the Latin stem: *simul-* = -like, as in *simulare* = simulate), and the suffix *-mime* (from Greek stem: $\mu\iota\mu\eta$ = imitate, as in pantomime), appear best suited. Of these two, *sim-* (or *simu-*) is the more difficult to identify in formations such as: simuneuron, simunephron or simoculus (artificial eye).

I would, therefore, propose that the suffix *-mime* be adopted to designate artificial devices simulating biological functions. The suffix should be understood to mean that the device under discussion belongs to this general class of artifices. Thus, the *-mime* ending can be considered to yield a generic name, whereas authors are, of course, free to name their inventions by any specific name they please. So ARKID is a species of *nephromime*, cardiotron is a *cardiomime*, and the Perceptron (a mathematical model) is an *oculomime* or *neuromime*, depending on one's emphasis. The various types of transistorized artificial neurons are all neuromimes, and so are nets of computer-simulated models.

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Acetylcholinesterase Regeneration in Peripheral Nerve after Irreversible Inactivation

Abstract. The return of acetylcholinesterase activity was studied in several cholinergic structures in the cat after irreversible inactivation by di*iso*propylfluorophosphate. It was found that enzymic activity returned uniformly along the hypoglossal and cervical sympathetic nerve trunks. No evidence for somatoaxonal convection of enzyme was obtained.

It is widely held that axonal protein is formed in the perikaryon of the neuron and conveyed peripherally by centrifugal displacement of neurocytoplasm or axoplasmatic currents. This conception developed first as a result of the comprehensive studies of Weiss and Hiscoe (*1*), which suggest the phenomenon of somatoaxonal convection of axoplasm. Secondly, when the essential role of ribonucleic acid in protein synthesis became appreciated, its absence from the axon in the form of visible aggregates of ribonucleoprotein (variously termed Nissl substance, granular endoplasmic reticulum, and ribosomes) lent further credence to the concept of dependence on the perikaryon for biosynthesis of protein for the remainder of the neuron. However, it might be pointed out that the complete absence of ribonucleic acid from the normal, mature axon has never been proven, and that ribonucleic acid is certainly not absent from embryonic nerve (*2*).

The evidence that enzymes, specifically cholineacetylase and acetylcholinesterase, migrate distad in the axon after their formation in the cytoplasm has been suggestive, albeit somewhat tenuous. Thus, Hebb and Waites (*3*) and Sawyer (*4*) demonstrated an increase in enzymic activity of cholineacetylase and acetylcholinesterase, respectively, in the regenerating proximal stump of peripheral nerve after sectioning. In addition, Lewis and Hughes (*5*), using a nonspecific histochemical method, demonstrated that cholinesterase is present in the outgrowing embryonic nerve of the toad *Xenopus laevis*. However, a histochemical study by Schwarzacher (*6*) of acetylcholinesterase content in neuronal somata during peripheral nerve regeneration indicated that this enzyme is not present in the perikarya during the outgrowth period. The last-mentioned finding controverts the validity of the conclusion, generally drawn in previous studies by many workers in the field, of somatoaxonal migration of acetylcholinesterase. The observations of Sawyer (*4*) and Schwarzacher (*6*), considered together, suggest rather that the biosynthetic systems for acetylcholinesterase may migrate only during growth (that is, during regeneration and embryogenesis). If the dissolution of Nissl substance (chromotolysis) which invariably follows axonal sectioning is regarded as the mobilization of ribonucleic acid for its axonal passage, this phenomenon lends further support to the latter hypothesis.

The most direct evidence for axonal migration of protein was obtained by H. Koenig (*7*) and Waelsch (*8*), employing intrathecal administration of isotopically labeled amino acids in the cat and frog, respectively. "Peaks" of radioactivity in peripheral neuronal protein were observed by Koenig to be displaced peripherad in the ulnar and sciatic nerves at rates ranging from 2 to 11 mm/day.

Fukuda and Koelle (*9*), in a histochemical study, using the thiocholine method of Koelle (*10*), showed that the cytoplasmic acetylcholinesterase of the neurons of the cat ciliary ganglion has a distribution pattern closely resembling that of the Nissl substance. In addition, following inactivation by di*iso*propylfluorophosphate, the regenerating acetylcholinesterase appeared in the cytoplasm before its presence in the surrounding preganglionic axonal terminals could be detected. Such observations support the general contention of somatic synthesis of the enzyme prior to its transit to the periphery.

On the assumption that the "somatoaxonal convection hypothesis" is true for acetylcholinesterase, it should be possible to demonstrate a proximodistal "activity" gradient of the enzyme along the nerve after its irreversible inactivation. In order to test this hypothesis, a high dose (40 μmole/kg, given intravenously) of the irreversible anticholinesterase agent, di*iso*propylfluorophosphate, was administered to anesthetized cats previously treated with atropine (5 mg/kg), and the regeneration of acetylcholinesterase was followed over a period of 1 to 15 days in selected cholinergic structures. The enzyme activity was assayed by a modification of the method of Bonting and Featherstone (*11*), with methacholine as substrate. The following structures were studied: (i) hypoglossal nucleus; (ii) three successive segments of peripheral hypoglossal trunk; (iii) two successive segments of the distal portion of the cervical sympathetic trunk; (iv) superior cervical ganglion (acetylcholinesterase localized chiefly in cholinergic axonal terminals); (v) ciliary ganglion (acetylcholinesterase localized chiefly in neuronal cell bodies); and (vi) the inferior oblique muscle of the eye. The structures were selected to yield as complete a picture as possible of the regeneration of acetylcholinesterase in cholinergic somata, axons and axonal terminals, and a nonneuronal tissue.

Figure 1 shows the patterns of acetylcholinesterase regeneration observed in the structures studied. Of primary

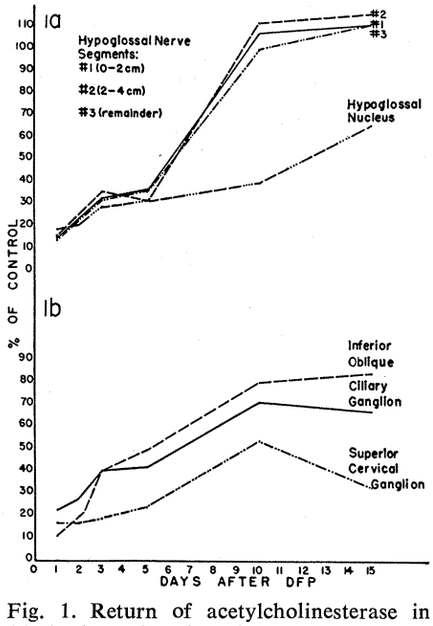

Fig. 1. Return of acetylcholinesterase in (*a*) the hypoglossal nucleus and nerve, and (*b*) the superior cervical and ciliary ganglia and inferior oblique muscle, after irreversible inactivation by di*iso*propylfluorophosphate. The time intervals of the study were 1, 2, 3, 5, 10, and 15 days. Each point represents the average for three animals with the exception of the point for the 2-day period, which represents the average for two animals.

significance is the uniform rate of regeneration of acetylcholinesterase along the entire length of peripheral hypoglossal nerve (Fig. 1*a*) at the time intervals of the study. A uniform rate of regeneration of the enzyme was found, also, for the cervical sympathetic trunk. However, unlike the finding for the hypoglossal nerve, the acetylcholinesterase of the cervical sympathetic trunk was regenerated only to approximately 45 percent by the 15th day. A possible explanation for this difference may lie in the fact that the cervical sympathetic trunk has approximately 15 times as much acetylcholinesterase activity per mg wet weight as the hypoglossal. Nonetheless, it is highly improbable that the regeneration of acetylcholinesterase is a result of spontaneous hydrolysis, since evidence indicates convincingly that once the ageing of the phosphorylated enzyme is complete (within 1 day), it is irreversibly inactivated (*12*). Even if the highest rate (11 mm/day) reported by H. Koenig (*7*) for axoplasmic flow in some fibers of the sciatic nerve is assumed to obtain in the hypoglossal nerve, axoplasmic flow is far too slow to account for the uniform return of enzyme along the trunk.

Additional evidence for the relatively independent nature of the peripheral return of the enzyme may be inferred from a comparison of the curves for the hypoglossal nerve and its nucleus (Fig. 1*a*). It can be seen that no gradient exists between the nucleus and peripheral nerve; in fact, a reverse gradient is in evidence between days 5 and 15. It is noteworthy that the total acetylcholinesterase contents of the nucleus and of the whole trunk are of the same order of magnitude; hence, the amount of enzyme in the peripheral nerve is not just a small fraction of the cellular enzyme.

The pattern of regeneration in the superior cervical ganglion (Fig. 1*b*) appears somewhat anomalous. Since most of the acetylcholinesterase is associated with the preganglionic terminals (*13*), its pattern of regeneration might be expected to be similar to that of peripheral nerve. However, no significant return was noted until the 5th day. This may account for the difference in the time of appearance of enzyme in the cell bodies and in the preganglionic terminals of the ciliary ganglion after di*iso*propylfluorophosphate inactivation (*9*) (see above). The reasons for these differences can be only a matter of conjecture at present. Furthermore, although the average enzymatic activity appears to decline between days 10 and 15, the decline is probably apparent rather than real, since the small number of experimental animals, coupled with wide variation in normal, control values for the superior cervical ganglion, probably accounts for the apparent reduction. The pattern of regeneration in the extraocular muscle (Fig. 1*b*), on the other hand, is similar to what other workers have reported (see Denz, *14*).

Experiments are now in progress in which an attempt is being made to dissociate the regeneration of acetylcholinesterase in the peripheral nerve trunk from that in the nucleus by chronic suppression of the build-up of enzyme at the latter site with periodic intraventricular injections of di*iso*propylfluorophosphate through an indwelling cranial cannula. The preliminary data appear to substantiate the present observations, which suggest that the peripheral enzyme regenerates relatively independently of the cell body. Whether the return of acetylcholinesterase activity along the trunk is a result of newly synthesized enzyme (that is, *de novo* synthesis of protein) occurring in the periphery or a function of one or more conceivable mechanisms of regeneration remains to be elucidated fully (*15*).

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29 April 1960

Relation of Jupiter's Radio Emission at Long Wavelengths to Solar Activity

Abstract. Since the spring of 1960 a strong positive correlation between Jupiter's decametric emission and solar decametric continuum emission observed at Boulder has been evident. The time delay of 1 to 2 days, with solar emission preceding Jupiter's emission, suggests that fast solar corpuscles, at velocities of the order of 0.1 *c*, are directly involved in the planet's atmosphere or magnetic field.

The rate of occurrence of Jupiter's long wavelength emissions appears to have decreased from the time of the discovery observations in 1955 through the period of sunspot maximum, and only this spring has it shown signs of returning to its initial high level. On records made in 1951, Shain (*1*) found many instances of Jupiter emission, following by several years the maximum of the sunspot cycle in 1947. The suggested anticorrelation of Jupiter's emission with sunspot number (*2*) indicates that relatively heavy ionization of Jupiter's atmosphere at sunspot maximum may mask a deep-seated source of emission.

An ionosphere on Jupiter has been widely invoked to explain both the strong polarization of the bursts and the directivity of the source or sources. The magnetic field conditions where the radiation emerges from the ionosphere can be deduced from such observations, and, on the assumption of a limiting cone of emission, the plasma density may also be estimated (*2*, *3*, *4*).

The High Altitude Observatory initiated, on 28 January 1960, a series of observations of Jupiter's decametric emission that have continued until the present. In the period from 28 January to 28 June, emission was detected on 30 separate rotations of the planet. The emission was positively identified and separated from sources of radio interference by the characteristic diurnal motion of the source on our swept-frequency interferometric records. The range of the observations is 15 to 34 Mcy/sec, covered on most of these records in 0.7 second. The total period of observations represented in this interval is about 700 hours, and the minimum detectable flux density at 18 Mcy/sec is about equal to that from the radio source Cassiopeia A—that is, 5×10^{-22} web m^{-2} $(cy/sec)^{-1}$ (*5*).

It was soon apparent on our records that a general correspondence exists between the days of detection of Jupiter's emission and the level of solar activity observed with the radio spectrograph. In particular, strong solar activity in the intervals of 28 March to 4 April and 21 April to 14 May was accompanied by several outstanding occurrences of Jupiter emission. To establish the sug-