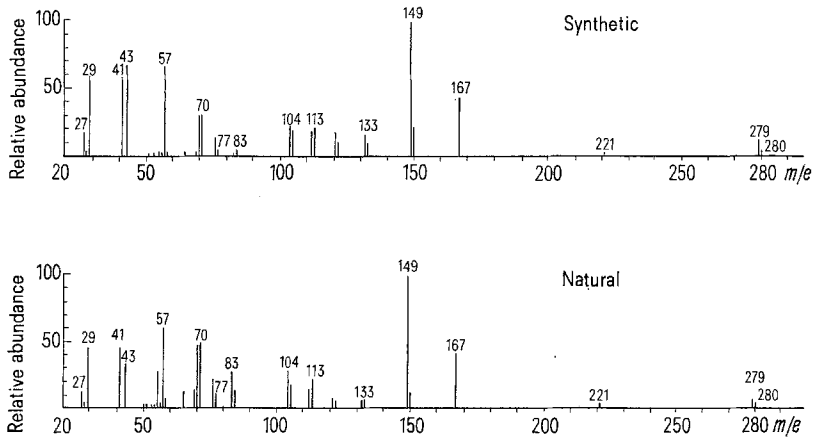

Fig. 2. Mass spectrum of Di-N-TFA-β-phenyl-β-alanine ethyl ester obtained with 70volt electrons.

Edeine D gives positive ninhydrin and sodium nitropruside reactions and negative Sakaguchi and Pauly reactions, indicating the presence of free primary and secondary amino groups and the lack of guanyl and phenol groups.

The composition of the antibiotic was determined after hydrolysis in 6 *N* HCl at 100 °C for 27 h. The hydrolysate was analyzed by paper and thin layer chromatography and high voltage electrophoresis. The following compounds were identified: glycine, 2-hydroxy-3-aminopropionic acid, 2,6-diamino-7-hydroxyazelaic acid and its dehydratation product 2,6-diamino-Δ^7-azelaic acid, 2,3-diaminopropionic acid, spermidine and an amino acid which could not be identified with any of the standards. All products, except the last one, were also found in the hydrolysate of edeine A[8] which also additionally contained β-tyrosine not found in edeine D. Instead of β-tyrosine edeine D contained the above-mentioned new amino acid residue.

For structural elucidation the non-identified amino acid was preparatively isolated from the hydrolysate by column chromatography on silicagel in the solvent system: n-propanol: water = 19:1. Its $[\alpha]_D^{23} = -10{,}3°$ (in water). Ethyl ester of N,N-di-trifluoracetyl derivative was prepared and analyzed by mass spectrometry on LKB instrument (Model 9000) with E-301 column. The mass spectrum was identical with that of the authentic sample ethyl ester of N,N-di-trifluoracetyl-β-phenyl-β-alanine obtained synthetically[9]. Both mass spectra are shown on Figure 2.

In both spectra parental ions (P) did not appear and the fragment ions of highest molecular weight were: *m/e* 280 = P-(C_6H_5 + C_2H_4) and *m/e* 279 = P-(C_6H_5 + C_2H_5). The base peak was *m/e* 149 = P-($C_4F_6NO_2$ + C_2H_4).

N,N-di-TFA-methyl esters of both natural and synthetic amino acids exhibited also identical retention time in gas chromatography in 2 columns (both 2 m): OV-17 (temp. 210°, retention time 9.9 min) and SE-30 (210°, ret. time 13.1 min).

Natural and synthetic amino acids in free form and as their ethyl esters were also identical in paper and thin layer chromatography and in high voltage electrophoresis.

The results obtained indicate that edeine D is a close analogue of edeine A in which the residue of β-tyrosine was replaced by β-phenyl β-alanine. The rotation data of the latter one indicate the L-configuration.

β-phenyl-β-alanine is an amino acid very rarely found in natural products. It was previously isolated from islandotoxin[10]. It exhibits an interesting property of inducing morphological changes in *Bacillus brevis* producing gramicidine[11].

Zusammenfassung. Edein D wurde aus dem komplexen Antibioticum Edein mit Hilfe von Verteilungs-Chromatographie abgetrennt. Als Bausteine von Edein D wurden 3-Amino-3-phenyl-propionsäure, 3-Amino-2-hydroxypropionsäure, 2,3-Diaminpropionsäure, 2,6-Diamin-7-hydroxy-azelainsäure, Glycin und Spermidin ermittelt.

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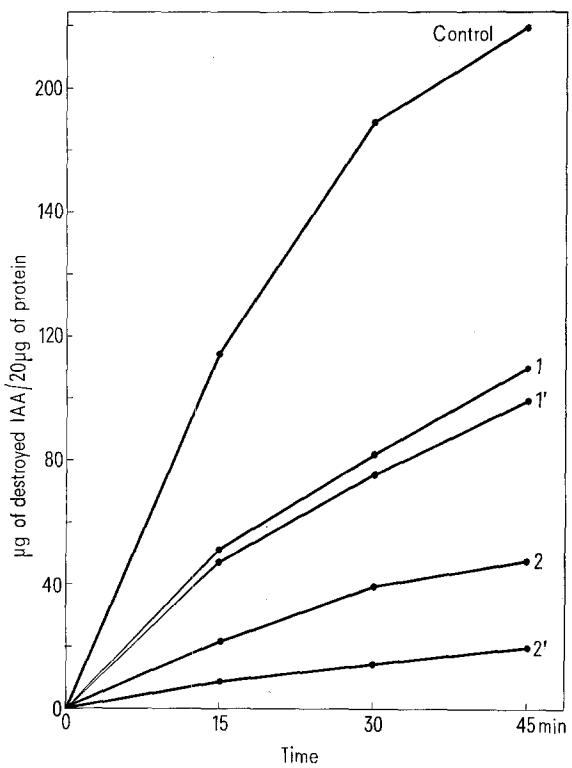
The Effect of *p*-Benzoquinone and Quinol on the IAA-Oxidase Activity

Phenolic compounds form a special group of the growth regulators – a group of natural growth inhibitors[1]. They effect the growth processes most frequently through the IAA-oxidase system[2]. This enzyme controls the endogenic concentration of IAA[3,4].

The free phenolic substances in the plant cells succumb to transformation reactions (glycosylation, esterification and methylation) or they are substrates of peroxidases and phenoloxidases. In the phenol-phenoloxidase system, quinones are products of the reaction. Quinones are compounds of wide occurence in nature, which importance in biochemistry is becoming constant more recognized[5]. Quinones effect a number of enzyme reactions owing to their oxido-reduction potential[6,7]. The electronic structures of quinones are in relation to their biological activity[8].

The effect of *p*-benzoquinone (*p*-BQ) and quinol (Q) on the IAA-oxidase activity

Concentration (*M*)		µg IAA destroyed/45 min	Inhibition (%)
5×10^{-6}	*p*-BQ	112	50
	Q	101	55
10^{-5}	*p*-BQ	98	56
	Q	92	59
5×10^{-5}	*p*-BQ	49	78
	Q	20	91
10^{-4}	*p*-BQ	21	90
	Q	17	93
5×10^{-4}	*p*-BQ	0	100
	Q	0	100
Control		220	—



Activity of IAA-oxidase in relation to the duration (in min) of incubation in the presence of quinol ($1' = 5\times10^{-6}M$; $2' = 5\times10^{-5}M$) or *p*-benzoquinone ($1 = 5\times10^{-6}M$; $2 = 5\times10^{-5}M$); K, control.

Stom[9] observed *p*- and *o*-benzoquinone influence on the growth of the maize coleoptile segments. It was found that the segments grow more rapidly in the presence of quinol and pyrocatechol than in the presence of their quinone derivates. He considers quinones to be more effective growth regulators than the corresponding phenolic compounds.

Methods. The enzyme was isolated from the roots of 3-day-old maize seedlings, which were cultivated at 25 °C in the dark. After homogenization in the $^1/_{15}M$ phosphate buffer, pH 5.8 (0.8/1 ml) and centrifugation (10,000 × *g*, 0 °C) the solution was dialyzed against this buffer for 48 h. The dialysate was purified on a column of SE-Sephadex C-50 (1 × 20 cm). The proteins were eluted with stepwise gradient of NaCl in phosphate buffer (from 0.1 to 0.5 *M*). The fractions with highest specific activity (eluted with 0.1 *M* NaCl) were pooled and used as the enzyme solution. The activity of enzyme was determined according to Dinant et al.[10] using the equation according to Pilet et al.[11] for calculation of the amount of destroyed IAA.

Results and discussion. The effect of phenolic compounds on the activity of IAA-oxidase depends on their concentration and structure[2,12]. The endogenous level of phenolic substrates in plants is linked to the amount of chlorophyll present[13]. The in vitro degradation of IAA was inhibited by catechol and quinol and activated by resorcinol[14,15]. The latter at low concentration promoted the growth of root sections as well as of intact roots[14]. The equilibrium phenol-quinone may be affected by activities of phenolases and quinone reductase. The function of quinones has not been very often discussed[16,17]. Studying the effect of *p*-benzoquinone and quinol on the in vitro degradation of IAA, it was found (Table) that quinol is a somewhat more effective IAA-oxidase inhibitor than *p*-benzoquinone. The largest difference has been observed at $5\times10^{-5}M$ concentration. The results obtained show that quinones can be involved in the regulation of IAA-oxidase activity.

Zusammenfassung. Bei Prüfung des Einflusses von *p*-Benzochinon auf die Aktivität der partiell gereinigten IES-Oxidase aus Wurzeln von Maiskeimlingen wurde bei Konzentrationen von 5×10^{-6} bis $5\times10^{-4}M$ eine 50- bzw. 100%ige Hemmung beobachtet.

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