Weiner's group have since begun to investigate the virus by propagating it in cultured cells.

Now on page 343 of this issue of *Nature* a South African group, Lecatsas *et al.* working in Pretoria, report detecting a papova virus in the urines of no less than eight patients who had received kidney transplants. The concentration of virus in the urine and the duration of its presence apparently varied considerably from patient to patient and there was no obvious correlation between the date of detection of the virus and the date of the kidney transplantation. There may, however, be a correlation between the appearance of the virus and the use of antilymphocytic serum as an immunosuppressive. Apparently, all eight of the South Africans excreting the virus were given horse antilymphocytic serum, whereas, when working in London in 1971, Lecatsas failed to detect the virus in 150 urine samples from 31 kidney transplant patients who were not receiving antilymphocytic serum but were receiving immunosuppressive drugs. These various findings indicate, of course, that the multiplication of this latent human virus seems to depend on the immune status of an individual.

Obviously it must now be discovered whether this human papova virus(es), like SV40, has an oncogenic potential and can, for example, induce tumours in rodents and transform rodent cells in culture. From what is known about SV40 it would not be at all surprising if the human virus has an oncogenic potential. Whether, however, it is ever involved in the induction of cancer in its natural host, man, is another question. Lecatsas *et al.* propose to follow up the patients which they know have excreted the virus to see if they develop tumours and if they do whether or not the papova virus can be detected in the tumour cells, and no doubt other groups will use serological tests to see if there is any correlation between appearance of the virus and the development of tumours. But if what is known about SV40 is anything to go by nobody will be surprised if these tests all prove negative, and this human papova virus joins the ranks of other viruses which are not apparently involved in carcinogenesis in their natural hosts even though they are oncogenic in other species.—From a Correspondent.

ORGANOCHLORINE PESTICIDES

DDT Levels in Barracuda

GROSSLY misleading information can result from spot checking fish tissues for fat soluble pollutants in order to estimate local environmental pollution, according to the evidence of Deichmann, Cubit, MacDonald and Beasley (*Arch. Toxikol.*, **29**, 287; 1972) who have looked for organochlorine pesticides in the abdominal fat of adult barracuda shark caught in Florida waters. These pesticides enter coastal waters by terrestrial runoff and relatively large quantities of soil-bound DDT and related pesticides settle as the current decreases. As a group they are very insoluble in water, but quite soluble in fats, oils and organic solvents. DDT, as traces in solution, floating in surface films and as bottom sediment is subsequently partially absorbed by aquatic plants and marine organisms and moves through the food chain which forms the chief source of pesticide intake by fish.

Pesticide monitoring programmes have revealed that most of the fish in the waters of the United States contain DDT. They also frequently contain dieldrin, sometimes heptachlor epoxide, chlordane and heptachlor, and occasionally lindane, aldrine or endrin. Most analyses show that the "total" DDT (DDT plus its metabolites) contained in fish is less than the United States Food and Drugs Administration tolerance level of 5.0 p.p.m. for the edible portions. Concentrations of pesticide in individual tissues vary considerably; the highest concentrations are generally found in body fat. Deichmann *et al.* analysed 281 giant barracuda (*Sphyraena barracuda*); 63 per cent were found to have more than 5 g abdominal fat, and of these "fat" fish, 96 per cent contained DDT or one of its isomers or metabolites. Except for the smallest and the largest fish, the ratio of the milligram "total" DDT in fat to kilogram body weight remained constant, although the p.p.m. increased with increasing size.

Little abdominal fat was found in the barracuda with fully or nearly fully developed gonads, at the height or near the end of the spawning season (June to September). The concentration of "total" DDT was calculated in other organs and, unexpectedly, only traces were found in the ripe gonads; the fish had lost approximately 75 per cent of their "total" DDT, the female fish carrying the smaller load. Starving was not the cause of this loss of body fat plus pesticides during spawning because the stomach contents of these fish were even slightly greater than at other times. Other workers have reported high levels of pesticides in prespawning gonads, and high mortality of larvae in lake trout has been attributed to metabolization of yolk fats containing DDT. Deichmann *et al.* do not know what happens to the pesticides during the spawning season, except that they are returned to the water, to a cycle of uptake and excretion.

Two important facts emerge from this work. First, a high level of DDT in fish does not necessarily mean that greater than FDA levels will be ingested as the fat and oil containing the pesticide separate from the fish during heat-

Localization of Human *X*-Linked Genes

DURING the past several years cell geneticists have exploited the parasexual event of fusion of somatic cells in culture to identify linkage groups; for example, by fusing mouse and human cells and correlating the loss of human chromosomes with the loss of human enzymes from the hybrid cells it has proved possible to identify which chromosomes carry the genes for at least a few human enzymes. Obviously such experiments do not localize genetic markers to particular regions of any chromosome but, by exploiting recognizable translocations, a more precise localization of markers to particular regions of chromosomes is possible, as Ruddle and Ricciuti report in *Nature New Biology* next Wednesday (February 7).

Ruddle and Ricciuti have studied a translocation between the human *X* chromosome and the human *D*-14 chromosome detected in a mother and her mentally retarded son. Fibroblasts from these two persons were placed in culture, fused with cells of a mouse line, RAG, lacking hypoxanthine guanine phosphoribosyl transferase (HGPRT) and grown in HAT medium which selects for the retention of the human *HGPRT* gene which is *X*-linked, as are the genes for glucose-6-phosphate dehydrogenase (G6PD) and phosphoglycerate kinase (PGK).

Analyses of the pattern of segregation of these human genetic markers and the segregation of the *X* chromosome translocation led Ruddle and Ricciuti to the following conclusions. First, all these three markers are apparently located on the long arm of the *X* chromosome. Second, it seems probable that "*G6PD* and *HGPRT* are more closely linked to each other than either are to *PGK*". These experiments also prove that with patience and ingenuity somatic cell geneticists can assign markers not just to chromosomes but to particular regions of those chromosomes.