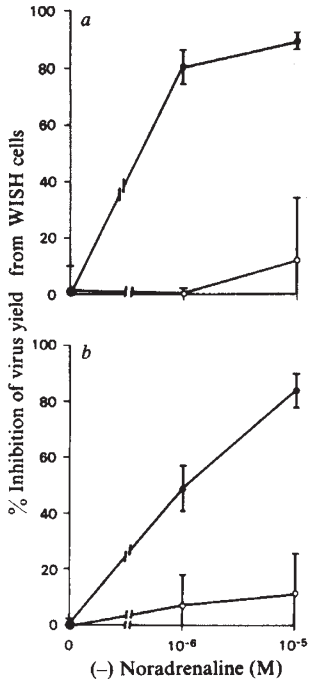

Fig. 3 Effect of (−)noradrenaline on virus replication in co-cultures of mouse myocardial cells and human amnion (WISH) cells. Mouse myocardial cells (3.0×10^4 per well) and human WISH cells (3.0×10^4 per well) were cultured alone or in combination (1:1 ratio) in Falcon microtitre tissue culture plates. After 24 h medium was replaced with the indicated concentrations of noradrenaline and incubated overnight. Supernatant fluids were removed and cultures were infected with 300 PFU of either VSV (*a*) or poliovirus (*b*). Virus yields were determined 24 h later by a microplaque assay[1]. Each point represents the mean % inhibition of the control virus yield ± s.d. ($n = 6$). VSV yield from myocardial cells alone was inhibited (Fig. 1*b*). Poliovirus did not replicate in mouse myocardial cells. ●, Myocardial cells + WISH cells; ○, WISH cells.

Thus, we have demonstrated that interferon can have hormonal activity and that hormonal stimulation can result in interferon-type antiviral activity. From these findings we conclude that interferon and hormonal action are probably mediated by common pathway(s). The transmission of the reciprocal actions of interferon and noradrenaline not only gives further credence to a common pathway of their actions but also suggests that common transferred molecule(s) are generated after interaction of either substance with the appropriate cell membrane. Superficially, cyclic AMP seems a candidate for the interferon-induced increase in beat frequency, because cyclic AMP can cause this response[2,9,10] and interferon in certain conditions can elevate cyclic AMP levels[11,12]. However, cyclic AMP alone cannot account for the antiviral effects as it is not antiviral[13] and interferon does not stimulate adenyl cyclase in all cells[11,12]. A more likely situation is that cyclic AMP and/or another small molecule(s) is responsible.

A question which results from these studies is, is interferon a hormone? The many similarities between interferon and polypeptide hormones indicate that interferon should be classified as such, and this is supported by our inability to distinguish interferon action from a hormonal response. As such, the natural role of interferon may be regulatory, with its effects on virus infections being secondary. The instances of low levels of interferon in normal individuals may not result from inapparent virus infections but may reflect this more general interferon regulatory mechanism. Additionally, this could be related to the side effects observed during clinical trials using high levels of interferon[14] as well as some aspects of viral pathogenesis. If interferon can cross-activate for other hormonal activities, this might explain the many diverse actions of interferon (for review see ref. 15). A shared mode of action of interferon and hormones would suggest that interferon action is not unique and makes questionable the view that the varied biochemical changes in interferon-treated cells are interferon specific.

A second question is, what are the limits of responses to hormones? The present findings suggest that hormones may, in addition to their known actions, protect tissues against viruses or maintain differentiation. If this could be documented *in vivo*, a new strategy of tissue-targeted antiviral and antitumour therapy might evolve.

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Corrigendum

In the letter 'Gravitational magnetism' by S.-P. Sirag, *Nature* **278,** 535 (1978), in Table 1 the maximum magnetic moment, *P*, for Her X−1 should be 35×10^{26} A m^2.

Errata

In the letter by C. D. B. Bridges and S.-L. Fong, *Nature* **282,** 513 (1979), the title should have read: 'Different distribution of receptors for peanut and ricin agglutinins between inner and outer segments of rod cells'.

In the letter 'Oxidant damage mediates variant red cell resistance to malaria' by M. J. Friedman, *Nature* **280,** 245 (1979), the second sentence of Fig. 2 legend should read: 'The interaction of H_2O_2 and Hb results . . .'.

In the letter 'Inhibition of mixed lymphocyte response by monoclonal antibody specific for a rat T lymphocyte subset' by M. Webb *et al.*, *Nature* **282,** 841 (1979), the symbols in Fig. 1 have been wrongly given. The correct designation is: –●– w3/25 IgG, –○– W3/25 F(ab')$_2$, –□– W3/13 IgG.

In the News and Views article 'Is anyone out there?' *Nature* **281,** 528 (1979), two lines were inadvertently omitted leading to the attribution of views to I. Shklovsky entirely opposite to those he actually stated. The correct sentence should read: "I. Shklovsky of the Sternberg Astronomical Institute suggested that the choice of a value for *N is a function of the age of the investigator. He attributed 'youthful optimism' to T. Kuiper of the Jet Propulsion Laboratory who suggested that N may be a very large number* ($\geq 10^9$).