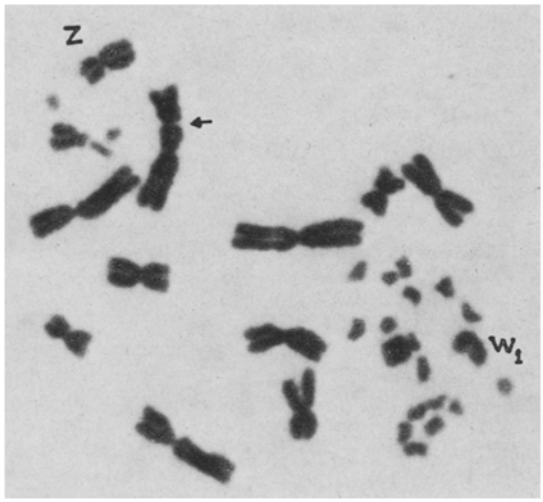

Fig. 4. Metaphase plate of somatic cell from spleen of *E. schistosa* female showing secondary constriction in 1 of the chromosomes of pair 1. ×2000.

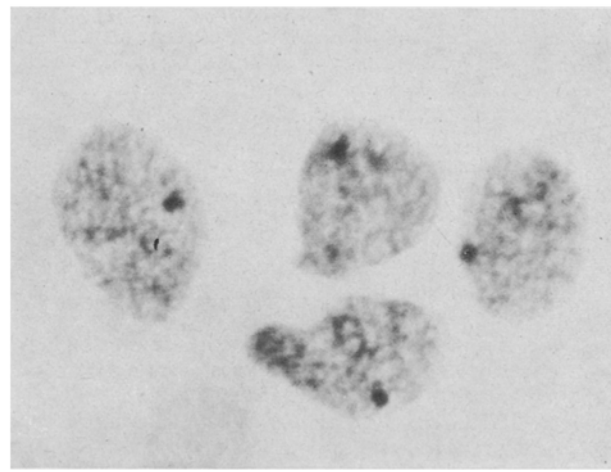

Fig. 5. Interphase nuclei of *E. schistosa* female from kidney showing single *W* chromatin body. ×2000.

chromosome mechanism. The only publication on the chromosomes of sea snakes is that of NAKAMURA[4], who studied only the males of *Laticauda semifasciata* with the help of classical techniques and reported 38 as the diploid number in that species. A number of species of sea snakes have now been collected by us and our preliminary observations reveal that multiple sex chromosome complex of the kind described here is quite widespread in Hydrophiidae, specially in the genus *Hydrophis*. A fuller account of the chromosomes of all the species, together with the probable mechanism of the origin of multiple mechanism, will be the subject of a future communication.

The *W* chromosome in snakes, whenever it is distinguishable from the *Z* either by its morphology and/or by its allocycly in its DNA replication, forms a heteropycnotic body in cells at the interphase stage in various somatic tissues which has been termed *W* chromatin[3]. Therefore the *W* chromosomes in *E. schistosa* should show in the interphase nuclei two *W* chromatin bodies, one bigger and one smaller corresponding in size to W_1 and W_2 respectively. Further, our unpublished observations on the chromosomes of various species of snakes of the genus *Hydrophis* reveal that there is an exact correspondence between the number of *W* chromosomes at metaphase and the *W* chromatin bodies in the interphase nuclei. Contrary to our expectation, only 1 *W* chromatin body has been detected in the interphase nuclei of brain, kidney and leucocyte culture. Perhaps the very minute size of the W_2 prevents the detection of the second *W* chromatin body.

Résumé. Un serpent marin (*Enhydrina chistosa* Daudin, Hydrophiidae) a 32 chromosomes chez le ♂, 33 chez la ♀, cette dernière ayant un *W*-chromosome supplémentaire. La digamétie serait: ♂-*ZZ*; ♀ *Z* W_1W_2.

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Discrepancies between the Sites of Replication and Cytocidal Action of Encephalitogenic Viruses

It is generally taken for granted that in such viral encephalomyelitides as result in a severe and widespread nerve cell destruction, the virus is replicated mainly by the neurons. How erroneous this assumption is became evident in the course of comparative studies on the ultrastructural pathomorphology of several experimental viral infections of the simian and murine central nervous system.

In the first part of our experiments, adult cynomolgus monkeys were inoculated i.m. or s.c. with a highly virulent strain of type 3 poliovirus. Beginning with the 4th or 5th post-inoculation day, the animals developed a rapidly progressive paralysis of the limbs often terminating in a total tetraplegia. Electron microscopically, numerous spinal motoneurons of the paralytic monkeys exhibited unequivocal signs of incipient or advanced necrobiosis. Rather frequently, nerve cells in the stage of complete cytolysis and/or undergoing actual neuronophagia were observed. However, no one ultrastructural finding could be observed in the more or less damaged neurons which would have been directly indicative of the intracellular presence of poliovirus. On the other hand, many mononuclear elements of the inflammatory infil-

trates as well as numerous endothelial cells of the intraspinal blood vessels, contained within their cytoplasm crystalline aggregates of small spherical corpuscles. These, with respect to their size, shape and electron density, closely corresponded to poliovirus particles[1-4]. From these electron microscopic observations which were largely corroborated by the results of extensive immunofluorescence examinations[3,4], it was clear that in experimental poliomyelitis of the monkey, the nerve cells cannot be considered as the principal replication sites of the virus, though they are by far the most severely affected elements. The poliovirus, once having entered a neuron, is obviously capable of disturbing its complex metabolic machinery in such a pernicious manner that necrobiosis takes place even before a single replication cycle is achieved. On the contrary, the mesenchymal elements seem to be much less sensitive to the damaging action of the poliovirus genome, and evidently play the most significant role in poliovirus multiplication.

In the second part of our experiments, adult albino mice were inoculated intracerebrally with 4 different group B arboviruses, namely with Central European encephalitis virus (HYPR strain), Zimmern virus[5,6], Gressthal virus[5,6], and Yellow Fever virus (17 D vaccine strain). After intracerebral injection of the 3 former viruses which belong to the tick-borne encephalitis complex, the animals developed paralytic signs of severe CNS involvement at the 4th or 5th post-inoculation day. With the Yellow Fever virus, the incubation period was about 8 to 10 days. Several regions of the neuraxis from the mice with group B arbovirus encephalomyelitides were examined by electron microscopy. The observations made herein were quite different from the findings obtained in monkeys with experimental poliomyelitis. Within the perikarya of numerous nerve cells, varying amounts of typical group B arbovirus particles were encountered. In further contrast to poliomyelitis, the cyto- and nucleoplasmic fine structure of the arbovirus infected murine neurons appeared amazingly well preserved. Admittedly, many nerve cells showed mild to moderate degrees of 'degenerative' alterations, e.g., formation of vesicular and vacuolar aggregates in the cytoplasm, disorganization or reduction of the endoplasmic reticulum, and chromatolysis of variable severity. However, no single neuron was seen which could be said to be actually necrotic or to undergo active phagocytosis by leukocytes and microglial cells. Moreover, in all arbovirus infected mice thus far examined, the non-neuronal elements within the CNS, especially the inflammatory cells, were absolutely free from structures suspicious as being of viral nature[7-9].

From the latter observations it can be concluded that, in group B arbovirus encephalomyelitides of mice, the neurons are the primary or even the only sites of viral replication within the CNS. Nevertheless, the viral action upon the nerve cells is by no means so deleterious as that exerted by the poliovirus in the simian neuraxis. The clinical symptoms and even the lethal termination of the encephalomyelitic process seem to be due more to slight functional disturbances of the vast majority of the neurons in the sense of a summation effect rather than to a widespread nerve cell destruction. From classical neuropathological studies, it is well known that in human cases with group B arbovirus infections of the CNS, especially in cases of tick-borne encephalomyelitides, a serious loss of nerve cells may take place. We are therefore of the opinion that in murine arbovirus infections the absence of severe neuronal damage which contrasts with the luxuriant viral growth in the nerve cells, depends less on the properties of the virus than on that of the host species. Small rodents, particularly mice, probably play a very significant role in the ecology of many arboviruses, namely of the non-mosquito-transmitted arboviruses, in that they are most often the prevailing vertebrate hosts in the endemic foci, whereas infected human beings represent merely lateral dead ends in the natural virus cycle. For instance, in an endemic area of Central European encephalitis in Lower Austria, a primary independent virus cycle involving ticks and only some species of wild mice could be demonstrated[10,11]. One may suggest, therefore, that the reaction of mice to arbovirus infections has been specifically modified by a gradual adaptation process, and is largely different from that of other animals. Future electron microscopic studies of experimental group B arbovirus encephalomyelitides in various mammals, especially in primates, should give more precise information about this intringuing problem. In the present paper, we merely intended to point out that the site of replication and the site of cytocidal action of an encephalitogenic virus need not necessarily coincide. When we speak of a neurotropic or neurovirulent virus, we should always make clear what this designation properly means, i.e., whether the virus prefers neurons for its growth or the virus has a rapidly ruinous effect upon this type of cell.

Zusammenfassung. Die Ergebnisse elektronenmikroskopischer Untersuchungen spechen dafür, dass encephalitogene Viren nicht immer jene Zellarten am stärksten schädigen, durch welche sie bevorzugt repliziert werden. Bei der experimentellen Poliomyelitis des Java-Affen fanden sich Polioviruskristalle häufig in weitgehend intakt erscheinenden mesenchymalen Zellelementen, niemals dagegen in den meist nekrobiotisch alterierten spinalen Motoneuronen vor. Bei einigen experimentellen Arbovirus-Encephalomyelitiden der Albinomaus wiederum konnten Viruspartikel ausschliesslich in Nervenzellen beobachtet werden, welche aber gewöhnlich nur geringfügige Strukturveränderungen aufwiesen.

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