Role of HTLV III/LAV envelope protein

SIR—Sodrosky *et al.*[1] recently reported that the envelope protein of HTLV III/LAV, the virus that causes AIDS (acquired immune deficiency syndrome), might cause the lysis not only of infected T-helper cells (T-4), but also of uninfected T-4 cells. This would explain the total depletion of T-4 cells, even though only some of them are infected with the virus. I would like to draw attention to another possible role for the protein in the pathophysiology of AIDS.

Developmental stages of AIDS are characterized by a selective major histocompatibility complex II (MHC II) restricted immunodeficiency which shows a striking resemblance to class I graft-versus-host immunodeficiency in mice[2,3]. This immunodeficiency is immunologically mediated by parental T-4 cell recognition of class II MHC alloantigens on the host's B-lymphocytes and results in a polyclonal proliferation of these B-cells and the formation of autoantibodies against a variety of polymeric antigens and also against envelope proteins of murine leukaemia virus bound on kidney cells[4]. These autoimmune processes lead to different pathophysiological symptoms, such as the selective immune deficit and autoimmune glomerulonephritis[4].

In the case of AIDS these antibodies might be especially focused on the envelope protein of HTLV III/LAV on T-4 cells. The antibodies against the envelope protein might initiate an autoimmune destruction of T-4 cells so that a vicious circle between T-4-cell immunodeficiency and T-4 directed autoimmunity can develop. This autoimmune destruction of T-helper cells might synergistically amplify the direct T-helper cell impairment by LAV/HTLV III and its envelope protein.

It is noteworthy that other autoimmune phenomena in patients with AIDS also show a striking resemblance to those in class II graft-versus-host disease in mice[4] and some human connective tissue diseases[5].

I suspect that not only classical cellular and antibody-mediated cytotoxicity[6] but also polyclonally activated transformed B cells might play a major role in the pathophysiology of AIDS. Strategies which inhibit this polyclonal B-cell profliferation might therefore delay T-4 deletion in early AIDS.

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What is an extended probability?

SIR—While grateful for your open-minded discussion[1] of my review of extended probabilities[2], I should correct one minor point. I introduced the term 'extended probability' not (only) to avoid the charge of sensation-mongering but because probabilities exceeding unity are closely connected to, and in fact a consequence of, negative probabilities. Hence, the former have to be taken into account — if the latter "exist".

I confess my desire that somebody should give meaning to the term extended probability, but considering that Leonardo of Pisa in the thirteenth century was the first to interpret negative solutions of algebraic equations as debts, and even Descartes in the seventeenth century called them "false solutions" (the common term at that time), I do not believe this will happen in the near future. I have to disagree with the definition of negative probability given by D.E. Parry[3]. If, for instance, extended probabilities are used to obtain a formal resolution of the Einstein–Podolsky–Rosen paradox[4] they have to obey the following rule: an event leading with negative probability to a certain result has to extinguish not only the observation of a (hopefully) preceding exhibition of this result, but has also to extinguish the observation of this event at all, that is it has to reduce the number of accumulated events. (This is why a negative probability cannot be interpreted as a positive probability for observing some extinguishing counter-result, but only as a positive probability for the reduction of the number of preceding events.) And, of course, extended probabilities can only then creep into the picture when we deal with sets of indistinguishable results, as in the domain of quantum mechanics.

Unfortunately, no known physical mechanism allows for this kind of interaction or, therefore, provides a local and realistic resolution of the Einstein–Podolsky–Rosen paradox in terms of extended probabilities. Thus, the paradox will presumably continue to bother 'realistic' physicists, perhaps until this species becomes extinct.

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Migration and hominid bipedalism

SIR—Discoveries in palaeoanthropology have taken the story of hominid evolution back to a time just subsequent to the evolution of bipedalism[1]. The outstanding evolutionary question now is: what was the selection pressure that produced bipedalism? The current explanation[2] suggests that bipedalism evolved when the gathering of plants for food became necessary in a drier savannah habitat, and arms were needed to carry food back to a home site. Although this idea is feasible is it not compelling. We suggest a new idea in which bipedalism developed for long distance migration to scavenge from migrating ungulate populations; the only population that could provide sufficient food by scavenging. Bipedalism was a necessary adaptation to exploit this food supply.

In Serengeti, Tanzania, several ungulate species perform long-distance annual migration[3]. Migration provides an abundant and constant food supply, and hence these populations are large compared to sedentary populations of the same species. Mammal predators and scavengers do not follow the migrations because their young are slow growing and cannot travel with adults. Consequently breeding predators are sedentary. If a scavenger could migrate it would have access to an abundant and constant supply of carcasses (at least 1 carcass per 20 km^2 per day)[3], an order of magnitude greater than in non-migratory systems. In fact, humans can find enough to eat by following the wildebeest migration on foot[4]. Almost 70 per cent of carcasses are the result of undernutrition[3,5], so hominids would not be displacing predators from their kills.

Two vulture species follow the migrating ungulates, and they are considerably more numerous than sedentary species[5]. Vultures, however, have the disadvantage that they cannot easily break through the skin of intact carcasses. Therefore, there is in Africa an unfilled niche for a mammalian scavenger that can follow migrating ungulates; but such a mammal would need to carry its young.

We suggest that the selection pressure for the evolution of bipedalism in early hominids was access to this new rich and constant food supply. Morphs that had developed the ability to walk long distances would increase rapidly in number and displace the less numerous sedentary quadrupedal types dependent on plant gathering.

We envisage the protohominid quadruped as a plant gatherer and occasional scavenger, much like the baboon. For this type to follow migrating ungulates two essential adaptations are required simultaneously. First, members of the type must carry their young efficiently. This requires arms for carrying, and an upright stance. Chimpanzee females carry their young for at least the first year, so do humans, and thus it is reasonable to assume that early hominids also did so. Pro-