pheochromocytomas. This question cannot be answered fully; however, one factor might be the increased rate of oxidation of epinephrine commonly found in schizophrenia(8).

Summary. Increased 24 hr excretion of urinary epinephrines was found in 14 of 17 patients with various psychiatric disorders. These increased outputs varied from about 200 to 500 μg/day; the largest was 1184. Such values are within the range of those found cases of pheochromocytoma and emphasize the fact that increased epinephrine excretion is not pathognomonic of adrenal medullary and chromaffin chain tumors.

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Effects of Aminopterin and Estrogen on Phosphoprotein Phosphatase of Rat Uterus.* (23182)

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In a previous investigation of the effects of estrogen and 4-aminopteroylglutamic acid (Aminopterin) on phosphate metabolism of rat uterus(1), it was observed that Aminopterin caused an almost complete inhibition of synthesis of phosphoprotein in response to estrogen. Aminopterin also inhibited uterine growth in response to estrogen which suggested that synthesis of phosphoprotein, might be a significant factor in growth of this organ. Therefore, it was felt that a study of the enzyme concerned in metabolism of uterine phosphoprotein would be of interest.

Methods. 45 female rats of the Holtzman strain, weighing 45-55 g, were used. The animals were 22 days old when spayed and were placed on experiment 1-2 days after spaying. They were grouped according to treatment as follows: Group AE contained 22 animals, receiving 10 μg Aminopterin/day for 4 days intraperitoneally and 0.1 μg 17-β-estradiol/day for 3 days subcutaneously. Estrogen was given simultaneously with the last 3 Aminopterin injections. Group E contained 6 animals which received estrogen injections only and Group C contained 17 animals serving as untreated controls. At necropsy uteri were removed, stripped free of mesometrium, weighed, pooled as described in Table I, and 10% water homogenates prepared. Assay procedure for phosphoprotein phosphatase activity was that described by Norberg(2) except that whole homogenates rather than extracts were used. The substrate was commercial casein dissolved in dilute NaOH solution and precipitated with HCl 3 times, then stored in frozen state. This procedure was necessary to prevent precipitation of proteoses after addition of molybdate in phosphate determinations. Inorganic phosphorus was determined according to Lepage(3).

Results. Activity of phosphoprotein phosphatase in the different groups (Table I) cannot be correlated with observations on phosphoprotein fraction of uterus studied under identical experimental conditions, as described previously(1). Values for this

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TABLE I. Phosphoprotein Phosphatase Activity of Rat Uterus.*

Group	Body wt change, g†	Uterine wt, mg‡	No. determinations§	Units, μM P/min./g ‡‖	Total $\times 10^{-3}$ ¶
C	+16	25.6 ± 1.061	3	1.636 ± .075	41.85
AE	−11	29.8 ± 2.162 (>.05)	4	2.297 ± .008 (<.01)	68.45
E	+15	86.7 ± 2.173 (<.01)	3	1.961 ± .151 (>.05)	170.0

* Mean values and stand. error.
† Gain or loss in body wt during 4-day injection period.
‡ Significant difference between means of exp. and control groups indicated by P = .05 or less, shown in parentheses.
§ 2-7 uteri pooled for each determination.
‖ μmoles phosphorus released/min./g fresh tissue.
¶ Units × avg uterine wt × 10^{-3}.

fraction (taken from previous publication) have been included in Fig. 1 to facilitate comparison with enzyme activity. Total phosphoprotein/uterus is used as index of phosphoprotein synthesis and incorporation of radioactive phosphorus by this fraction is included as a relative index of turnover. P^{32} uptake was calculated as counts/min/μg P divided by injected dose.

In response to estrogen alone (Group E) phosphatase activity g fresh tissue did not increase significantly over the control value but total activity/uterus increased as a consequence of 240% increase in uterine weight. Synthesis of phosphoprotein, however, increased 810% while P^{32} incorporation decreased 55%. In Group AE, where Aminopterin completely inhibited uterine growth in response to estrogen, phosphatase activity increased significantly, but synthesis of phosphoprotein was not significantly greater than in controls, and turnover increased 300%. It appears, then, that phosphoprotein synthesis and turnover are not directly related to phosphatase activity and phosphoprotein turnover is inversely related to synthesis of phosphoprotein and uterine growth.

Discussion. A similar discrepancy between activity of phosphoprotein phosphatase and synthesis of phosphoprotein in a growing organ was observed by Norberg(4), who studied regeneration of rat liver after partial hepatectomy. Total enzyme/liver increased at the same rate as liver mass but enzyme concentration did not change appreciably, as observed for uterus in Group E. Total phosphoprotein likewise increased at the same rate as liver mass except on the 3rd and 10th days of regeneration when phosphoprotein increase was 50% greater than the increase in liver mass. In this respect the 3rd and 10th days of regeneration are similar to the finding for Group E uteri. Also, Johnson and Albert (5), who studied phosphoproteins of various rat organs, noted an inverse relation between phosphoprotein content and turnover rate, and a lack of correlation between enzyme activity and turnover.

At present only a theoretical explanation can be offered for the results presented here. It is possible, if not probable, that phosphoprotein phosphatase functions only as a

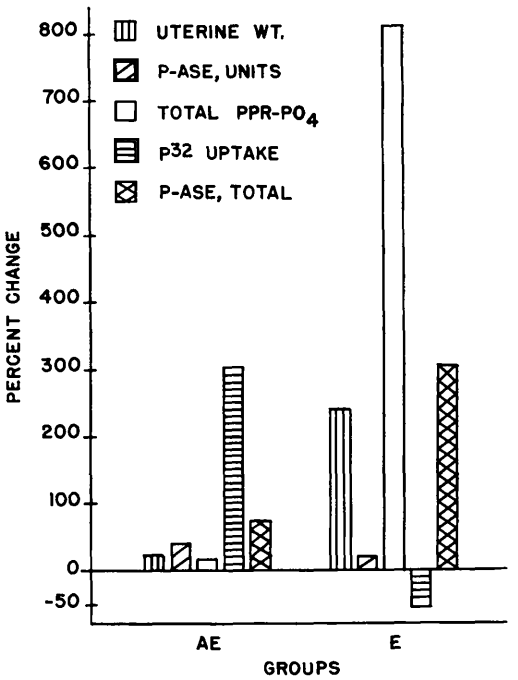

FIG. 1. Experimental results calculated as % of control value. Values for phosphoprotein fraction taken from Davis, Meyer, and McShan(1).

catabolic enzyme that is controlled by a specific protein inhibitor, similar to the inhibitor described for deoxyribonuclease(6). Such an inhibitor would not necessarily be apparent in the assay procedure for enzyme activity. In Group AE Aminopterin apparently interferes with synthesis of inhibitor, which is consistent with ability of Aminopterin to inhibit incorporaton of glycine and serine carbon into proteins(1). Estrogen in Group AE stimulates synthesis of phosphoprotein but hydrolysis proceeds at an equal rate, thus preventing a net increase in phosphoprotein and accounting for the very high turnover of phosphorus. In Group E production of inhibitor would be unimpeded, preventing the high turnover rate and resulting in increased accumulation of phosphoprotein.

Harris(7) observed that some controlling factor must exist for phosphoprotein phosphatase of frog eggs but he believed that pH of the yolk was that factor. Johnson and Albert(5) suggested that a specific inhibitor might exist in mammalian tissues, but the hypothesis obviously requires direct experimental support, particularly in view of Feinstein's unsuccessful attempt to demonstrate such an inhibitor in rat intestine(8).

Summary. Total phosphoprotein phosphatase activity of rat uterus increases in response to estrogen as uterine weight increases, but activity/g fresh tissue does not change appreciably. Aminopterin administered simultaneously with estrogen inhibits the increase in total activity/uterus as it inhibits uterine weight increase. The changes in enzyme activity could not be correlated with changes in uterine phosphoprotein and the possible existence of a phosphatase inhibitor was discussed.

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Influence of Water-Treatment on Nutritional Value of Barley.* (23183)

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Fry *et al.*(1) showed that feeding value of barley for chicks was not improved greatly by removal of the fibrous hull through pearling. Results on metabolizable energy content analysis (with chicks) of barley and pearled barley obtained on material sent from our laboratory showed that pearled barley contained *less* metabolizable energy than regular barley.† These findings suggested that the carbohydrate in barley, aside from crude fiber, is much less available than similar carbohydrates in corn. Studies were undertaken to determine whether the nutritional value of barley could be improved by different treatments. The results obtained in 2 of these experiments are here presented.

Procedure. As the basis of a working hypothesis, it was assumed that the lower nutritional value of barley could be due to a number of reasons such as: (1) Presence of an inhibiting substance or substances; (2) existence of structural linkages in the carbohydrates of barley that are not attacked by enzymes endogenous to the chick; or (3) outright deficiency in carbohydrate content of

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