tion. It was signed by 44 eminent scientists in the field. The specific recommendation was to limit xenotransplantation to pig donor tissue and not to pursue other cell sources such as nonhuman primates. Interestingly, those scientists, including the senior author on the Patience *et al.* paper and the author of the accompanying News & Views piece, were also members of the group that recommended careful, limited but continued use of pig tissue in xenotransplantation trials.

More basic research is needed to responsibly and scientifically examine any potential benefits or safety risks of neural xenotransplantation compared with continued use of human fetal or adult cell donors.

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The Editor notes — Although clearly addressing different aspects of transplantation, the two articles[1,2] had in common the theme of pig-to-human xenotransplantation. It therefore seemed perfectly appropriate to draw to the attention of readers this commonality. The accompanying News & Views article, however, was only designed to address the precise consequences of the pig retrovirus infection of human cells and did not comment on the Parkinson's disease treatment paper.

Patience et al. *reply* — Schumacher and Isacson consider our use of the term pig (or porcine) endogenous retroviral (PERV) genome, misleading and suggest instead the abbreviation PERS, where the S means sequences. We have no objection to PERS but we are inclined to continue to use PERV for the following reasons: PERV is the porcine equivalent of human endogenous retroviral (HERV) genomes and the term HERV has become standard nomenclature among virologists and geneticists. Second, we showed that the porcine retrovirus infecting human cells contains *pol* sequences indistinguishable from those found in normal pig DNA. We therefore think that perhaps Schumacher and Isacson misunderstood the nature of pig endogenous C-type viruses. While many, and probably the great majority, of this subset of PERV genomes may be defective, as we explained in the Discussion section of our paper, some of them give rise to complete and infectious retroviruses. As Allan said in his News & Views article[3], the news is that PERV-PK is replication-competent for human cells.

Schumacher and Isacson may be unaware that pig C-type retroviruses have been associated with leukemia[7], and their statement that endogenous retroviral sequences are usually untranslated is misleading. Endogenous retroviral genes were first discovered through their expressed phenotypes, including complementation of defective, exogenous retroviruses, and several are translated, for example, in human tissues[8,9]. There is ample evidence, cited in our paper and previously[10], that mouse and cat endogenous retroviral genomes infect human tumor xenografts, although sharing our homes with these species has not led to zoonosis. The *Boston Globe* may well be right in predicting the existence of "viruses" in all pig cells. Complete proviruses exist in normal chickens, cats, baboons and several strains of mice[11]. At least we all agree that more basic research is needed.

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Animal models of psoriasis

To the editor — When SCID/SCID mice were reconstituted with minor histocompatibility mismatched naive CD4$^+$ T cells, a skin rash developed and was labeled "psoriasis-like"[1]. This approach differs from other SCID mouse-based psoriasis models employing human skin xenografts combined with autologous immunocompetent cells[2,3]. We urge caution in using labels like psoriasiform or psoriasis-like when interpreting data from animal models that have key etiological, genetic, pathophysiological and mechanistic implications[3]. In our opinion, psoriasis is a specific human disease with distinctive histological hallmarks including confluent parakeratosis, loss of granular cell layer, epidermal hyperplasia with elongated rete pegs, influx of memory T cells, and prominent dermal vessels. This latest mouse model[1], lacked almost all of these characteristics, having focal rather than confluent parakeratosis, accentuated granular cell layer, no elongation of rete pegs, and an influx of naive T cells. In addition to histological differences, there were other discrepancies. Some characteristics of the mouse condition are absent from human patients, including a wasting syndrome, colonic inflammation and splenomegaly. In our estimation this mouse model has more in common with a graft-versus-host-like disease process than psoriasis, as recently characterized by Christofidou-Solomidou *et al.*[4].

Based on follow-up studies using SCID mice engrafted with human skin, we believe the pathogenic immunocyte responsible for causing psoriasis is a memory CD4$^+$ T lymphocyte, and not a naive CD4$^+$ T cell[5]. While we agree with Schön *et al.* that it is becoming more apparent that psoriasis is probably not intrinsically a