commendations of the ICRP and the scientific data.

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Declining support for Imanishi

SIR—Last year, Halstead criticized Imanishi's evolutionary theory saying that "Imanishi is not simply a popular writer. In Japan he is often held up as an intellectual giant equivalent to Charles Darwin"[1]. Subsequently, two letters from Japanese scientists, Sibatani[2] and Nakahara, *et al.*[3], seem to praise Imanishi and his evolutionary theory. We fear that readers will receive the erroneous impression that many Japanese scientists oppose darwinian theory and agree with Imanishi's theory, whereas the truth is that the influence of Imanishi and his theory are declining in Japan.

Imanishi greatly influenced Japanese primatologists at one time. Under his supervision, many creative studies were carried out on the social systems of Japanese macaques in the 1950s and 1960s. But times have changed. From 1983 to 1986, a large-scale project on optimal strategy and social structure was sponsored by the Ministry of Education, Science and Culture. Most of the ecologists working within it did so within a darwinian framework. And at the international symposium marking the end of the project, only a few scientists seemed to disagree with darwinian theory and support Imanishi.

Recently, Kishi[4] and Kawata[5,6] have pointed out that Imanishi's theory slowed the development of darwinian theory in Japan. The "Imanishi school" is still active, especially in primate ecology and sociology. Although Japanese primatologists, including us, are still hampered by the influence of Imanishiism, we criticize his theory and try to eliminate its negative influence[7,8].

We argue that Imanishi's description of phenomena is not incompatible with darwinian theory that deals with mechanisms of evolution. Although Imanishi himself considers his theory to be "anti-Darwinism", his view is completely misleading[5–7]; his theory is but a part of darwinian theory. We think that Imanishi's theory should be reconstructed within a darwinian framework, thereby contributing to the development of theories of mechanisms of evolution[7,8]. Undoubtedly it is nonsense simply to say, "Darwinian theory is bad", or, "Imanishi's theory is nonsense". To develop scientific theories, it is important to consider and discuss theories and opinions without dogmatism.

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Glucagon receptor number and the MHC

SIR—The recent report of glucagon activation of both cyclic AMP and inositol phosphate pathways by Wakelam *et al.* was interpreted in terms of two different glucagon receptors[1]. Alternatively, there could be one glucagon receptor molecule with differing association constants for glucagon (or its analogue, TH-glucagon[1]) determined by regulatory proteins which also direct whether adenylate cyclase or inositol phosphate breakdown will be stimulated. The genetic effects of the major histocompatibility complex (MHC) class I antigens on both adenylate cyclase and membrane phospholipid pathways seems more compatible with the hypothesis of one receptor protein.

Originally, the mouse MHC (H–2) was found to influence liver cAMP levels and glucagon binding to hepatocyte membranes[2,3]. More recently, the genomic region encoding H–2 has also been found to influence membrane methyltransferase I, which synthesizes phosphatidylmonomethylethanolamine from phosphatidylethanolamine[4]. This methylation consumes phosphatidylethanolamine, depleting the precursor pool, diacylglycerol, and presumably decreasing the amount of diacylglycerol available for phosphatidylinositol synthesis. The finding that there is an inverse relationship between mouse liver cAMP levels and membrane methyltransferase I activity[4] (both H–2 influenced) suggests that the MHC affects the two second messenger pathways differently. It is controversial whether coupling of membrane methyltransferases to hormone receptors occurs[5] but associations of the class I antigen complexes with insulin receptors[6] and platelet endoperoxide-thromboxane receptors[7] have been described. The same type of mechanism could be involved in MHC influences on β-adrenergic receptors in man[8].

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Monoclonals and marrow transplants

SIR—E. Donnall Thomas's News and Views article[1] on our paper[2] may have unintentionally created a misunderstanding about the way in which we suggest monoclonal antibodies could be used to prevent graft-versus-host disease and rejection in bone marrow transplantation. We did not wish to advocate monoclonal antibody treatment nor thymectomy of the donor. Indeed we have been involved in extensive trials using a complement fixing antibody CAMPATH-1 to purge T cells from donor marrow[3]. In the experimental model we described thymectomy and T-cell depletion of the donor as a convenient way of providing a marrow source purged of T-lymphocytes. May we also add that our own preliminary studies using monoclonal antibody therapy to prevent marrow rejection in man (CAMPATH-1 and CAMPATH-2) have been reported[4].

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