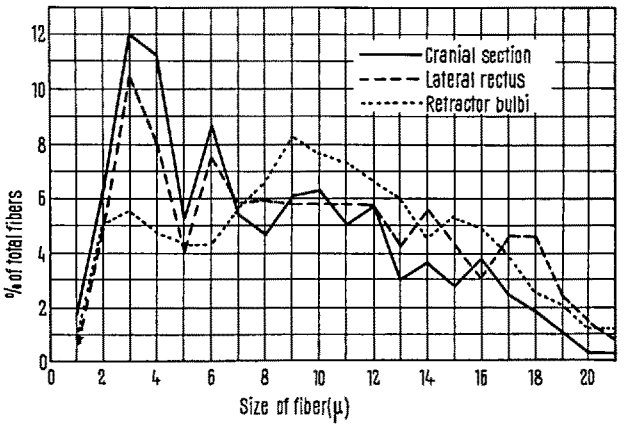

Fiber diameter in 1 μ size increments plotted as % of total number of fibers in the nerve. Each value is the average of measurements at that diameter in 4 nerves. Average range of variability at any fiber size was 4.89% for the retractor bulbi, 5.68% for the lateral rectus, and 2.18% for the cranial sections. Statistical evaluation courtesy of Dr. J. HARRISON.

Total fiber count of 4 VI nerve preparations

Nerve	Cranial		Lateral rectus		Retractor bulbi	
	Total fiber count	Thinly myelinated	Total fiber count	Thinly myelinated	Total fiber count	Thinly myelinated
1	1840	432	2074	425	308	27
2	1610	375	2035	370	190	16
3	1590	250	1704	240	320	12
4	1456	–	1629	–	201	–

Fiber counts of 4 nerves at the 3 areas of section. Thinly myelinated figures were not determined for nerve No. 4 because of poor photographic clarity.

naked axon to myelin sheath was determined to be 3.1:1 (S.D. ± 0.58) for the thickly myelinated fibers, and 8.1:1 (S.D. ± 0.49) for the thinly myelinated fibers. The proportion of thinly myelinated fibers in the total population of cranial fibers was 16–23%, and, in the branch to the lateral rectus, 14–20%. In contrast, the proportion in the branch to the retractor bulbi was 4–8% of the total population. The average of 30% increase in fiber number from nerve trunk to branches is seen predominantly in the thickly myelinated fibers and does not occur to any appreciable extent in the thinly myelinated fibers (Table).

By weight, the superior lateral slip constituted an average of 25% of the total muscle. To determine the innervation ratio to the retractor bulbi, ALVARADO's count of 3118 muscle fibers at the midregion of the muscle in 1 superior lateral slip was utilized. Assuming this represents 25% of the fibers, one can estimate that the 4 slips of the retractor bulbi muscle contain approximately 12,472 muscle fibers. The average nerve fiber count of the VI branch to the retractor bulbi was 255 (Table). From these figures an approximate innervation ratio of 1:50 can be estimated for the retractor bulbi[5].

Zusammenfassung. Das Faserspektrum des VI Hirnnerven der Katze wurde an 3 Stellen bestimmt: Austrittsstelle aus dem Pons, Verzweigungsstelle zum M. rectus lateralis und zum M. retractor bulbi. Wenigstens 3 verschiedene Typen motorischer Fasern wurden ermittelt: 1. grosse, dick myelinisierte, 2. kleine, dünn myelinisierte, 3. kleine, dick myelinisierte. Die Verzweigungsstelle zum M. retractor bulbi besteht hauptsächlich aus dem 1. Fasertyp.

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The Mode of Formation of Intracytoplasmic Structures in Tumours Induced by Viruses of the Avian Sarcoma Leucosis Group*

In the cytoplasm of avian tumours produced by the viruses of the avian sarcoma leucosis group, structures are sometimes electron-optically observed whose development and relation to the tumour growth and virus multiplication is unknown. Morphologically these aggregates of varying size, in which ribosome-like particles are arranged in a more or less homogeneous matrix with both components often showing the development of spheres or incomplete spheres. Furthermore, these formations contain virus nucleoid-like corpuscles[1–4].

The following considerations render it very probable that these aggregates are antigen-antibody precipitates of the living cells. The size and the localization of the spatial arrangement is ambiguous. The sphere formation of antigen-antibody complexes has been electron-optically described by models[5]. The ribosome aggregates often described as viroplasm can be especially observed close to the cell membrane and often without simultaneous indications of virus production by budding. As in the formation of secondary lysosomes the smaller immune precipitates are contracted to several larger ones[6]. Whereby the mass is not enclosed by a membrane; a sign that

* This paper is dedicated to Prof. H. LETTRÉ on the occasion of his 60th birthday.

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the cell does not treat the structures as foreign to it. The antibodies must have passed the cell membrane unaltered which is, however, only possible if the normal function of the cell membrane has been damaged[7].

Except in nephroblastomas, all authors mention the rare occurrence of the described intracytoplasmic structures in various tumour cells. In the nephroblastomas they are often observed, and only in the podocytes of the glomerulus and especially in the less differentiated cells[3]. The importance of the podocytes for the normal functioning of the kidney and the effects which a change in their permeability has, is known[8]. Swelling of the mitochondria and damage of the cristae in the podocytes of kidney tumours have been described[3]. Localization, morphology and function of these cells form the preconditions of an intracytoplasmic immune precipitation in vivo, under the pathological conditions described.

The morphology of antigen-antibody complexes suggests antibody formation against virus structures, ribosomes and ribosome mRNA. That the ribosomes appear smaller after pepsin digestion is explained by the breaking of RNA from the ribosomes surface[9], whose organization was recently newly formulated[10].

The immune aggregates have hitherto only been observed in systems that also permitted the presence of antibodies; a precondition that is also given by cells in vitro. Besides the natural occurrence of antibodies in the medium, immune competent small lymphocytes are often present in cultures of myeloblastose virus infected myeloblasts of chickens[11]. The only known exception was found in chicken cells infected by Rous sarcoma virus in vitro in a heterologous medium[12]. Apart from one rather uncertain exception[13], no Rous sarcoma virus neutralizing factor has been found up to now in the serum of mammals[14]. The first observation of the intracytoplasmic enclosures was made in tumours of the hatched chickens, produced by high doses of Rous sarcoma virus[15].

Zusammenfassung. Die durch Viren der Vögel-Sarkom-Leukose-Gruppe hervorgerufenen intrazytoplasmatischen Strukturen sind sehr wahrscheinlich Immunpräzipitate.

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Blockade of Ovulation in the Rat Initiated by an Antimetabolite

3-Acetylpyridine (3-AP) and several structurally related antimetabolites'[1] interfere with the coenzyme function of pyridine nucleotides by competing with nicotinamide for sites within the molecule[2,3]. The toxicity of 3-AP to mammals is to a certain extent determined by the dietary intake of nicotinamide, nicotinic acid or tryptophane[1,3] and is neutralized, in the acute experiment, by equimolar amounts of nicotinamide[1] or diphosphopyridinenucleotide (NAD)[3].

Coenzymes of the nucleotide type play a role in ovarian steroidogenesis in mammals, but it is controversial whether they directly mediate the steroidogenic action of luteinizing hormone (LH)[4,5] or whether the conversion of steroids observed in their presence is independent of LH[6,7].

In this study, 3-AP has been found to interfere with ovulation in the intact rat if administered at appropriate times during vaginal pro-oestrus. Although there is no evidence for a specific effect of 3-AP on ovarian pyridine nucleotides (as opposed to those located in other body compartments), a fairly high degree of temporal specificity with respect to reproductive events is achieved by the rapidity of interchange between 3-AP and nicotinamide on the one hand and by the well-defined 'critical period' for LH release in rats exposed to controlled conditions of illumination[8] on the other.

3-AP[9] was diluted 1:100 v/v with distilled water and injected i.p. into adult female rats of the Wistar strain at various times during pro-oestrus. The animals were exposed to 14 h of light and 10 h of darkness and fed a standard diet containing 2.5 mg nicotinic acid/kg. All exhibited regular 4-day cycles as assessed by daily vaginal smears. 3-AP was administered at a dose level of 0.6 ml (approximately 54 μmoles)/100 g body weight. This dose was close to the LD_{25} when used without subsequent antidote. Ovulation was assessed by counting tubal ova during the following morning (oestrus).

Tubal ova were absent in all cases in which the antimetabolite was administered 3 h or more prior to the onset of the 'critical period' (Table I). In view of this, 3-AP was injected at − 4 h in all subsequent experiments. The blocking effect of 3-AP was counteracted by equimolar doses of nicotinamide, irrespective of whether this

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