poration in these cultures were similar to those in the autologous mixed leucocyte culture (see table). On the other hand, vigorous stimulation was observed in allogeneic cultures containing non-spermatozoal seminal cells. We conclude that spermatozoa per se are not responsible for the stimulation observed in mixed sperm/leucocyte cultures.

All semen samples contain non-spermatozoal cells, but the numbers vary considerably between donors and at different times in the one donor. It is not surprising that variable levels of stimulation are a common feature of mixed sperm/leucocyte cultures, since the degree of stimulation is likely to be modulated not only by the numbers of non-spermatozoal cells but also by the types of cells present in the semen. Further studies are being undertaken to determine the types of cell present in semen, and their capacity to induce immunological responses, and to investigate the stimulatory ability of spermatozoa in secondary mixed sperm/leucocyte cultures.

**PROSTAGLANDINS AND ACTINIC CANCER**

Sir,—Greaves and others have shown that prostaglandins (P.G.) are increased in human skin after exposure to ultraviolet light in the "sunburn" spectrum (290—320 nm). Since there is evidence that P.G.E reduces proliferative activity in the epidermis, Professor Greaves (Jan. 28, p. 189) proposes that this substance may reduce the vulnerability of epidermal cells to the mutagenic effects of ultraviolet light. We have also been interested in the relation between P.G.E formation and skin malignancies, but would suggest that the inhibitory effect of P.G.E on epidermal proliferation may be more than countered by other effects of this substance.

Immunological surveillance seems to play an important role in limiting premalignant and malignant lesions of the skin. This is suggested by the increased incidence of light-induced cancers in immunosuppressed patients and by the increased incidence of light-induced cancers in immunosuppressed laboratory animals. Acute exposure to ultraviolet light greatly reduces the ability of mice to reject transplanted skin tumours. Prostaglandins, particularly P.G.E., suppress immune function and may be the mediator by which suppressor cells exert their effects on immune responses.

We suggest that the P.G.E, released in the skin after irradiation with ultraviolet light may suppress immune responses in the skin and interfere with normal immunological surveillance. This effect probably completely offsets any protective effects of prostaglandin formation. Suppression of immune responses by ultraviolet-evoked prostaglandin formation would explain the increased incidence of human skin cancers in the months of more intense and more prolonged light exposure. It could also explain the more severe manifestations of viral exanthems in light-exposed skin.


**AFLATOXIN B1 AND REYE'S SYNDROME**

Sir,—The aetiology of Reye's syndrome, a distinct clinical entity characterised by acute encephalopathy and fatty degeneration of the viscera, remains obscure. Numerous aetiological agents including viruses, fungi, toxins, and drugs have all been incriminated but no direct cause-and-effect relationship has been established. The hepatotoxic effect of aflatoxin B, and the isolation of aflatoxin B1 at necropsy from the tissue of 22 of 23 cases of Reye's syndrome in Northern Thailand have stimulated interest in the possible role of aflatoxin B in the aetiology of the syndrome. Aflatoxins have been associated at necropsy with Reye's syndrome in New Zealand (2 cases), Czechoslovakia (2 cases), and in the United States (1 case).

We know of no previously reported cases in which aflatoxin B1 was found in the blood of patients with Reye's syndrome during the acute phase of the disease. We have seen two such cases.

Both cases were preceded by viral illness and the patients showed vomiting, hyperventilation, hepaticomegaly, decerebrate posturing, seizures, and coma.

Serum glutamic-oxaloacetic transaminase was 396—888 and 585—616 units/ml respectively; lactic dehydrogenase was 897—1224 and 1653—5820 units/ml respectively; blood-ammonia was 200—720 and 175—229 μg/dl respectively; blood-glucose was 39 and 141 mg/ml respectively; and aflatoxin B1 was 11.93 and 31.3 ng/ml respectively.

Both children died shortly after admission. Reye's syndrome was confirmed at necropsy in case 1. Necropsy was denied in case 2.

We now have studies under way to determine the significance of these findings.

**ORIGIN OF MONOCLONAL ANTIBODIES**

Sir,—In your editorial of Feb. 4 (p. 252) you cite us as having found a monoclonal antistreptolysin O antibody in a patient only a few months after the onset of symptoms, stating that such a time interval would not be incompatible with a monoclonal immune response. In fact this interval was no more than three weeks in one of our two cases; in the other the abnormal component was discovered much later.

Could an observable monoclonal immune response be mounted in such a short period? Hardly any data are available on this point. Rádl et al. detected monoclonal antibodies against human serum in two bone-marrow depleted and reconstituted monkeys about ten days after challenge. However, in