When $K = 0$, we recover eqs. (6) and (7) of I from eqs. (5) and (6) respectively.

It is seen that the conclusions of *I*, *i.e.*, when $K = 0$ also hold good for other values of K, while as in Soundalgekar and Gipta's work[4], the dispersion coefficient $F(\alpha)$ or $G(\alpha, \beta)$ increases with increase in K for fixed values of all the other parameters.

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ANTIBODY FORMING CELL RESPONSE OF MICE TO DENGUE VIRUS GIVEN BY DIFFERENT ROUTES

Dengue type 2 virus produces a lethal infection in adult mice *via* the intracerebral route, but fails to produce morbidity or mortality when administered intraperitoneally to adult mice[1]. We have further noted that dengue virus remains ineffective when given intraperitoneally or intravenously to adult mice (unpublished data). Is this resistance to peripheral route of inoculation of dengue virus due to a greater production of interferon, or to a specific immune response? Interferon does not appear to be responsible for this resistance as it has been shown that the administration of 500 units of interferon had no effect on the outcome of dengue infection[2]. It was, therefore, considered worthwhile to investigate the immune response of mice following inoculation of the virus by different routes.

The study was carried out on an inbred strain of male albino mice, weighing 20 to 25 g (aged 4 to 6 months). The dengue type 2 virus (strain 23085) was used in the form of an infected mouse brain suspension. The virus was given in doses of about 10^3 LD_{50} intracerebrally (i.c.) to mice of group A, intraperitoneally (i.p.) to group B and intravenously (i.v.), through the tail vein, to group C. From each group, 5 to 7 mice were sacrificed daily between post-inoculation days 1 to 15 and the spleens collected aseptically. The splenic cells were squeezed out, washed thrice and suspended in MEM medium. The antibody plaque-forming cells were counted by the localized haemolysis-in-gel technique of Jerne *et al.*[3]. The coating of sheep RBC with the dengue virus was carried out using the technique of Russell *et al.*[4]. Only direct plaque forming cells (PFC) were counted.

From each mouse, multiple slides were prepared, depending upon the count of the splenic cells. The mean values of PFC per 2×10^7 splenic cells (nucleated) of slides from 5 to 7 mice, studied daily are preseneted in Fig. 1. The mice given

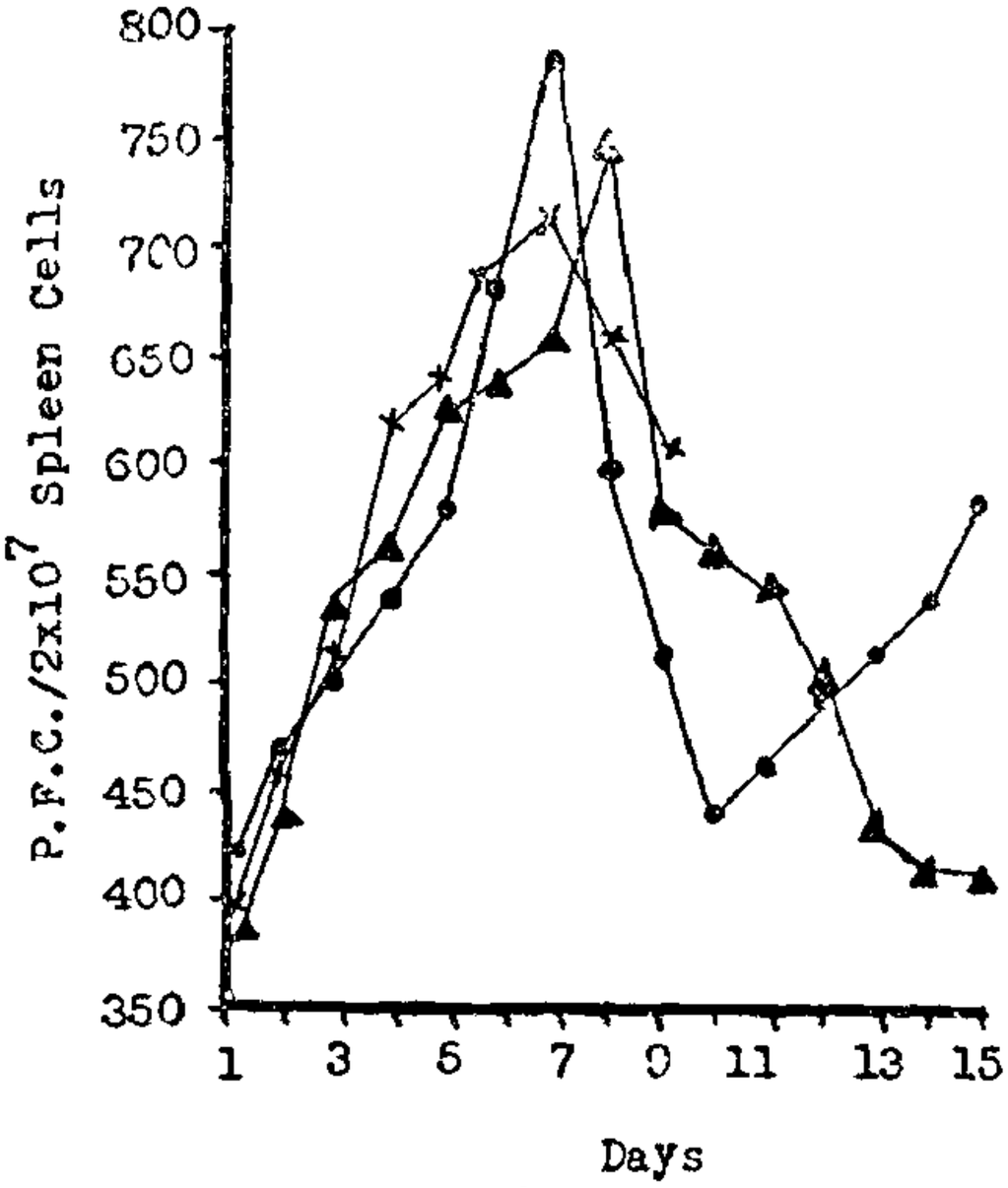

Fig. 1. Antibody plaque forming cells (PFC) in the spleen of mice given dengue 2 virus by i.c. (×—×), i.p. (●—●), or i.v. (▲--▲) routes.

virus i.c. sickened and died by the 10th day, hence, in group A, the observation could be recorded upto day 9, only. In mice from group A, when given virus by the i.c. route, the PFC increased from $396/2 \times 10^7$ cells on day 1, reached a peak on day 7 ($716/2 \times 10^7$) and then decreased to $608/2 \times 10^7$ on day 9. The mice from group B, given virus i.p. showed 420 PFC/2×10^7 cells on day 1, which figure increased to 792 PFC on the 7th day. This number then declined until the 10th day, when the count was 440. The cells increased again and, on 15th day, the count was 584. Mice from group C, which were given virus by the i.v. route showed 384 PFC/ 2×10^7 splenic cells on day 1; this figure gradually increased to 748 PFC on 8th day. There was subsequently, a constant decline of PFC until the 15th day, when the count was 404.

The findings of the present study thus indicate that the immune response of mice administered dengue virus by different routes, as determined by the antibody plaque forming cells of the spleen, does not differ materially. Hence, a lack of the proper immune response does not appear to be

responsible for the observed morbidity or mortality of mice when virus is given by the i.c. route. It appears that the replication of virus in the brain tissue is an essential factor leading to illness in mice.

We are thankful to Prof. R. M. L. Mehrotra, for the help.

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TRANSFER OF JUVENOIDS AND INHIBITION OF EMBRYOGENESIS IN *DYSDERCUS CINGULATUS* (HETEROPTERA)

INHIBITION of embryogenesis by juvenile hormone analogues has been reported in the silk worm, *Hyalophora cecropia* (Riddiford, 1968), European corn bug, *Pyrrhocoris apterus* (Slama and Williams, 1966), and in locust, *Schistocera gregaria* (Novak, 1969). In the present investigation, the juvenile hormone analogue, P–169 (Ethyl-pivaloyl-L-alanyl-*p*-aminobenzoate) was tested on the red cotton bug, *Dysdercus cingulatus*, and its effects on oogenesis were studied.

The bugs were regularly reared (Judson *et al.*, 1976) and the freshly ecdysed adult females were topically treated with 1 μg/sp of the analogue. Its ID 50 value on the V instar nymphs of *D. cingulatus* has been shown to be 0·00004 μg/sp (Slama *et al.*, 1974). They were kept with freshly ecdysed untreated males and were mated. Controls were treated with 1 μl/sp of acetone only. On the third day, the females were dissected and their ovaries were examined and fixed for further histological studies. After removing the female on the third day another freshly ecdysed untreated female was introduced in its place. These insects mated normally and oviposited, but the eggs oviposited by this female failed to hatch.

The dissected ovaries of the treated females showed morphological differences when compared with the controls. Compound egg chambers (2 to 3 oocytes in each) were found in many ovarioles, in some ovarioles only one or two oocytes were developed instead of four to six as in the controls. Furthermore, a few ovarioles were in a degenerated condition (Divakar and Novak, 1974; Judson *et al.*, 1976). In the histological preparations also, the same effects found after treatment with 'Paper Factor' (Judson *et al.*, 1976), such as the compound egg chambers with continuous ooplasm were observed. This compound was thus found potent enough to cause sterility and contaminate the untreated ones through transfer of the analogue during copulation as reported by Masner *et al.* (1970) on *Pyrrhocoris apterus* with other synthetic juvenile hormone analogues. These results strongly support the statement of Williams (1967) that the juvenoids can be used as third generation pesticides and the auttors are strongly of the opinion that this compound could also be used in the field against plant bugs.

The authors are grateful to Dr. K. Slama, for providing the analogue and to the C.S.I.R., New Delhi, for the financial assistance to the first two authors.

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