from 2 to 9 mM. Mn$^{2+}$ had about 25% the activity of Mg$^{2+}$. With Mg$^{2+}$ in excess, ADP activated the kinase up to 5-fold as measured by colorimetric or radiotracer assay. With ATP at 5 mM and Mg$^{2+}$ at 15 mM, activity increased with ADP up to 5 mM. If phosphoenolpyruvate plus pyruvate kinase were added to controls, reconverting ADP into ATP, their activity was decreased and an 8-fold stimulation of activity by ADP was observed. Stimulation was due to an increase in the $V_{\text{max}}$ for the reaction rather than a decrease in the $K_m$ for either ATP or the substrate. It was noted that high concentrations of ATP relative to ADP often decreased the kinase activity, even with excess of Mg$^{2+}$ present. The ratio of coenzyme to effector appeared to be important and the effect of varying the molar ratio [ATP]/([ATP]+[ADP]) on activity was measured at various total nucleotide concentrations. Curves similar to that shown in Fig. 2 were obtained.

These results show that *Pseudomonas* sp. N.C.I.B. 8858 contains an ATP-1-amino-propan-2-ol phosphotransferase, the L-isomer being the preferred substrate. Modulation of activity by ADP (and ATP) indicates a catabolic role for the kinase, probably in aminoacetone catabolism. A biosynthetic role in phospholipid formation has been postulated for an analogous enzyme from rat liver mitochondria (Willetts, 1974), which in contrast is unaffected by ADP, Mg$^{2+}$ or EDTA and is virtually inactive with ethanolamine.

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Compartmentation of Amino Acids and Change in the Fate of Radioactive Glucose in Rat Brain after Electroshock and Reserpine Pretreatment

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Electroshock therapy is regularly used as treatment against depression and various other psychiatric disorders. Pretreatment with the 'major tranquillizer' reserpine potentiates the effect of electroshock by inducing convulsions in rats at lower currents than in controls. Electroshock induces a sequence of behavioural changes, i.e. tonic/clonic convulsions, decreased motility and extinction of conditioned reflexes in animals, the last of which may correspond to the temporary loss of memory in humans. No significant data are yet available which specify the molecular changes underlying the altered behaviour or effect on memory after electroshock treatment. In this communication a search for the earliest events of the sequential changes, the utilization of glucose carbon and changes in nucleotide/nucleic acid metabolism at various stages of altered behaviour is presented.

White male Wistar rats (175g body wt.) were injected with 0.9% NaCl or 5 mg of reserpine/kg 3 h before decapitation. Then 16.6 μCi of [14C]glucose was injected intravenously before decapitation, and 1 min after injection of radioactive glucose an electric current of 22 mA for 5 s was delivered to the rats through bitemporal electrodes. All animals reacted with tonic/clonic convulsions and subsequent loss of postural reflexes. At half of the current, only the animals pretreated with reserpine showed all phases of electroshock effect. At various times after electroshock, the animals were decapitated, their brains were removed within 25 s and frozen with liquid N$_2$. The brains were homogenized in 4% (v/v) HClO$_4$ and samples of the blood were extracted with 4% HClO$_4$ for radioactivity counting. The acid-soluble fraction of the brain was fractionated by ion-exchange chromatography on Zeo-Karb and Dowex-1. The individual amino acids were further separated by paper chromatography. In experiments to assess
RNA synthesis, UTP was separated from the acid-soluble fraction by t.l.c. and the RNA extracted with hot HClO₄ or phenol (Nievel & Kirby, 1966).

Reserpine pretreatment increased the glutamate and, to a lesser extent, the glutamine and aspartic acid contents of the brain. No significant change in alanine and γ-aminobutyric acid content was observed. Under the conditions used, electroshock also increased the glutamate and aspartate content of brain; however, the amount of alanine and γ-aminobutyrate was decreased during recovery from convulsions. Reserpine pretreatment or electroshock changed the distribution of the radioactivity of the [U-14C]-glucose in the amino acids. The specific radioactivity of alanine, glutamine and γ-aminobutyrate was decreased and that of aspartate and glutamate increased after reserpine treatment. Electroshock also decreased the specific radioactivity of glutamine and γ-aminobutyrate and increased that of aspartate and glutamate; the specific radioactivity of alanine did not change.

Changes in the fate of radioactive glucose carbon in the brain amino acids after administration of the electric current may reflect the increased activity state of the biphasic reaction of brain to electroshock. Under identical conditions, however, no effect on RNA synthesis was observed in spite of the fact that some species of macromolecular RNA in brain turn over at high rate (Nievel & Kirby, 1966) and synthesis of 'pulse-labelled' RNA in brain is generally expected to change with altered behaviour. Overall changes in the functional activities of the brain may also be reflected in the distribution and staining properties (Einarson, 1932; Hyden, 1943) or the function of the Nissl substances (Nievel & Cumings, 1967), which are composed of free and protein-bound ribosomal aggregates, synthesizing protein at a high rate. Electroshock treatment with or without reserpine did not influence the protein-synthesizing capacity of these aggregates, although the amount of brain polyribosomes and their protein-synthesizing activity were found to have increased after environmental stimulation (Appel et al., 1967).

It appears therefore that the stimulation of the brain by the electric current and the subsequent changes in behaviour and memory do not affect macromolecular synthesis in the brain under the conditions used. However, a primary influence on glucose utilization and compartmentation of glucose carbon among the amino acids in the cytoplasm of the brain cells was detected. The current results, in agreement with my earlier observations (Nievel, 1975), emphasize the role of the changes in the metabolism of small molecules in the cytoplasm rather than those of macromolecules as an initial underlying biochemical mechanism after electrical stimulation or during the various phases of altered behaviour in rats.

Changes in nucleotide metabolism at the outset of induced liver growth brought about by chronic administration of coumarin and other model compounds are among the earliest biochemical events detectable in liver (Nievel, 1974).