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 modificators 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[3] in lupine, as well as by POAPST and DURKEE[4] in bean plants through increasing the endogenous IAA-level indirectly by inhibition of enzymatic IAA destruction.

Zusammenfassung. Die enzymatische Oxydation der Indolylessigsäure 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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Activities of Catecholamines on the Rat Muscle Glycogenolytic (β-2) Receptor

Since earlier discussions[1-3] showed that β-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[4], was utilized as an index of muscle glycogenolysis on the basis of the findings of HORNBROOK and BRODY[5] and of KENNEDY and ELLIS[6].

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-α-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[7] 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[8] $M = x_s - x_u - (y_s - y_u)/b_c$, 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[9]). The relative activities of *N*-ethylnorepinephrine and *N*-*t*-butylnorepinephrine are in the same range as that noted earlier by PRATESI et al.[10]. The effects noted with norepinephrine and nordefrin were best determined after α-receptor blockade to reduce the toxicity of these vasopressor amines[11].

The responses, as blood lactic acid, to the 9 catecholamines of Table I may be compared with the β-1 and β-2 responses to each of them noted earlier[2,3,12,13]. 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

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Table I. Rat muscle glycogenolytic response to catecholamines

Dose[a] (μg/kg)	Route	No. of rats	Blood lactic acid (mg/100 ml)[b]	Activity relative to isoproterenol = 1000
l-Isoproterenol × bitartrate × $2H_2O$				
None	s.c.	5	13.2 ± 2.04	
40		5	10.0 ± 1.02	
60		3	25.6 ± 1.89	
90		4	41.6 ± 3.82	
None	s.c.	4	15.0 ± 0.27	
60		4	24.6 ± 3.59	
104		5	47.8 ± 7.31	1000
l-Epinephrine × bitartrate				
None	i.p.	6	13.8 ± 2.20	
130		6	19.6 ± 2.58	
225		6	26.6 ± 1.90	
None	i.p.	6	13.8 ± 1.96	
150		6	20.7 ± 2.46	
225		6	27.6 ± 1.09	
337		5	37.2 ± 3.39	293
l-Norepinephrine[c] × bitartrate × H_2O				
None	i.p.	5	7.4 ± 0.52	
25×10^3		5	17.4 ± 4.64	ca. 2
l-Nordefrin[c] × mucate				
None	i.p.	5	7.4 ± 0.52	
5×10^3		5	9.2 ± 1.19	
11.2×10^3		5	25.7 ± 3.16	
25×10^3		4	36.3 ± 3.78	5
l-*N*-Ethylnorepinephrine × HCl				
None	i.p.	5	10.4 ± 0.33	
130	i.p.	5	20.2 ± 2.53	
225	i.p.	5	34.2 ± 2.52	364
dl-*N*-*t*-Butylnorepinephrine × methane sulfonate				
None	s.c.	4	5.1 ± 1.01	
120	s.c.	5	20.7 ± 5.69	
180	s.c.	4	27.0 ± 4.17	
270	s.c.	5	34.3 ± 3.54	
None	s.c.	6	7.8 ± 0.74	
120	s.c.	6	13.7 ± 1.61	
180	s.c.	6	21.9 ± 1.94	
270	s.c.	6	30.7 ± 1.91	305
dl-*N*-Methyl-α-methylnorepinephrine × HCl				
None	i.p.	7	13.57 ± 1.21	
576	i.p.	7	17.86 ± 2.57	
1000	i.p.	7	29.66 ± 4.07	
dl-Isoetharine × HCl				
None	s.c.	5	8.1 ± 2.48	74
250	s.c.	5	25.2 ± 2.25	
433	s.c.	5	31.9 ± 3.47	
750	s.c.	5	35.9 ± 3.50	
None	s.c.	5	10.0 ± 2.02	
500	s.c.	5	28.6 ± 4.77	
750	s.c.	5	41.4 ± 2.76	151
dl-*N*-cyclopentyl-α-ethylnorepinephrine				
None	s.c.	4	8.4 ± 1.06	
173		5	16.9 ± 2.52	
300		5	27.5 ± 4.71	235

[a] As base. [b] Mean ± S.E. [c] After 3.16 mg phenoxybenzamine/kg pre-treatment.

Table II. Correlation of rat muscle glycogenolysis with published β-1 and β-2 adrenergic receptor responses to catecholamines

	Rat muscle glycogenolysis *vs.*	n	Correlation coefficient *r*
β-1	Rat adipose tissue lipolysis[a,b]	9	0.061
	Guinea-pig adipose tissue lipolysis[c]	6	< 0.001
	Rabbit jejunum inhibition[d]	7	0.108
	Rabbit heart force[a,d]	8	0.215
	Rabbit heart rate[d]	8	0.227
β-2	Rat diaphragm contraction[d]	7	0.960
	Rat uterus inhibition[d]	8	0.982
	Guinea-pig bronchodilatation[a]	9	0.971
	Dog vasodepression[a]	9	0.931

[a] Data from LANDS et al.[2]. [b] Data from ARNOLD and MCAULIFF[12]. [c] Data from ARNOLD and MCAULIFF[13]. [d] Data from LANDS et al.[3].

and may be obtained by reference to the original publications.

On the basis of the correlation coefficients of Table II it is seen that the rat muscle glycogenolytic receptor in no instance corresponds to any of the 5 β-1 receptor mediated responses. Contrariwise, it uniformly corresponds well with each of the 4 previously defined β-2 receptor mediated responses with correlation coefficients between 0.931 and 0.982.

As a separate point of interest, it may be germane to point out that the metabolic responses to the catecholamines clearly do not group within a single receptor concept. Thus, rat and guinea-pig lipolysis is β-1[2,12,13], rat muscle glycogenolysis is β-2 (present data) and rat liver glycogenolysis is α[14–18].

Thus, on the basis of studies with 9 catecholamines, rat muscle glycogenolysis may be seen to correspond closely to the responses given by dog vasodepression, guinea-pig bronchodilation, rat uterine inhibition and rat diaphragm contraction. It was suggested earlier that the receptor mediating the latter responses might be termed β-2 to distinguish it from the β-1 receptor for rat and guinea-pig lipolytic, rabbit heart force and rate, and rabbit jejunum inhibition mediated responses[19].

Zusammenfassung. Es wird gezeigt, dass der Glykogen-Rezeptor im Rattenmuskel vom Standpunkt der Reaktion mit den Katecholaminen als β-2 anzusehen ist.

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