



Fig. 1-6. Sorex unguiculatus. Métaphase spermatogoniale (1 et 2), cellule diakinétique (du même mâle) (3 et 4) et métaphase I (de l'autre mâle) (5 et 6). Prétraitement hypotonique à l'eau distillée, fixation à l'acide acétique, coloration à Feulgen. ×1700.

S. araneus<sup>2</sup>. Des études plus récentes montrent, que la sexdétermination appartient au type XY<sub>1</sub>Y<sub>2</sub><sup>3</sup>. Le N.F. 44 chez S. araneus est nettement plus bas que le 71 chez S. unguiculatus, tandis que chez Néomys fodiens, par exemple, le N.F. monte à 72. L'espèce ici considérée attire l'interêt surtout par la découverte, à l'intérieur du genre Sorex, d'un nombre chromosomique qui ne peut être expliqué ni comme dérivé de l'espèce araneus conformément à la loi Robertson, ni comme un cas de polyploïdie, bien que la relation des nombres centromériques entre araneus et unguiculatus soit presque exactement de 1:2. Le nombre constaté s'approche en même temps du nombre relativement élevé de plusieurs insectivores et nous semble indiquer la position exceptionnelle de S. araneus avec son nombre chromosomique restreint.

Summary. In Sorex unguiculatus, a species closely related to the common shrew, 41 chromosomes were observed in the spermatogonia of two male specimens. The meiotic cells show about 21 formations at diakinesis and

first maturation division metaphase. The mode of sex determination still remains unsatisfactorily clarified. However, the species investigated is of interest as a further instance of the very complex chromosomal evolution characterizing the genus Sorex. Within this genus, the observed variation in chromosome numbers now ranges from 21 to 41 at the diploid level.

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- <sup>2</sup> R. Bovey, Arch. Julius Klaus-Stiftung 23, 507 (1948). R. Bovey, Exper. 5, 72 (1949).
- <sup>8</sup> G. B. SHARMAN, Nature 177, 941 (1956). C. E. FORD, J. L. HAMERTON et G. B. SHARMAN, Nature 180, 392 (1957). R. MATTHEY et A. MEYLAN, Rev. suisse Zool. 67, 223 (1961).

## Inhibition of Endotoxin-induced Platelet Agglutination, Histamine and Serotonine Release in vitro by Thiol-compounds

It was shown that platelet agglutination can also be observed in vitro by bacterial endotoxins, while some platelet-factors are released 1,2. The basic mechanism of the effect of endotoxin on platelets is not yet clear, and for that very reason, our observation may be of interest that some SH-compounds are able to inhibit in vitro the endo-

toxin-induced platelet agglutination, histamine and sero-tonine release.

To 1.5 ml citrated platelet-rich rabbit plasma<sup>1</sup>, 0.2M neutralized solutions of the substances to be examined were added in different amounts. The AET (=S-2-amino

<sup>&</sup>lt;sup>1</sup> R. M. DES PREZ, H. I. HOROWITZ, and E. W. HOOK, J. exp. Med. 114, 857 (1961).

<sup>&</sup>lt;sup>2</sup> H. I. Horowitz, R. M. Des Prez, and E. W. Hook, J. exp. Med. 116, 619 (1962).

ethyl-isothiouronium bromide) was added after neutralization and incubation at 20°C for 30 min. The final concentration of  $E.\ coli$  endotoxin³ was 100 µg/ml. The mixtures with a final volume of 2 ml were incubated at 37°C for 90 min in polyethylene tubes. After centrifugation, histamine and serotonine determinations⁴ were carried out in supernatants and sediments.

Among the thiol-compounds examined, l-cystein, mercaptoethanol, thioglycolic acid and AET show 20-50% inhibitory effect on the histamine release also in a concentration of 5 mM, while reduced glutathion has such an effect only at 10 mM concentration (see Table). The above-mentioned compounds - with the exception of glutathion - inhibited completely, in a concentration greater than 20 mM, the endotoxin-induced histamine release and platelet agglutination, furthermore the morphological changes: the platelets did not lose their double refracted contours in such a medium under the effect of endotoxin. Such an effect of d-penicillamine (=d- $\beta$ - $\beta$ 'dimethylcystein) was not observed, and likewise the platelet agglutination was not inhibited by ascorbic acid, which is a well-known reducing agent. The above-mentioned compounds inhibited also the serotonine release in a similar manner to the histamine release.

There are data that the endotoxin effect shows in many respects a close similarity to the consequence of the antigen-antibody interaction. These similarities can be well explained with the natural endotoxin hypersensitivity 5,6 and further that normal sera contain also anti-

Inhibition of Inhibition of histamine release Substances agglutination  $20\,\mathrm{m}M$  $5 \,\mathrm{m} M$  $10 \,\mathrm{m}M$ 80-100 20-30 40-60 Cystein 20-30 30 - 40Reduced glutathion 30-50 80-100 100 Mercaptoethanol 100 60 - 8025 - 40Thioglycolic acid 100 30-50 80-100 + AET Ø Penicillamine Ø Ø Ø Ascorbic acid

bodies against endotoxin 7-9. GILBERT and BRAUDE's experiments a call attention to the possible participation of complement. The one possibility in explaining the effect of SH-compounds is that these substances may reduce the natural antibody against endotoxin. The examinations of the structure of antibody demonstrated the importance of the S-S linkage in the function of antibodies 10,11. According to our experiments, the abovementioned SH-compounds inhibited the guinea-pig complement in sensitized sheep's red blood cells; furthermore the cystein inhibited the precipitation of ovalbumin and native ovalbumin antiserum 12. The reducing and dissociating action of some SH-compounds on the S-S linkages is well known in the case of the  $\gamma$ -globulins and macroglobulins 10. On the basis of the above-mentioned data, it is suggested that the inhibitory effect of the thiolcompounds described is due to the reduction of the natural antibodies or other competent macromolecules.

Zusammenfassung. Es wird festgestellt, dass gewisse Thiolverbindungen wie Cystein, Mercaptoethanol, Thioglycolsäure und AET die von Endotoxin ausgelöste Thrombocyten-Agglutination, die Freisetzung von Histamin und Serotonin im Kaninchen-Thrombocytenplasma hemmen.

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Pathophysiological Institute, Medical University of Debrecen (Hungary), December 10, 1963.

- 3 A. Boivin and L. Mesrobeanu, C. R. Soc. Biol. 128, 5 (1938).
- <sup>4</sup> H. Weissbach, T. P. Waalkes, and S. Udenfriend, J. biol. Chem. 230, 865 (1958).
- <sup>5</sup> C. A. Stetson Jr., J. exp. Med. 94, 347 (1951).
- <sup>6</sup> T. G. Kováts, G. Lázár, and P. Végh, Acta physiol. hung. 23, 169 (1963).
- 7 S. SELL and A. I. BRAUDE, J. Immunol. 87, 119 (1961).
- 8 A. I. Braude and I. Siemienski, Bull. New York Acad. Med. 37, 448 (1961).
- <sup>9</sup> V. E. GILBERT and A. I. BRAUDE, J. exp. Med. 116, 477 (1962).
- 10 R. R. PORTER and E. M. PRESS, Ann. Rev. Biochem. 31, 625 (1962).
- <sup>11</sup> A. NISONOFF, F. C. WISSLER, L. N. LIPMAN, and D. L. WOERULEY, Arch. Biochem. Biophys. 89, 230 (1960).
- 12 I. Jókay and M. Kávai, not published.

## Monosynaptic Reflex and Natural Sleep

Since the re-appreciation of the so-called paradoxical phase of sleep<sup>1</sup>, various features of this sleep have been described and possible mechanisms have been suggested<sup>2</sup>. There are certain typical activities in the motor system, such as the silence of neck muscles and occasional twitch-like contractions of muscles, including the rapid eye movement. The present paper will show that the monosynaptic reflex (MSR) discharge from the lumbar spinal motoneurons is depressed during this phase of sleep in the cat.

Pairs of silver electrodes, similar to those used by Swett and Pompeiano<sup>3,4</sup>, were implanted in the tibial or peroneal nerve at the popliteal fossa of the chronic

cat. In order to avoid the contamination of muscle contraction and proprioceptive  $\gamma$  motor activity, nerves were crushed at the distalmost part. Stimulating the central portion of the recording nerve bipolarly, the action potential of reflex origin could be recorded from the distal part of the tibial or peroneal nerve, either bipolarly or monopolarly, with a latency of about 4 msec. Recorded in

<sup>1</sup> W. DEMENT, EEG clin. Neurophysiol. 10, 291 (1958).

<sup>&</sup>lt;sup>2</sup> М. Jouver, Arch. ital. Biol. 100, 125 (1962).

<sup>&</sup>lt;sup>3</sup> O. Pompeiano and J. E. Swett, Arch. ital. Biol. 100, 311 (1962).

<sup>4</sup> S. GIAQUINTO, O. POMPEIANO, and I. SOMOGYI, Exper. 19, 481 (1963).