compromise between the classification of Linnaeus, in which all species were lumped under *Lepas*, and a current system[2,3]; namely, return to Darwin's classification of 1851–54 (ref. 4) and limit formal appellation of subsequently recognized species–group taxa to the subgeneric level.

It is possible, for some taxonomic reason or another, that a current genus might be more appropriately recognized as a subgenus. But it does not take a monkey wrench thrown into the gears of an entire system, as Crisp and Fogg have proposed, to enact such changes; when fine tuning is necessary, it can be made on an individual basis. No precedent would be set for, after all, "the despised" subgeneric category is still in use among the barnacles. Therefore I would like to offer the following rejoinder.

Darwin's attempts to estimate relationships between taxa, and concomitantly their patterns of diversity, were clearly hindered by taxonomic inadequacies and perceptions prevailing at the time. Many biological species were lumped as single species and, concomitantly, many now well recognized genera were lumped as single genera. Of *Balanus concavus* he wrote[4], ". . .a good instance of the amount of variation which seems especially to occur in most of the species which have very extensive ranges." But this and the half dozen other 'variable' species he recognized have all turned out to represent several to numerous species[5–7]. Thus, with their diversity and relationships poorly understood, it is no wonder Darwin found barnacle biogeography of "no particular interest, for the species are not sufficiently numerous; and, what is still more adverse, the genera, with unimportant exceptions, range all over the world; so that there is no scale of difference, and it cannot be said that these two regions differ in their species".

Thus knowledge of much of the diversity and our perception of the biogeography of barnacles languished for the more than a century after Darwin, despite the results of numerous expeditions and collections from deep as well as shallow water, and the advances in our understanding of the species concept and ecology. It took major systematic revisions to reach the point where barnacles became biogeographically interesting, and then to where they began not only to corroborate but to elucidate general principles in marine biogeography[8–11].

What Crisp and Fogg fail to appreciate is that taxonomic recognition of the relationships between species and species groups, at and above the generic level, is necessary if evolutionary pathways and patterns are to be recognized. Continuing adjustments to such a system, usually enacted when knowledge has increased to the point where relationships are no longer being accurately portrayed, are inevitable inconveniences. If someone eventually comes up with a better method of handling such a database, fine, but in the meantime the remedy of compromise proposed by Crisp and Fogg[1] does not satisfy these prerequisites.

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Antigen presentation by B cells

SIR—The recent letter of Lassila *et al.*[1] clearly demonstrates that B cells cannot be the antigen-presenting cells that lead to priming of helper T cells. This contrasts with data from several laboratories[2–4] demonstrating that B cells are required for the priming of $CD4^+$, class II MHC-restricted T cells that proliferate in response to antigen as discussed by De Franco[5]. We would like to offer the following explanation for these apparently conflicting results.

CD4 T cells belong to two separate subsets that differ in their functions[6–9] and in their requirements for priming[2–4,8–10] and accessory factors[11]. One subset of CD4 T cells (helper T cells, or Th2) is effective in helping B cells make antibody, while the reciprocal subset of CD4 T cells (inflammatory T cells, Th1) is active in T-cell proliferation assays, cytolysis, macrophage activation, delayed type hypersensitivity and in fact will suppress B-cell responses[12,13].

Studies in B-cell-deficient mice indicate that B cells are required for the priming of the subset of CD4 T cells that proliferate in response to antigen[2–4], but are not required for the priming of the subset of CD4 T cells that help B cells secrete antibody[8–10]. The data also demonstrate that the B cell required for priming the proliferating CD4 T cell must be antigen-specific and MHC matched to the CD4 T cell[4].

By contrast, the clonal expansion of the Th2 subset requires IL-1[10,11], a molecule expressed abundantly on macrophage membranes but not on B cells or dendritic cells. Thus, the macrophage is almost certain to be the cell that presents antigen: self MHC complexes required for the priming of this CD4 T cell subset[8,9].

Given these facts, it is not surprising that in the experiment of Lassila *et al.* the subset of CD4 T cells that helps B cells could not be primed in the absence of syngeneic macrophages. However, this observation cannot be taken to mean that no priming of CD4 T cells has occurred. Had they measured priming of the CD4 T cell subset that participates in *in vitro* proliferative responses, one would expect such priming to have occurred. Likewise, the seemingly paradoxical observation that transgenic mice fail to make antibody responses if they have B-cell but not macrophage class II MHC expression[14] is probably due to the requirement for macrophages in helper T-cell priming. Finally, if Th1 tend to be suppressive of antibody production, the problem of avoiding the priming of helper T cells by B cell idiotypes, on which the Lasilla *et al.* letter is based, may be moot.

The paper of Lassila *et al.* is an elegant demonstration of the fact that B cells are ineffective at priming those CD4 T cells that can help B cells. It will, however, require further study to determine whether this inability of B cells to prime CD4 helper T cells applies to all CD4 T cell subsets. Unlike the virgin CD4 T cells of Lassila *et al.*, we are certainly turned on by the idea that macrophages are required only to activate the subset of CD4 T cells that is most effective at helping B cells make antibody, while B cells almost certainly can prime those CD4 T cells that mediate inflammatory responses such as cytotoxicity, macrophage activation, or delayed-type hypersensitivity, as well as suppression of antibody responses[9,12,13].

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Scientific Correspondence

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