difficult task.

Although this is a negative result, the importance for theoretical physics of keeping the natural constants constant makes these occasional cosmological checks well worthwhile. In the absence of a theory of natural constants, the significance of their numerology will have to be left to the philosophers to ponder.

Herpes virus induction of F_c receptors

from R. S. Kerbel

WHEN cells are infected with viruses or are transformed by oncogenic viruses, alterations in plasma membrane components often occur. The characteristics of these surface changes and how they may relate to the biological behaviour of the cells have been popular areas of study for both virologists and immunologists alike.

One example of how spectacular these changes can be concerns induction of surface receptors, specifically F_c receptors. Over 12 years ago, Watkins noted that cultured HeLa cells were able to absorb antibody-coated erythrocytes and form rosettes after the cells were infected with herpes simplex type 1 virus (*Nature*, **202**, 1364; 1964). This was later confirmed by Yasuda and Milgrom who used various different cell lines derived from both man and monkey (*Int. Arch. Allergy*, **33**, 151; 1968).

We now know that a variety of cells, mostly lymphoreticular in nature, have surface receptors for the F_c portion of IgG immunoglobulin molecules (F_c receptors). These receptors can be detected by erythrocyte–antibody (EA) rosette formation. Thus, it seems that infection with herpes simplex virus (HSV) results in induction of F_c receptors, a conclusion substantiated by Yasuda and Milgrom who showed that virus-infected cells could not absorb erythrocyte coated with $F(ab')_2$ antibody fragments, but only with the fully intact antibody molecules.

The gene which codes for the receptor seems to be virally coded, and not a repressed host gene switched on by viral infection (Westmoreland and Watkins, *J. gen. Virol.*, **24**, 167; 1974). Subsequent studies have shown that mammalian cells such as hamster embryo fibroblasts oncogenically transformed by inactivated HSV types 1 and 2 can also display F_c receptors (Westmoreland, Watkins and Rapp, *J. gen. Virol.*, **25**, 167; 1974).

It was only natural to ask whether other members of the herpes family of viruses could also induce the appearance of F_c receptors in infected cells and an affirmative answer was recently provided by Furukawa *et al.* who found human fibroblasts displayed F_c receptors after infection with human cytomegalovirus (*J. clin. Microbiol.*, **2**, 332; 1975), an observation also made by Rahman *et al.* (*J. Immun.*, **117**, 253; 1976), Keller *et al.* (*J. Immun.*, **116**, 772; 1976) and Westmoreland, Jeor and Rapp (*J. Immun.*, **116**, 1566; 1976).

Now that every type of cell thus far tested from over six different species has been shown to develop F_c receptor sites after infection by at least two different herpes viruses, the possibility looms larger than ever that these receptors have a role in the natural history of herpes virus infections. It has been suggested by Westmoreland and Watkins and also by Costa and Rabson (*Lancet*, **i**, 77; 1975) that the development of F_c receptor sites may play a part in conferring a biological advantage on the virus and therefore in maintaining the latent nature of herpes infections, since the coating of infected cells with IgG molecules or immune complexes by the F_c region of the immunoglobulin molecules might protect the cells from the destructive effects of potentially cytotoxic antibodies and lymphocytes.

Lehner, Wilton and Shillitoe (*Lancet*, **ii**, 60; 1975) suggested an alternative way in which F_c receptors may be involved in the latent and recurrent nature of HSV infections, namely "double" binding of anti-HSV IgG antibodies to virus-infected cells to induced F_c receptors and HSV surface antigens, by way of their F_c and F_{ab} portions, respectively. The F_c portion of the anti-HSV antibodies would then be unavailable for either complement binding or for F_c receptors on effector cells capable of mediating antibody-dependent cell-mediated cytotoxicity. Lehner *et al.* also speculate that the spread of putative HSV-induced squamous cell carcinomas might also be facilitated by double-binding of F_c receptors and HSV antigens by IgG antibodies on the surface of dividing carcinoma cells. If there is indeed a relationship between HSV and squamous cell carcinoma, it will be of interest to see whether F_c receptors can somehow be exploited as a tumour cell marker in this particular case.

The *in vivo* significance of induced F_c receptors on herpes virus-infected cells awaits further experimentation. For the moment, the emphasis is on the various practical *in vitro* considerations of the phenomenon. A particularly illuminating example is the recent work of Rager-Zisman, Grose, and Bloom (*Nature*, **260**, 369; 1976) who found that HSV-infected target cells could be non-specifically lysed by F_c receptor-positive killer cells in the absence of specific anti-HSV antibodies; the authors provided further evidence that this phenomenon might be mediated by the cross-linking of effector cells and target cells, both displaying F_c receptors, by aggregated immunoglobulins or soluble immune complexes present in the serum of the culture medium. This supports the suggestion first made by Lehner *et al.* (*op. cit.*) that complexes of HSV and IgG antibodies might bind to F_c receptors of both HSV-infected target cells and effector cells resulting in killing of the target cell.

Immune complex (or aggregated Ig)-dependent cell-mediated cytotoxicity may therefore represent one of the mechanisms by which F_c receptor-positive lymphocytes from normal donors can spontaneously lyse tumour target cells *in vitro*, a phenomenon observed by several investigators (for example, Pross and Jondal, *Clin. exp. Immun.*, **21**, 226; 1975; Bean *et al.*, *Cancer Res.*, **35**, 2902; 1975) and which has often been a serious technical problem in specific cell-mediated microcytotoxicity assays. If so, the use of F_c receptor-negative tumour target cells or culture media free of immune complexes and/or aggregated Ig should minimise or even prevent, in some cases, spontaneous lymphocyte-mediated cytotoxicity. □

Plant–microorganism interactions

from R. M. Cooper

A symposium on cell wall biochemistry related to specificity in host–plant pathogen interactions (organised by J. Raa and B. Solheim) was held at the most northerly university in the world in Tromsö, Norway on August 2–6, 1976. The proceedings of the meeting are to be published.

THE molecular basis of the specific parasitism or symbiotic associations of certain microorganisms with a plant species, or even variety, has fascinated but eluded scientists for many years. One such specific interaction is the so-called 'gene-for-gene' relationship between biotypes of plant pathogens and their hosts 'evolved' as a result of pathogens' overcoming new resistance genes introduced by man into crops. Similarly the symbiotic association between roots of legume species and strains of the soil bacterium *Rhizobium* (a major source of biologically-fixed nitrogen) can exhibit a marked degree of specificity. These two types of specificity comprised the