as GOT and GPT respectively, and protein content[19] were determined on the fractionated portion. The enzyme activity was expressed as Karmen unit.

The elution diagrams of the supernatant and the soluble mitochondrial fraction from the rat liver are illustrated in Figure 1. The highest activity of the mitochondrial GOT (mGOT) and GPT (mGPT) was found in tube 29 and tube 28, whereas the supernatant GOT (sGOT) and GPT (sGPT) were recovered in tube 26 and tube 24 respectively. The results were fairly reproducible. The faster effluent portion of mGOT and mGPT may be due to the contamination of sGOT and sGPT. The effluent volume for the maximum activity of mGOT, mGPT, sGOT and sGPT was 130 ml, 126 ml, 117 ml and 108 ml respectively. Because the void volume was 74 ml, the range of the calculated molecular weight of mGOT, sGOT, mGPT and sGPT was 77–72, 115–120, 86–90 and 140–150 · 10^3 respectively. After the gel filtration, the recovery of GOT and GPT was 6–20%.

The elution diagrams of serums from the rat with carbon tetrachloride intoxication and a patient with infectious hepatitis are represented in Figure 2. The highest activity of GOT and GPT was found in tube 26 and 24 respectively, which correspond to a molecular weight of 115–120 and 140–150 · 10^3. The recovery of the activity was 32–93%.

The molecular weight of mGOT and sGOT, as calculated in the present report coincide with the molecular weight of crystallized GOT from beef liver[7]. The molecular weight of serum aminotransferase, which has not been elucidated so far, coincides fairly with sGOT and sGPT, but not with mGOT and mGPT. These results suggest that the elevated aminotransferase activity in serums with liver injury originates mainly from the supernatant fraction of the liver, as has been assumed from other evidence[10–13]. The present report, however, does not exclude the possibility of a release of mGOT and mGPT, which have a very short intravascular half-life[11].

Zusammenfassung. Mit der Gel-Infiltrationsanalyse von Serum und Leberfraktion normaler und CCl_4-exponierter Ratten wurde die Herkunft der Serum-Aminotransferase untersucht, was zur Annahme führte, dass seine Aktivitätssteigerung bei der Leberschädigung von der überstehenden Leberfraktion abhängt.

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Effects of Dehydration on Rat's Hypothalamic Acid-Phosphatase

A strong acid phosphatase activity has been demonstrated hystochemically in the hypothalamic neurosecretory cells of several mammals[1–3], including the rat[4,5]; a further increase in this activity takes place after submitting the hypothalamo-hypophyseal system to functional demands[6,7]. It was considered of interest to assess quantitatively this variation in the hypothalamus of dehydrated rats as an increase seems to take place in the pars nervosa after dehydration in the sparrow[8].

Acid phosphatase was estimated quantitatively in hypothalamic samples containing the magnocellular neurosecretory nuclei (supraoptic, paraventricular, accessory supraoptic) and median eminence of rats dehydrated by withholding water intake during 7 days. Control animals were allowed ad libitum drinking. The hypothalamus was defined by making incisions 3 mm deep just rostrally to the optic chiasm and mammillary bodies and along the lateral borders of the tuber cinereum.

Phosphatase activity was estimated by a procedure similar to the one used by Kobayashi and Farner[9]. Enzymatic activity is expressed in micrograms of para-Nitrophenol liberated per mg of wet tissue at 37 ± 0.2 °C in 45 min. At pH 5.4 phosphatase activity was a linear function of the enzyme concentration, both in the region of the supraoptic nucleus and median eminence. All determinations were controlled with blanks processed in identical fashion as the test tubes, except for the incubation period.

The results in the Table indicate that acute dehydration significantly increases acid-phosphatase activity in the hypothalamic zone containing the magnocellular neurosecretory neurons, the axons of paraventricular cells and the neurohemal structures of the median eminence, thus confirming previous histochemical findings. A similar increase in phosphomonoesterase activity has been reported

Effect of dehydration on rat's hypothalamic acid phosphatase

Condition	Activity/mg[a]	*P* Value
Normal	6.96 ± 0.14 (8)[b]	> 0.001
Dehydrated	8.00 ± 0.20 (7)	

[a] Phosphatase activity is expressed in μg of *p*-nitrophenol/mg wet tissue liberated in 45 min. [b] Mean ± S.E.; No. of animals is indicated in parentheses.

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to take place in supraoptic nucleus and median eminence in photosensitive birds exposed to prolonged daily photoperiods[9,10], and correlated to an increased gonadotropin release.

As dehydration implicates antidiuretic hormone release associated with depletion of neurosecretory material, a similar parallel can be drawn between the augmented phosphatase activity and the liberation of the octapeptide from the neurosecretory cells. This relation seems moreover strengthened by the fact that the breakdown of neurosecretory material from the pars nervosa of dehydrated mice has been seen to correspond with deformed and ruptured membranes of neurosecretory granules[8]. Acid phosphatase is apparently localized in lysosomes, a sub-cellular particle engaged, among other activities, in programmed cellular breakdown taking place in cases of apocrine and holocrine secretion[11]. Since optical[12] and ultrastructural[13-15] observations on neurosecretory systems of several species, including the rat[16,17], suggest that the release of neurosecretory material is made through a holocrine or apocrine mechanism, the increased hypothalamic acid phosphatase activity, reported in the present paper, provides a reliable biochemical basis for these morphological findings.

Resumen. Se constató un aumento significativo en el contenido de fosfatasa ácida del hipotálamo de ratas deshidratadas; estos resultados se discuten en relación a posibles mecanismos de secreción apócrina de las células neurosecretoras.

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The Role of the Pentose-Phosphate Pathway in Adjustment of the Heart to a High Load and the Development of Myocardial Hypertrophy

The compensatory hypertrophy represents an essential factor in adjustment of the heart to a prolonged increase of physiological load in deficiency, hypertension and intense physical work. The development of the compensatory hypertrophy proceeds on the base of a significant increase of the synthesis of nucleic acids and proteins in the myocardium. This ensures not only a timely replacement of worn myocardial structures but also a rapid augmentation of the mass of the myocardium[1].

Apparently the activation of the biosynthesis of nucleic acids and proteins in the myocardium in its hyperfunction is reached by way of coordinated increase of activity of a number of fermentative systems. It may be suggested that an important role in realization of these processes must belong to pentose-phosphate pathway as far as this metabolic process is the fundamental source of reduced NADP-H_2 and riboso-5-phosphate for the nucleic acids and protein synthesis.

In this connection, in the present work the change of activity of one of the most important ferments of the pentose pathway, transketolase, in the hyperfunction of the heart has been studied. Simultaneously the study of action of the antagonist of vitamin B_1 – oxythiamine, on the transketolase activity, protein synthesis and hypertrophy of the heart in its hyperfunction has been carried out.

The compensatory hyperfunction of the heart was experimentally produced in rabbits by creation of stenosis of the aorta with the earlier described method[1] ensuring a persistent narrowing of the transverse section of the aortic lumen 3 times. The activity of the transketolase was determined by the method of BRUNS[2] in control animals and in rabbits with hyperfunction of the heart 2 and 45 days following creation of stenosis.

As seen from Table I, 2 days following the onset of hyperfunction when the processes of biosynthesis of nuclear acids and proteins[3,4] in the myocardium are sharply intensified, the activity of transketolase in this organ is increased by more than 60%. Following 45 days when the process of hypertrophy is essentially completed, the transketolase activity decreases, approaching the normal level.

Apparently, these changes are specifically connected with the compensatory hypertrophy of the heart. They take place only in the myocardium and are lacking in other tissues, for instance in blood.

The close correlation between the activity of transketolase and the intensity of development of the myocardial hypertrophy is revealed in the analysis of data relating to different animals at the initial stage of hyperfunction and hypertrophy of the heart. From the Figure it is seen that the higher the activity of transketolase in the myocardium of the animal, the more increased the relative weight of the left ventricle, i.e. the more intense the development of the process of hypertrophy.

This suggests that activation of the transketolase is apparently one of the links of the biochemical mechanism of hypertrophy of the myocardium in its compensatory hyperfunction.

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