problem see ref 5.)

Wolpert returns to old criticisms of Boltzmann's pioneering work. In view of the recent developments of unstable dynamical systems it is now well understood[6] that the objections raised against Boltzmann, such as the Zermelo paradox, are not meaningful, as the Poincaré recurrence time is much larger than the Liapunov time, being the time after which we lose control over the trajectories of a dynamical system.

In short, recent developments, including those of the Brussels School, permit one to incorporate the second law into the formulation of dynamics for highly unstable dynamical systems. In this way, the seeming contradiction between the reversibility of dynamics and the irreversibility of the second law has been overcome.

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'Amylin' hormone

SIR—Leighton and Cooper[1] recently reported a potential physiological function of a newly discovered pancreatic β-cell hormone which they refer to as 'amylin'. The introduction of the term amylin is a source of potential confusion because this peptide has also been called islet amyloid polypeptide (IAPP)[2–4] and diabetes-associated peptide (DAP)[5]. This fact prompts us also to point out recent progress in relating IAPP/DAP/amylin, which we prefer to call IAPP, to type-2 diabetes (age-related diabetes mellitus).

The 37-amino-acid IAPP is the main component of amyloid deposits of the islets of Langerhans occurring in association with type-2 diabetes in humans and cats[2–5]. It is a normal product of pancreatic β-cells[3,6,7] that is colocalized and probably cosecreted with insulin[8,9] and generated through proteolytic processing of an 89-amino-acid precursor (ref. 10; C. Betsholtz *et al.*, unpublished data).

IAPP inhibits insulin-stimulated glycogen synthesis in skeletal muscle preparations *in vitro*[1]. This is interesting in relation to the pathogenesis of type-2 diabetes because this disease is characterized by peripheral insulin resistance in addition to islet dysfunction.

That amyloid formation is of importance to the pathogenesis of type-2 diabetes is implied by the correlation between the presence of an amino-acid motif in the IAPP molecule and both amyloid formation and the spontaneous onset of type-2 diabetes in adult humans, cats and racoons; this motif is lacking in species that do not develop islet amyloid or type-2 diabetes (C. Betsholtz *et al.*, unpublished data).

Thus, changes in IAPP synthesis, processing or release may relate to both major traits of the type-2 diabetic syndrome; islet dysfunction through amyloid formation and insulin resistance through a peripheral action.

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On the SCIDs?

SIR—We recently reported (*Nature* **335**, 256–259; 1988) that a functional human immune system could be established effectively in mice with severe combined immunodeficiency (SCID mice) by inoculating them intraperitoneally with peripheral blood leukocytes (PBL) of human origin. The major immunological conclusions of that study are, first that recipient mice show prolonged survival with no ill effects of graft-versus-host disease; second, that the human leukocytes, including both B and T lymphocytes, increase in number and survive in these mice now for at least one year; third, that the reconstituted mice show significant levels of spontaneously secreted human immunoglobulin; and, last, that specific human antibody responses are inducible by immunization. These have all been confirmed in subsequent studies involving many more PBL-reconstituted mice.

More recent studies, however, have indicated that the numbers of intraperitoneally inoculated human PBL that migrate to the spleen, lymph nodes and peripheral blood during the first five-week period after reconstitution may be significantly less than those reported in Fig. 3 of our paper. For reasons not yet fully understood, antibodies used for detection of human cells show high levels of non-specific binding to splenic cells in these mice soon after reconstitution. Nevertheless, human cells do appear in these lymphoid organs in larger numbers at later times.

We caution other workers to be alert for this staining artefact, and we regret any inconvenience that our overestimates may have caused.

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Quasar enhanced

SIR—During the recent appearance of Halley's comet, observations were reported[1,2] of enhanced scintillations of quasars occulted by the cometary ion-tail; however, Ananthakrishnan *et al.*[3] dispute such an origin for these scintillations.

The geometry at the time of both the 103-MHz observations[1] on 18–20 December 1985, and those at 327 MHz (ref. 3) on 10–12 February 1986, is depicted in the figure. On 18 December 1985, the source, PKS2314+03, lay at a solar elongation of about 85°, such that the solar wind contributed only to background scintillations, with an average scintillating flux of about 3 Jy (ref. 1). The observed enhancement was six times this value and 1.5 times the maximum flux normally caused by the solar wind, which, for 103 MHz, occurs at a solar elongation of about 30°.

During the 327-MHz observations[3] on 11 February 1986, the source 2052−106 was presumed to be occulted by the cometary tail. As the perihelion occurred on 9 February, the period 10–12 February is most inappropriate for observing such events: the solar elongation of 2052−106 on 11 February was about 10°, for which, at 327 MHz, strong scattering prevails. The scintillation index is near its maximum at around 14°. Consequently, most of the scattering occurs in a thin layer of the solar plasma centred around the point of closest approach to the line of sight from the Sun. The three control sources were also within 12° of the Sun. Therefore, the scintillation enhancements of all of the sources were due solely to the solar plasma. The source also gets broadened by strong scattering close to the Sun and enhanced outflow of cometary plasma, which results in reduced scintillation. These conditions are therefore unsuited to observing enhancement of scintillations by Halley's comet.

Thus, enhanced scintillations of