechanisms of Foxp3+ iTreg cells;
(c) what is the new mechanism of Foxp3+ iTreg discovered in this study. (20%)

Foxp3+-Inducible Regulatory T Cells Suppress Endothelial Activation and Leukocyte Recruitment (*Journal of Immunology,* 2011, 187: 3521–3529)

The ability of regulatory T cells (Treg) to traffic to sites of inflammation supports their role in controlling immune responses. This feature supports the idea that adoptive transfer of in vitro expanded human Treg could be used for treatment of immune/inflammatory diseases. However, the migratory behavior of Treg, as well as their direct influence at the site of inflammation, remains poorly understood. To explore the possibility that Treg may have direct anti-inflammatory influences on tissues, independent of their well-established suppressive effects on lymphocytes, we studied the adhesive interactions between mouse Treg and endothelial cells, as well as

(背面仍有題目,請繼續作答)

their influence on endothelial function during acute inflammation. We show that Foxp3+ adaptive/inducible Treg (iTreg), but not naturally occurring Treg, efficiently interact with endothelial selectins and transmigrate through endothelial monolayers in vitro. In response to activation by endothelial Ag presentation or immobilized anti-CD3ε, Foxp3+ iTreg suppressed TNF-α– and IL-1β–mediated endothelial selectin expression and adhesiveness to effector T cells. This suppression was contact independent, rapid acting, and mediated by TGF-β–induced activin receptor-like kinase 5 (ALK-5) signaling in endothelial cells. In addition, Foxp3+ iTreg adhered to inflamed endothelium in vivo, and their secretion products blocked acute inflammation in a model of peritonitis. These data support the concept that Foxp3+ iTreg help to regulate inflammation independently of their influence on effector T cells by direct suppression of endothelial activation and leukocyte recruitment.