(2) J'ai publié[3,4] récemment les observations faites sur 12 *M. triton* de même provenance: les 5 ♂♂ montraient 32 chromosomes, tous les autosomes et l'*Y* étant acrocentrique, l'*X*, de grande taille, étant submétacentrique (*I.C.* = ~0,35). 3 des 7 ♀♀ présentaient 2 *X* normaux alors que, chez les 4 autres, il n'y avait qu'un *X* typique, le second, acrocentrique, ayant donc perdu son bras court et sa longueur égalant exactement celle du bras long d'un *X* normal (♀ $X-X_{dc}$). L'échantillon était trop petit pour que l'existence – attendue – de ♂♂ X_{dc}-*Y* et de ♀♀ X_{dc}-X_{dc} pût être exclue. Grâce aux captures faites à Lwiro, l'échantillon se compose maintenant de 39 individus, 18 ♂♂ et 21 ♀♀. Tous les mâles sont normaux (*X*-*Y*); 11 femelles ont la formule *X*-*X* et 10 la formule *X*-X_{dc}. Nous avions fait l'hypothèse, en analysant l'échantillon primitif que la population était concevable en admettant le modèle suivant:

4/9 de ♀♀ *X*-*X*. 4/9 de ♀♀ *X*-X_{dc}. 1/9 de ♀♀ X_{dc}-X_{dc}.

2/3 de ♂♂ *X*-*Y*. 1/3 de ♂♂ X_{dc}-*Y*.

Or, l'absence de femelles homozygotes pour la délétion, plus encore l'absence de mâles porteurs de cette délétion, rendent cette hypothèse dont la probabilité était déjà faible au vu de l'échantillon restreint, intenable. Mais alors, si ces 2 combinaisons sont létales ou non-viables, comment concevoir le maintien du chromosome X_{dc} dans la population?

Il est possible que ce cas soit à rapprocher de celui d'une autre *Leggada* du sud de l'Afrique, *M. minutoides minutoides* Smith ($2N = 18$) dont j'ai étudié[5,6] un échantillon de 23 individus, 13 ♂♂ et 10 ♀♀. 6 femelles étaient porteuses d'une inversion péricentrique intéressant un autosome de la sixième paire, dès lors formée d'un métacentrique et d'un acrocentrique. Chez les 4 autres femelles et chez les 13 mâles, cette sixième paire était invariablement constituée par 2 métacentriques.

Dans les 2 cas, une mutation chromosomique importante ne se rencontre que dans un seul sexe (♀), à l'état hétérozygote, et chez la moitié des représentants de ce sexe. Il y a là un problème apparemment insoluble dans l'état actuel de nos connaissances.

Summary. The author treats 2 topics connected with his studies on the cytogenetics of the African pigmy-mice. (1) *Mus* (*Leggada*) *bufo* Th. shows the 'primitive' complement already detected in other species or subspecies (*minutoides ssp*$_1$, *indutus, tenellus, setulosus*): $2N = 36$, *N.F.* = 36, sex chromosomes *PR*. (2) The deletion of the short arm of one *X*-chromosome (X_{dc}) was previously observed as heterozygous mutation by 4 ♀♀ in a sample of 7♀♀ and 5 ♂♂. A more important sample (18 ♂♂, 21 ♀♀) gives the following results: 11 ♀♀ *X*-*X*, 10 ♀♀ *X*-X_{dc}, 18 ♂♂ *X*-*Y*. The occurrence of ♀♀ X_{dc}-X_{dc} and of ♂♂ X_{dc}-*Y* is also very improbable. It is not easy to understand how the chromosome X_{dc} may persist in the population if the complements X_{dc}-X_{dc} and X_{dc}-*Y* are lethal, which seems to result from these observations.

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Modifications of RNA-Containing Structures of Dorsal Bulbocavernosus Muscle of Rat after Castration and Testosterone Administration

A promising object for analysis of the protein synthesizing process in skeletal muscle is afforded by rat levator ani muscle. This, according to HAYES[1], should more properly be named bulbocavernosus.

Castration of male rats causes a rapid atrophy of this muscle (WAINMAN and SHIPOUNOFF[2], GORI and PELLEGRINO[3]). Subsequent administration of testosterone brings about a biphasic increase in its weight and total N content (BERGAMINI, BOMBARA and PELLEGRINO[4]), which shows that protein synthesis is taking place under hormonal stimulation.

In these experimental conditions, RNA-containing structures of the muscle fibres, which are known to take part in protein synthesis, undergo marked modifications. These have been studied by electron microscopy.

Early modifications were observed both in the nucleolus and in the cytoplasm. The former is notably reduced in volume in the castrated rats and is composed mainly of the fibrillar phase, whereas the nucleolar ribosomes, conspicuous in normal rats, almost disappear.

Already, 24 h after beginning the testosterone treatment, the nucleolus is much enlarged[5]. The nucleolus shows a more abundant fibrillar phase disposed in a loose reticular shape, and the granular faction (nucleolar ribosomes) is evident. These modifications progress subsequently.

As regards the cytoplasm, while modest amounts of ribosomes and polyribosomes are present in the normal muscle, they are extremely reduced in the atrophic muscle. Already, 24 h after beginning testosterone treatment, in the sarcoplasmic spaces increasing numbers of ribosomes and polyribosomes are detected (Figures 1 and 2). Among the smaller polyribosomes, pentagonal structures were frequently seen. Those of a greater size showed a linear or a helical array of ribosomes, sometimes up to 20 (Figure 3). In fact, in a ribosomal fraction from muscle, polyribosomes composed of up to 60–100 units were observed[6].

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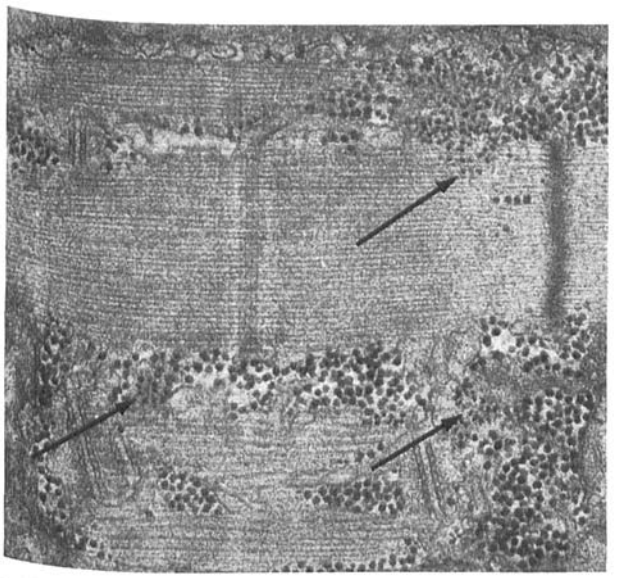

Fig. 1. Castrated muscle, 24 h after testosterone injection. Numerous ribosomes and polyribosomes (arrows) are visible; × 32,500.

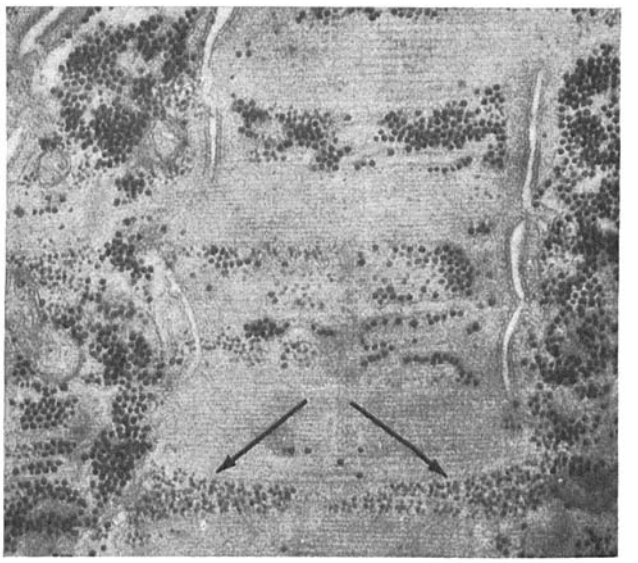

Fig. 2. Castrated muscle, 4 days after testosterone injection. Further marked increase of ribosomes and polyribosomes particularly evident in the interfibrillary spaces (arrows); × 37,000.

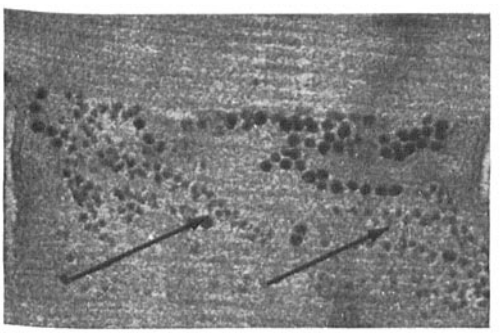

Fig. 3. Castrated muscle, after 4 days of testosterone treatment. Long polyribosomes (arrows) at the periphery of a myofibril; × 46,700.

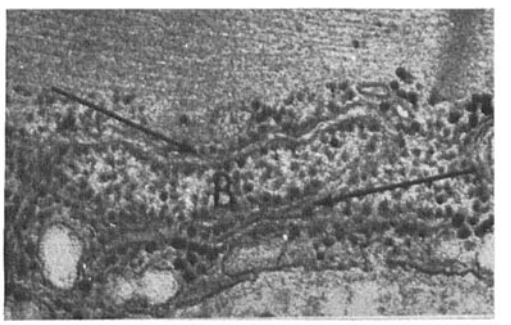

Fig. 4. Castrated muscle, after 4 days of testosterone treatment. An elongated body (B) containing numerous ribosomes and surrounded by a cisterna with partly granular membrane (arrows); × 53,000.

Besides these more common structures, round or elongated bodies, containing ribosomes and surrounded partially or completely by cisternae with partly smooth and partly granular membranes, were frequently seen (Figure 4); they can be assimilated to rough endoplasmic reticulum and have been very rarely detected also in normal dorsal bulbocavernosus muscle. The maximum increase of ribosomes in the cytoplasm was observed on the fifth day of treatment.

These observations indicate that on subtraction of the specific hormonal trophic stimulus and its restoration, dorsal bulbocavernosus muscle undergoes marked and opposed variations respectively in its protein synthesizing machinery. It has been shown that castration causes a decrease of the rough endoplasmic reticulum both in seminal vesicles[8,9] and in the epithelial cells of the prostate[9].

It is evident that, at least in dorsal bulbocavernosus muscle of the rat, the cytoplasmic modifications after castration are accompanied also by reduction in nucleolar RNA-containing structures.

Our observations are in agreement with biochemical data which indicate that testosterone causes an RNA increase[7] in androgen-sensitive tissues, in the prostate and in the temporal and masseter muscles of guinea-pigs[10]. They also give evidence on the precise intracellular localization of such increases[11].

Riassunto. Nel muscolo bulbocavernoso dorsale (elevatore dell'ano) di ratto per effetto della castrazione, e rispettivamente della susseguente somministrazione di testosterone, si producono notevoli modificazioni, in senso inverso, in strutture contenenti RNA e che sono importanti per la sintesi proteica (nucleoli e ribosomi citoplasmatici).

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