which have been made in the past thirty years in the therapy of pneumonia and in the study of the biology of the pneumococcus, the problem of preventing the occurrence of infection has lagged far behind. This question is undoubtedly bound closely to that of the epidemiology of pneumococcal pneumonia, about which so little is known except in the case of localized epidemics, and the findings in such instances do not seem readily applicable to pneumonia as it affects the general population. As Dr. Heffron points out, efforts at the control of pneumonia have been concerned chiefly with therapeutic measures, because procedures designed to prevent the disease have been unsuccessful.

The present volume has been issued at a time when certain of the ideas concerning the treatment of pneumonia are undergoing revision, particularly with reference to the newer sulfonamide preparations which are finding wide-spread use. The publication of Dr. Heffron's book at this time is peculiarly appropriate.

The summary of the data concerning the treatment of pneumonia with specific antiserum and discussion of the theoretical background of this procedure offer a point of view which should not be forgotten in considering newer therapeutic procedures.

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SPECIAL ARTICLES

THE CHROMOSOMES OF THE CHIMPANZEE

IN the past, studies on primate chromosomes have been largely restricted to man, the diploid numbers of only two other species being on record, so far as we are aware. One of these is an unidentified species of brown Cebus monkey from South America which has 54 chromosomes, and the other the common Indian monkey, *Macacus rhesus,* which has 48 chromosomes in both sexes, just as man has (Painter, 1924). Since none of the great apes has been studied, the present note will be of interest, even though our chromosome count must be taken as provisional and the material does not allow us to compare the morphology of the chromosomes in this ape with those of man.

FIGS. 1a—2b

The material was obtained at the Yale Laboratories of Primate Biology at the instance of the director, who subsequently very kindly allowed us to study it cytologically at the University of Texas.[1] The testes of two individuals were removed and preserved in three different fixatives (Flemming's, Bouin's and Helly's fluids), but as no attempt was made to separate the spermatogenic tubules other than making slices of the testes, good fixation is restricted to cells lying immediately adjacent to the cut surfaces. The testes of specimen "Don," twenty-two months old, and known to be sexually immature, showed no maturation stages, whereas those of specimen "Al," estimated age nine years, and sexually mature, exhibited all phases of spermatogenesis. Unfortunately, the fixation of metaphase chromosomes proved inadequate, though we have examined hundreds of plates and our counts are restricted to diakinesis stages when the pairs of homologous chromosomes are undergoing contraction to form the tetrads of the first maturation division. At diakinesis the nuclear wall is still intact with the chromosome pairs attached so that in effect the haploid number of chromosome pairs are arranged about the surface of a hollow sphere. This favors a wide separation of the elements, but their extended condition and irregular form—due in part to chiasmata—sometimes makes it difficult to separate elements which lie one above the other along the side walls of the nucleus. We have followed the practice of selecting the most favorable nuclei, then making a careful drawing and, if necessary, making an interpretation of a questionable complex. When all was complete a tally was made of the chromosome number. On this basis we have never found less than 23 nor more than 25 chromosomes, and nuclei which we consider free of ambiguities show 24 chromosomes, which is very probably

[1] Acknowledgment for assistance in connection with this study is gratefully made to Dr. Edgar Allen, Department of Anatomy, Yale School of Medicine, and Dr. James H. Elder.

the true haploid number. This would give a full diploid number for the chimpanzee of 48 chromosomes, the same as for man, the *Macacus rhesus* and the majority of the other mammals which have been studied.

Figs. 1*a* and 1*b* and 2*a* and 2*b* were made from two of the clearest nuclei which we have found so far. The chromosomes seen in the upper and lower focal planes are separated so as to avoid overlapping in the figures. Each of these nuclei shows 24 chromosomes.

The form of the young tetrads has been studied carefully so that the conspicuous types could be recognized and, also, in the hope of identifying the sex chromosome complex. All elements seem to be bivalent in nature, and one bivalent was markedly like the X-Y sex chromosome complex of man. One can not be sure of this identification, however, until maturation divisions are examined.

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CORTIN-LIKE EFFECTS OF STEROID GLYCOSIDES ON POTASSIUM

THE activity of cardiac glycosides is known to be enhanced by calcium, and one might therefore expect these glycosides to be potassium antagonists. Their relationship to potassium in muscle has been recently investigated.[1,2] With this and certain chemical considerations in mind, we tested the effects of digitalin, strophanthin and ouabain[3] on potassium.

A crucial test was the protection of normal animals against lethal amounts of potassium, by previous treatment with a glycoside. This type of experiment was successful with cats, rats and mice. Details of these experiments must await a more complete report, but with death as our criterion, the end point is certain. Zwemer and Truszkowski[4] previously found it possible to protect against potassium poisoning with adrenal cortical hormone.

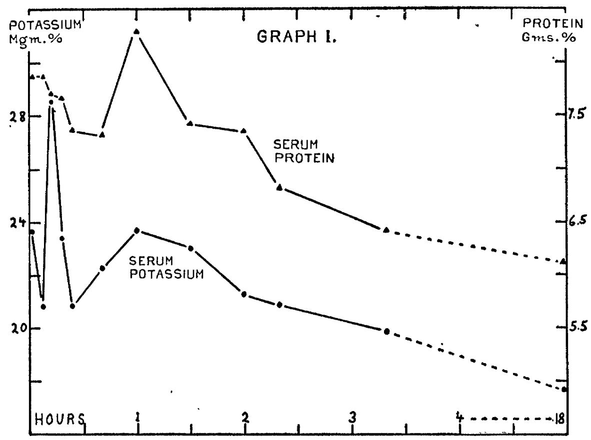
TABLE 1
PROTECTION AGAINST LETHAL KCl WITH STROPHANTHIN[1]

	KCl	Strophanthin	Number of animals	Per cent. survivals
Cats	100 mg/100 g	None	10	0
Rats	150 mg/100 g	None	10	0
Mice	100 mg/100 g	None	32	3.1
Cats	100 mg/100 g	0.015 mg/100 g	6	66.7
Rats	150 mg/100 g	0.200 mg/100 g	29	70.7
Mice	100 mg/100 g	0.150 mg/100 g	126	77.2

[1] Best results were obtained when strophanthin was given 18 to 24 hours before KCl. Protection also has been obtained with digitalin and ouabain.

The reverse, protection against glycoside poisoning with potassium, was not satisfactorily demonstrated.

Lowering of the plasma potassium level and blood dilution after glycoside injection is another measure of its activity. The seven curves so far obtained show effects on the plasma potassium similar to those following injection of adrenal cortex extract (Zwemer[5,6]) or desoxycorticosterone esters.[7]



GRAPH 1. Injection of 0.05 mg of strophanthin per kilogram body weight resulted in an immediate drop in serum potassium followed by a rise to above normal and another decrease. During the first half hour there was a gradual decrease in serum protein. At one hour they both increased, then decreased again and continued to fall for some hours.

The effect of 0.05 mg of strophanthin-in-water per kilogram of cat is given as an example. A significant decrease persisted for at least 18 hours after injection. In previous work we have found that fluctuations frequently appear when one attempts to alter the plasma potassium level. The final low potassium may be due to three factors; blood dilution as shown by the lowered plasma protein content, cell fixation and kidney elimination of potassium.

Prolongation of life in cats deprived of their adrenals (on a diet of salmon and milk with no added salt) is possible with 15 micrograms (.015 mgm) of strophanthin-in-oil per kilogram body weight daily. Withdrawal of material resulted in loss of weight and appetite, followed by death, as is true after withdrawal of corticoadrenal hormone. We realize that the survival of adrenalectomized animals can be accomplished by adjustment of the mineral intake; and with proges-

[1] McK. Cattell and H. Goodell, SCIENCE, 86: 106, 1937.
[2] A. F. Wedd, *Jour. Pharm. Exp. Therap.*, 65: 268, 1939.
[3] We thank the Upjohn Company for a generous supply of these drugs.
[4] R. L. Zwemer and R. Truszkowski, SCIENCE, 83: 558, 1936.
[5] R. L. Zwemer and R. C. Sullivan, *Endocrinology*, 18: 97, 1934.
[6] R. Truszkowski and R. L. Zwemer, *Acta Biol. Exper.*, 12: 1, 1938.
[7] D. Kuhlmann, C. Ragan, J. W. Ferrebee, D. W. Atchley and R. L. Loeb, SCIENCE, 90: 496, 1939.