This agrees with his earlier statements [in *An Appraisal of Anthropology Today,* Tax *et al.,* Eds. (Univ. of Chicago Press, 1953), pp. 29–30; *Weekly Evening Meeting Roy. Inst. Gr. Brit.* (20 Nov. 1953)] that the blackening of bones found at Makapan is due to manganese dioxide and is not due to charring, as claimed by Dart; furthermore, the other supposed indications of fire associated with the remains of *A. prometheus* were so equivocal as to leave Oakley unsatisfied that there was any evidence of hearths in that geologic layer.

Thus Oakley's critical studies of the Makapan material reveal *A. prometheus* as an unfortunate misnomer. Whatever the final verdicts regarding the posture, the cerebral status, and other moot characters of the Australopithecinae, it appears reasonably certain that these animals had not captured fire.

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Noncytopathogenic Variants of Poliomyelitis Viruses and Resistance to Superinfection in Tissue Culture*

In the first successful propagation of poliomyelitis virus in tissue culture in 1936, the virus multiplied in the nervous tissues but not in the visceral tissues of the same human embryos (*1*). Ever since unequivocal multiplication in nonnervous tissue was demonstrated 13 yr later (*2*), it had been an intriguing question why the 1936 experiments with nonnervous tissue had failed. Recent experiments have demonstrated that intracerebral passage in monkeys favors the segregation of variants that are devoid of cytopathogenic activity for fibroblasts growing out of monkey testicles in tissue culture but possess full cytopathogenic activity for epithelial cells derived from monkey kidney (*3, 4*). More recently I have found that from the nervous system of infected animals it is possible to segregate variants that have no cytopathogenic effect on epithelial cells derived from monkey kidney (*5*).

Since the "MV" strain of virus that was used in the 1936 cultivation experiments had had a large number of intercerebral passages in monkeys, it appeared possible that it might be a variant that lacked the property of multiplication in nonnervous tissue. A specimen of this virus that had been frozen for many years was found to have no cytopathogenic effect on epithelial cells derived from monkey kidney in any dilution tested, although intracerebral inoculation produced typical paralytic poliomyelitis in a monkey. The freshly passaged virus also produced no cytopathogenic effect on monkey kidney epithelial cells and four blind passages failed to yield a cytopathogenic agent, although in the third and fourth passages a few epithelial cells degenerated and fell off the glass within 4 days after inoculation, whereas the bulk of the growth remained unaffected for 10 to 12 days.

Last year, I observed that a strain of MEF_1 suckling mouse-adapted poliomyelitis virus derived from the viral progeny in a single suckling mouse at the end-point of an intracerebral titration exerted little or no cytopathogenic effect on monkey kidney cells, but after an incubation period of 7 to 12 days the majority of the epithelial cells that had been exposed to the virus (in dilutions up to 10^{-7} to 10^{-8}) had become resistant to the rapid cytopathogenic effect of the parent strain.

Similar tests performed with the MV virus revealed that, after an incubation period of 10 to 12 days in roller tubes, the epithelial cells derived from monkey kidney had acquired a limited resistance to the cytopathogenic effect of 100 TCD_{50} of the homotypic, Type 2 Y-SK strain. At 2 days after challenge, the epithelial cells in the control tubes, as well as in the tubes that received the higher dilutions of the MV virus, were completely destroyed. The epithelial cells in the tubes inoculated with the lower dilutions of MV virus showed little or no change at 2 days, but at 4 days almost all the cells were similarly destroyed. This limited resistance was not observed when a mixture of the "MV" and "Y-SK" viruses was added to normal epithelial cells or when the Y-SK virus was added 1 hr or 6 days after the addition of the MV virus.

It should be noted here that strict neurotropic variants that have recently been segregated in this laboratory from Type 1 and Type 3 viruses have conferred no such resistance on epithelial cells derived from monkey kidney, but it is possible that the challenge viruses may not have been added at the right time. Further work on the mechanism of the resistance produced by noncytopathogenic variants of poliomyelitis virus would be of the greatest interest particularly with respect to the possible operation of a phenomenon comparable to that of lysogenic phage.

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References and Notes

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