Tableau comparatif de la consommation d'oxygène (moyenne cm^3/g par h) de poissons *Aequidens latifrons* normaux, traités par la thyroxine et la thiourée réunis en groupes de poids environ égal. Différence en pourcentage par rapport aux normaux

Groupe	Poids en g	Durée de traitement en jours		Différences en % des traités thyroxine par rapport aux témoins	Valeur de «t»		Différences des moyennes	Thyroxine	Témoin	Thiourée	Différences en % des traités thiourée par rapport aux témoins	Valeur de «t»		Différences des moyennes
		Thyroxine	Thiourée		calculée P 0,05	théorique						calculée P 0,05	théorique	
I	1,20–1,50	70	170	+ 0,9			non	0,215	0,213	0,173	− 18,7	2,50	2,57	signi-
II	1,80–2,30	140		+ 12	1,46	2,45	signi-	0,187	0,167					fica-
III	2,40–2,80	163	142	+ 15,8	2,24	2,36	fica-	0,168	0,145	0,109	− 24,3	4,27	3,18	tive
IV	2,90–3,30	198	70	+ 13,3	1,83	2,45	tive	0,144	0,134	0,101	− 24,6	6,31	2,57	
V	3,75–4,10	245		+ 35,7	5,25	3,18	signi-	0,135	0,095					
VI	4,25–4,75	265		+ 22,3	3,57	2,78	fica-	0,115	0,094					
VII	5,70–6,20	286		+ 33	3,97	2,78	tive	0,105	0,079					

L'élévation de la consommation d'oxygène coïncide avec l'apparition des changements histologiques du tissu thyroïdien et l'arrêt de la fixation du radio-iode-131 dans la glande thyroïde.

Ces résultats montrent qu'après un temps de latence long, l'apport continu de thyroxine provoque chez les Poissons une augmentation de la consommation d'oxygène.

Les échanges respiratoires semblent être régis par la thyroxine exogène lorsque la thyroïde est complètement inhibée.

Le traitement par la thiourée provoque dès le 70e jour une baisse élevée de la consommation d'oxygène de l'ordre de 24,6% (hautement significative, tableau). Cette diminution des échanges respiratoires entre les traités et les témoins subsiste jusqu'à la fin de l'expérience.

Les tests historadiographiques permettent de conclure que la fixation de l'iode au niveau des vésicules thyroïdiennes est supprimée. La liaison organique de l'iode est bloquée et la synthèse de l'hormone thyroïdienne est inhibée; leurs incidences sur les échanges respiratoires sont aussitôt constatées. La consommation d'oxygène est fortement diminuée. Mais la diminution ne s'avère pas durable.

La thyroïdectomie totale par le radio-iode-I-131 en cours d'expérience, chez les Cichlidés, permettra de comparer les effets sur les échanges respiratoires des poissons avec ceux obtenus après un blocage partiel de l'activité thyroïdienne.

Summary. The study of the relationship between the activity of the thyroid of Cichlid fishes and oxygen consumption has given the following results: The stimulation by thyroxine causes a significant increase of oxygen consumption (+ 35%) after a long treatment. This increase is not immediate; a latent period is observed till the gland is in repose. The antithyroid rapidly decreases the oxygen consumption (− 24%). But this chemical blocking is not stable.

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Spontaneous Transmitter Release from Motor Nerve Endings in Muscle Fibres of Castrated and Old Animals

Many types of atrophy of muscle with intact nerve supply have been described under the common heading of 'disuse' atrophy[1,2], in which a decrease or lack of nerve impulse activity can be assumed. The differentiation of this type of atrophy from denervation atrophy should provide evidence for a specific neurotrophic influence of nerve on muscle relatively independent of nerve-impulse activity. For such differentiation, the use of an adequate model of disuse atrophy is necessary. Most of the experimental models used hitherto employed interference especially with the activity of motoneurones, or with vasomotor mechanisms[2]. The atrophy following castration (i.e. lack of adrogenous steroids) of the levator ani (LA) muscle of the rat appears to be suitable model of disuse atrophy, as the male sex hormone can be considered to have a permissive effect on muscle activity[3]. The metabolic changes in the 'desandrogenized' and the denervated LA muscle are qualitatively different[3]. Another type of marked 'disuse' muscle atrophy is found in muscles of old animals[4].

No data, however, are available concerning the changes in spontaneous transmitter release, i.e. ACh which has been thought to be the neurotrophic agent responsible

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	N	C	O
Frequency (sec^{-1})	1.69 ± 0.19 (5931)	1.74 ± 0.13 (6112)	0.30 ± 0.05 (1888)
Amplitude (mV)	0.78 ± 0.20 (98)	1.65 ± 0.20 (85)	1.68 ± 0.23 (40)
Rise time (msec)	0.9 ± 0.17 (20)	0.9 ± 0.21 (14)	0.9 ± 0.23 (10)
Half-decay time (msec)	1.7 ± 0.25 (20)	1.6 ± 0.31 (14)	1.5 ± 0.21 (10)
Resting potential	78.7 ± 0.59 (75)	78.0 ± 0.70 (30)	77.3 ± 0.5 (33)

Characteristics of m.e.p.p.'s recorded from muscle fibres at the end-plate zone of normal (N) castrated (C) and muscles of old animals (O). Numbers in brackets indicate numbers of evaluated m.e.p.p.'s

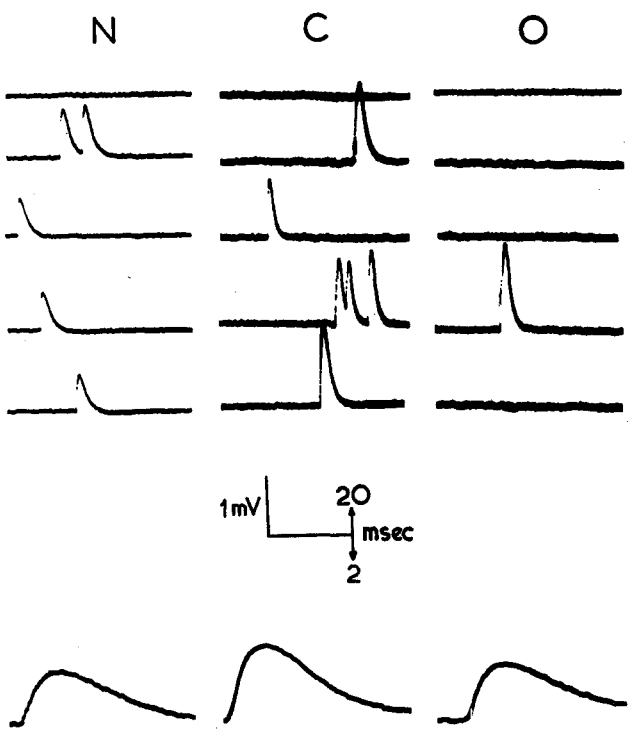
for the maintenance of structure and function of muscle[5,6]. We have therefore compared frequency and other properties of the miniature end-plate potentials (m.e.p.p.'s) in the LA muscle of normal castrated and old (28–32-month-old male rats).

Material and methods. The LA muscle of (a) normal 3-month-old animals, (b) animals 2 months after castration performed at the age of 1 month and (c) animals 28–32 months old were dissected and placed in a flat chamber, submersed in physiological Ringer solution[7]. 3 animals were used in each series and m.e.p.p.'s from 15–30 muscle cells were recorded in each case. M.e.p.p.'s were recorded by the usual microelectrode technique[8]. Frequency of the m.e.p.p.'s/sec was photographed off the screen of an oscilloscope and counted by a Tessla decadic counter.

Results and discussion. The Table and the Figure show that there is no difference in the time course of the m.e.p.p.'s and of the resting potential between normal, 'desandrogenized' muscle fibres (i.e. after castration) and muscle fibres of old animals. The reduction of the muscle fibre diameter after castration and in old age atrophy is reflected in an increase of m.e.p.p.'s amplitude approximately by 50%, as it is a well known fact that the amplitude of m.e.p.p.'s depends on the fibre diameter. No difference could be found in the frequency of m.e.p.p.'s from normal muscle fibres and from muscle fibres after castration. Disuse after castration thus does not reduce spontaneous transmitter release and the latter cannot therefore be responsible for the maintenance of the basic trophic condition of the muscle. However, there was a significant 85% decrease in m.e.p.p.'s frequency in muscle fibres of old animals as compared with muscle fibres of young ones. Castration disuse atrophy is therefore not due to a reduction or lack of spontaneous transmitter release. The latter is, of course, lost after denervation[9] and there is thus a basic difference between denervation and castration disuse atrophy. However, denervation muscle atrophy cannot be explained by lack of impulse directed release of ACh, as postsynaptic block of neuro-muscular transmission does not reproduce denervation changes[10]. The existence of specific 'neurotrophic agents', so far chemically not defined but not identical with the transmitter, therefore seems plausible.

The atrophy in muscles of old animals presents a different picture, as both disuse changes and changes analogous to denervation are observed[4]. Although no decrease in the number of motor neurones could be found, the defficiencies in proteosynthesis in these neurones and apparently a decrease in proximodistal axonal flow may account for disturbances in transmitter synthesis. However, the dramatic decrease in spontaneous transmitter release could also be due to membrane changes of the vesicles and the axonal terminals.

It can, however, be concluded, that disuse muscle atrophy after castration is not due to a reduction of spontaneous transmitter release[11].



Oscillographic recording of m.e.p.p.'s from a muscle fibres of a 3 months old normal (N) animal, from a muscle of a 3 months old castrated (C) and from a muscle of an animal 28 months old (O). Recordings were done at room temperature.

Zusammenfassung. Die spontane Transmitterfreisetzung von Nervenendigungen wird in stark atrophischen Muskelfasern des M. levator ani alter Tiere stark erniedrigt, nach Kastration aber normal gefunden. Die Atrophie nach Kastration wird als eine Inaktivitätsatrophie interpretiert und nicht als Folge einer Verminderung der spontanen Transmitterfreilegung. Im Gegensatz dazu führen denervationsanaloge Veränderungen bei der Altersatrophie zu starker Reduktion der Transmitterfreisetzung.

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