

tendencies; in fact, preliminary results obtained from 5 Ss and corrected by the Miller-Madow formula did not differ significantly from the actual data. This may be explained by the fact that most of the error responses appear to be random.

From Figure 1 the transmitted information appears to be a direct linear function of the logarithm of brightness. The relation outlined by FAUVILLE may thus be extended to this type of noise. Moreover, this function seems rather independent of the number of possible locations. The single exception is the material with 3 locations, at least when they are horizontally oriented, which provides less information transmission; further inspection of Figure 1 shows that the main difference is to be found in the slope. Using the least squares procedure, a straight line has been traced through the results of each Figure; then from the obtained origin on the abscissa, 2 other lines were drawn, 1 through the points relative to 5, 7 and 9 locations, the other through the points provided by the 3-locations material.

From these data Figure 2 has been constructed, where transmitted information is plotted against stimulus information, as for usual channel capacity functions. The continuous lines join the calculated values. Like in easy viewing conditions, transmitted information reaches a plateau (it is not always very flat, unfortunately, probably because of some anchoring effects). A primary effect of noise is to lower it. In addition, when stimulus information is especially low, the transmission loss may be less important in absolute value, but remains proportionally constant. It may incidentally be noted that the dots are better localized on the horizontal than on the vertical axis.

**Discussion.** The inverse logarithmic relation between noise and information transmission is confirmed to be independent of the physical nature of the noise, at least for visual localization tasks. But this seems also to be true when other transmission channels are involved; similar relations were found between stimulus duration and hue

identification<sup>5</sup>, or between signal-to-noise ratio and speech intelligibility<sup>6</sup>.

The independence of the relative loss towards stimulus information suggests that the disturbing effects of brightness reduction do not act upon the central categorization mechanisms, but upon the preliminary sensory coding operations. It may be interesting to recall here the results of an experiment by LEIBOWITZ et al.<sup>7</sup> on judging the inclination of a line. S was not forced to provide a response and thus had the faculty of disregarding presentations where viewing had not been satisfactory. Frequency of seeing increased roughly as the logarithm of brightness and of duration; but, provided the stimulus was seen, localization accuracy was not influenced by these factors<sup>8</sup>.

**Résumé.** Dans une tâche consistant à localiser un point sur une barre horizontale ou verticale, on mesure l'information transmise en fonction de l'éclairement et de l'information du stimulus. On trouve une relation linéaire directe entre l'information transmise et le logarithme de l'éclairement. Cette relation reste valable pour diverses quantités d'information émise.

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<sup>5</sup> H. H. SOMERS, *Les attributs de la perception et leur interaction* (Doct. Dissert., Univ. of Louvain 1961).

<sup>6</sup> J. COSTERMANS et G. DE VALCK, *J. Psychol. norm. path.* 421 (1965).

<sup>7</sup> H. W. LEIBOWITZ, N. A. MEYERS and D. A. GRANT, *J. opt. Soc. Am.* 45, 76 (1955).

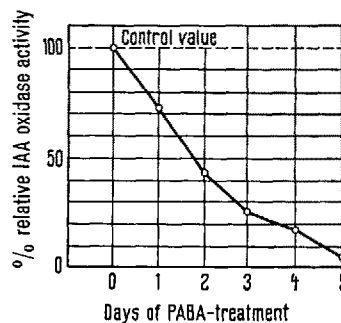
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## Influence of *p*-Aminobenzoic Acid on the Activity of Indoleacetic Acid Oxidase

It is known that some benzoic acid isomers and derivatives can influence the activity of indoleacetic acid (IAA) oxidase<sup>1</sup>, however, the effect of a biologically important compound, namely *p*-aminobenzoic acid (PABA), had never been investigated in this respect.

When PABA was added to a reaction mixture according to STUTZ<sup>2</sup> containing a crude enzyme extract prepared from hypocotyls of white lupine (*Lupinus albus* L.) cuttings, the manometrically measured O<sub>2</sub>-consumption did not differ from that of the controls, even if the concentration of PABA was as high as 200 µg/ml. By contrast, when the enzyme solution was prepared from cuttings grown in 50 ppm PABA, the IAA oxidase activities depending on the duration of PABA-treatment became continuously lower, falling almost to zero on the fifth day (Figure).

Adding of boiled extracts from PABA-cuttings to IAA oxidase preparations from water-controls resulted in a lag-phase and a same inhibition, respectively, as the PABA-treatment itself. The activities of dialyzed extracts from both PABA- and water-treated plants did not exhibit any



Time course of inhibition of IAA oxidase activity in lupine cuttings after PABA administration in vivo, compared to the activities of water-treated controls.

<sup>1</sup> TH. GASPAR, *Année biol.* 4, 437 (1965).

<sup>2</sup> R. E. STUTZ, *Pl. Physiol.*, Lancaster 32, 31 (1957).

significant differences. Thus, it seems to be valid that the in vivo developing effect of PABA on enzymatic auxin degradation could be due to the formation and accumulation of one or more inhibitory metabolic products rather than to the intact PABA molecules. This is supported by the observation that anthranilic acid (*o*-aminobenzoic acid) or novocain (diethylaminoethyl ester of PABA) are unable to inhibit the enzymatic IAA oxidation. Their metabolic products separated by paper chromatography differed completely from those of PABA. Sulfathiazole does not inhibit the PABA-effect described above on the IAA oxidase activity.

It is clear from these results that care must be taken in generalization of effects produced by phenolic IAA oxidase modifiers in vitro.

Our present findings give an additional explanation for the stimulating effect of PABA on the adventitious root formation reported by MANGENOT and CARPENTIER<sup>3</sup> in lupine, as well as by POAPST and DURKEE<sup>4</sup> in bean plants through increasing the endogenous IAA-level indirectly by inhibition of enzymatic IAA destruction.

**Zusammenfassung.** Die enzymatische Oxydation der Indolyllessigsäure bleibt durch *p*-Aminobenzoesäure (PABS) in vitro unbeeinflusst, während sie in vivo stark gehemmt wird. Dieser Effekt ist wahrscheinlich auf solche Inhibitoren zurückzuführen, die aus PABS in der Pflanze metabolisch entstanden sind.

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<sup>3</sup> G. MANGENOT and S. CARPENTIER, C. r. Séanc. Soc. Biol. 135, 1053 (1941).

<sup>4</sup> P. A. POAPST and A. B. DURKEE, J. hort. Sci. 42, 429 (1967).

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## Activities of Catecholamines on the Rat Muscle Glycogenolytic ( $\beta$ -2) Receptor

Since earlier discussions<sup>1-3</sup> showed that  $\beta$ -adrenergic responses to catecholamines are better classified into 2 groups than into 1 when viewed on the basis of relative responses to agonists, we were prompted to determine how well the rat muscle glycogenolytic receptor conformed to either of these group classifications. The blood lactic acid increase response to epinephrine, noted initially by CORI<sup>4</sup>, was utilized as an index of muscle glycogenolysis on the basis of the findings of HORN BROOK and BRODY<sup>5</sup> and of KENNEDY and ELLIS<sup>6</sup>.

**Methods.** These studies were conducted with young adult rats of either sex permitted food and water ad libitum. Blood was taken 1 h after the catecholamines in 0.05% ascorbic acid-stabilized solution had been given either s.c. or i.p. The route selected was the one that best yielded graded dose related responses to the test amine. For the pressor amines, epinephrine, norepinephrine, nordefrin, *N*-methyl- $\alpha$ -methylnorepinephrine and *N*-ethylnorepinephrine, this was i.p. For the vasodepressor amines it was s.c. The rats were sedated with 55 mg sodium pentobarbital/kg about 20 min before blood was taken. Blood lactic acid was determined according to the procedure of FRIEDLAND and DIETRICH<sup>7</sup> which depends on the enzymatic oxidation of lactate to pyruvate, the hydrogen liberated reacting with *p*-iodonitrotetrazolium chloride (INT) to yield a colored formazon. The latter is read spectrophotometrically at 490 nm.

**Results.** The graded dose related increases to 9 catecholamines, seen as increases in blood lactic acid, are summarized in Table I. Their activities, calculated by means of the log dose:response formula<sup>8</sup>  $M = x_s - x_u - (y_s - y_u)/b_s$ , relative to *l*-isoproterenol as 1000, are included. Since the control blood lactic acid values were relatively consistent, the comparisons were made directly on the values as given in Table I.

It may be seen that the relative effects of isoproterenol, epinephrine and norepinephrine are in accord with those of numerous investigators (see ELLIS<sup>9</sup>). The relative activi-

ties of *N*-ethylnorepinephrine and *N*-*t*-butylnorepinephrine are in the same range as that noted earlier by PRATESI et al.<sup>10</sup>. The effects noted with norepinephrine and nordefrin were best determined after  $\alpha$ -receptor blockade to reduce the toxicity of these vasopressor amines<sup>11</sup>.

The responses, as blood lactic acid, to the 9 catecholamines of Table I may be compared with the  $\beta$ -1 and  $\beta$ -2 responses to each of them noted earlier<sup>2,3,12,13</sup>. Correlation coefficients on the basis of log:log comparisons are summarized in Table II. The data used for the remaining receptor responses are too extensive to be reproduced here

<sup>1</sup> A. ARNOLD, J. P. McAULIFF, F. P. LUDUENA, T. G. BROWN JR. and A. M. LANDS, Fedn Proc. Fedn Am. Socs exp. Biol. 25, 500 (1966).

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<sup>4</sup> C. F. CORI, J. biol. Chem. 63, 253 (1925).

<sup>5</sup> K. R. HORN BROOK and T. M. BRODY, Biochem. Pharmac. 12, 1407 (1963).

<sup>6</sup> B. L. KENNEDY and S. ELLIS, Fedn Proc. Fedn Am. Socs exp. Biol. 22, 449 (1963).

<sup>7</sup> I. M. FRIEDLAND and L. S. DIETRICH, Analyt. Chem. 2, 390 (1961).

<sup>8</sup> C. I. BLISS, *Vitamin Methods* (Ed. P. GYÖRGY; Academic Press, New York 1951), vol. 2, p. 448.

<sup>9</sup> S. ELLIS, *Physiological Pharmacology* (Ed. W. S. ROOT and F. G. HOFMANN; Academic Press, New York 1967), vol. 4, p. 179.

<sup>10</sup> P. PRATESI, E. GRANA, L. LILLA, A. LA MANNA and L. VILLA, Farmaco, Ed. sci. 18, 920 (1963).

<sup>11</sup> F. P. LUDUENA and M. J. BRANIN, J. Am. pharm. Ass. 56, 951 (1967).

<sup>12</sup> A. ARNOLD and J. P. McAULIFF, Biochem. Pharmac. 17, 755 (1968).

<sup>13</sup> A. ARNOLD and J. P. McAULIFF, Experientia 24, 436 (1968).