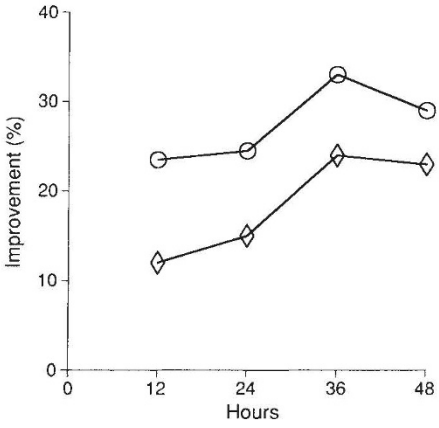
Predicting hurricane tracks

SIR — Predicting the paths of hurricanes has been extremely difficult. Over the past 30 years or so, the reduction in the 24-hour forecast track error has averaged only 0.5% per annum. This is largely a consequence of hurricanes spending almost all of their lives over the data-sparse oceans. Dynamical models of hurricanes have also been blamed, for being too simple. Indeed, such models have exhibited less forecast skill than statistically based methods, particularly in the first 24 or even 36 hours.

A superior data set has been collected in the hurricane-rich south China Sea during the 'TCM-90' experiment[1]. However, we find that the data alone do not lead to superior dynamical forecasts. These are only obtained after careful preparation of initial conditions at $t = 0$ hours, using a generalized inverse method developed originally for regional ocean modelling[2]. The method finds the flow field that is the weighted least-squares best-fit to the dynamical equations, boundary conditions and other data over the preceding 24 hours, $(-24 \leqslant t \leqslant 0)$, and also to the previous initial conditions valid at $t = -24$ hours. The huge number $0(10^5)$ of computational degrees of freedom in the fit is collapsed to a modest number $0(10^1)$, using the representer functions[3] associated with the problem. There is one representer for each effectively independent datum. The weights in the fit must be chosen to be the reciprocals of prior estimates of error covariances. The representers then yield the posterior error covariances for the fit. Serial Fortran-77 code for the algorithm is easily adapted to parallel computers[4].



Hurricane track predictions. Percentage improvement in (reduction of) mean forecast error to 48 hours ahead, relative to climatology persistence (CLIPER). Diamonds, standard initialization; circles, initialization by generalized inversion. By convention the 7% difference at 48 hours, for example, is scaled with respect to the 70% residual, yielding a '10% reduction'.

Our initial data at time $t = 0$, prepared by the inverse calculation, were complemented with boundary data for $0 \leqslant t \leqslant 48$ hours obtained from a coarse-resolution global forecast. The results of 10 cases from the TCM-90 data set are shown in the figure. The mean forecast error for the inverse initialization method is compared with the Australian Bureau of Meteorology's standard initialization method[5], and also with a statistical 'climatology persistence' method (CLIPER). The inverse method yields 14% improvements over the standard method at 24 hours, reducing to 10% at 48 hours. These improvements match those of the previous 30 years.

Finally we emphasize the simplicity of our dynamical model, which computes steering flows at only one pressure level, 500 hPa. Initialized by the generalized inverse method, this model also significantly outperforms more sophisticated, multilevel models.

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Superantigen data

SIR — In their Letter "Evidence for a viral superantigen in humans" (*Nature*, **358**, 507; 1992), Monique Lafon and colleagues describe the rabies N-protein as a superantigen, stimulating Vβ8-bearing human T cells and binding directly to major histocompatibility complex (MHC) class II antigens. However, some of this data may have been incorrectly interpreted. Western blotting (their Fig. 2*c*) clearly indicates that N-protein bound strongly to immunoglobulin heavy chain (M_r 50,000) run out on an SDS–PAGE gel following immunoprecipitation. Sadly, this binding was dismissed by that over-used term "non-specific", even though it was at least 10–20-fold stronger than the indiscernible MHC class II α-chain binding.

Further evidence for direct binding of N-protein to MHC class II-expressing B cells was given in Fig. 2*a* and *b* of Lafon *et al.* However, the recipient line probably also expresses surface immunoglobulin. This possibility was not tested, though the negative control was a class II-, immunoglobulin-negative T-cell line not an MHC class II-, immunoglobulin-positive B cell. Could the receptor for rabies superantigenic N-protein in fact be IgH, as suggested by Western blotting? This would be a most striking finding and may indicate that certain superantigens do not need MHC class II molecules to interact with T-cell receptor Vβ domains but may use surface immunoglobulin on B cells instead.

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LAFON REPLIES — Although pertinent, Fraser's point is only partially correct. Our data show that N-protein not only binds MHC class II molecules but also other cell surface molecules present on human B cells immortalized by Epstein–Barr virus (EBV). We draw this conclusion from the following experiments: (1) N-protein binds to the surface of murine fibroblasts transfected with MHC class II molecules but not to non-transfected ones, confirming that N-protein does bind to MHC class II molecules; and (2) N-protein not only recognizes EBV-transformed human B-cell lines expressing MHC class II molecules but also some negative MHC class II mutant cell lines, suggesting that N-protein reacts with additional surface molecules. These cell surface molecules could be the heavy chains of immunoglobulins.

It cannot be excluded that other receptors are expressed on the surface of B cells. It is noteworthy that the so-called "unspecific binding of superantigen to IgG" is visible in immunoblots presented in articles studying the reactivity of exotoxin superantigens with MHC class II (for example, J. A. Mollick *et al. Science* **244**, 817–820; 1989), indicating that binding to immunoglobulin G would be a general feature of superantigens rather than an exception.

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