transient decrease of the membrane potential whose characteristics are completely different from the action potential. The amplitude of this fall in membrane potential ranged from 1–10 mV in different fibres. At the neuromuscular junction its rising phase is about 1.2 msec and, after the summit, the potential falls by one half in about 3.7 msec. They are typical end-plate potentials similar to those described in the neuromuscular system of the frog[2]. Their amplitude falls and the potential changes are slowed down at points distant from the neuromuscular junction (Figure 2). During the process of curarization, it was possible to see that the step located at the foot of action potentials, recorded at the neuromuscular junctions, corresponds to the first part of the end-plate potential (end-plate component).

Summation of 2 end-plate potentials may give rise to propagated spike and twitches. The end-plate potential elicited by the second nerve volley was usually of larger amplitude than the first end-plate potential but of similar time course.

Blockade of neuromuscular transmission was also found when a saline solution containing a high magnesium concentration (12–15 m*M*) was used. Figure 2b shows end-plate potentials recorded from different muscle fibres after 5 min of equilibration in high magnesium solution. Similar results were described with magnesium in frog muscle[3]. The neuromuscular blockade observed with high magnesium solution or D-tubocurarine was completely reversible and no change on resting potential was found after the addition of these 2 blocking agents in a concentration strong enough to abolish the neuromuscular transmission. The average resting potential recorded in 20 muscle fibres in D-tubocurarine ($5 \cdot 10^{-7}$ w/v) was 94.7 mV (S.D. ± 1.7), and in high Mg^{++} (15 m*M*) 91.5 mV (S.D. ± 2.2). The average resting potential recorded in 60 muscle fibres in normal Ringer solution was 93.4 mV (S.D. ± 1.5). Calcium withdrawal also caused neuromuscular blockade, which suggests that in the neuromuscular system of *Electrophorus*, the cation also plays a facilitatory action on the release of the neuro-hormone by nerve impulse.

Although more detailed information is required about the neuromuscular system in *Electrophorus*, the results presented above seem to indicate that the mechanism of neuromuscular transmission in this fish is cholinergic and that the properties of the motor end-plate are, as a whole, very similar to those described in frog muscle.

The results also lead to the conclusion that the mechanism of neuromuscular and neuro-electroplate transmission in *Electrophorus* are similar. If the electric organ in *Electrophorus* originates from muscle as in other electric fishes, the process of differentiation does not include the mechanism of junctional transmission.

Résumé. Le système neuromusculaire dans *Electrophorus electricus* est étudié avec des électrodes intracellulaires. La transmission neuromusculaire est supprimée avec D-tubocurarine ($5 \cdot 10^{-7}$ *p*/v) ou avec un excès des ions Mg^{++} (12 m*M*) dans la solution.

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The Effect of Gibberellic Acid Treatment on the Alkaloid Content of Mature *Ipomoea violacea* L. Seeds

Several studies have shown that the alkaloid content of medicinal plants can be influenced markedly by treatment with gibberellic acid (GA). While in some cases such treatment has brought about an increase, it has, in others, led to a reduction of alkaloid concentration in various plant organs[1–6]. GA effects observed vary generally with the plant organ investigated and are, furthermore, dependent on such factors as the age of the plant and mode and concentration of the GA application. Studies of these effects on psychotomimetic constituents are particularly important since misuse of seeds of *Ipomoea violacea* has been reported[7–9]. The present study was initiated to investigate whether conditions for GA treatment could be found which would reduce the alkaloid content of the seeds. 11 plants of *I. violacea* L. (Heavenly Blue) were grown under greenhouse conditions (60–70 °F). The seeds were sown on March 10, 1965. The small plants were transplanted into pots and after they reached a height of 15 inches (April 13, 1965) 4 groups of 2 plants each were treated weekly with the following GA sprays[10]: 100 ppm for 2 weeks (GA [1]); 500 ppm for 2 weeks (GA [2]); 100 ppm for 9 weeks (GA [3]); 500 ppm for 9 weeks (GA [4]). 3 plants served as controls. After reaching maturity the seeds from each individual plant were collected and analysed for total alkaloids, lysergic acid amide, *iso*lysergic acid amide and clavine alkaloids by methods reported previously[11]. Morphological observations were also recorded and will be reported elsewhere. Flower production started during the first week of June, lasted for 5 weeks and reached a peak 12 weeks after the first GA treatment. The seeds were mature about 60 days after fertilization. 11 weeks following the first peak a second smaller peak in flower production was produced by all plants. The seeds from both peaks were collected separately and are referred to as first and second crop, respectively.

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Total alkaloids in mature seeds (% of dry substance)

	Controls			GA [1]		GA [2]		GA [3]		GA [4]	
Sample	1	2	3	4	5	6	7	8	9	10	11
1st crop	0.0719	0.0742	0.0623	0.0577	0.0623	0.0637	0.0586	0.0681	0.0669	0.0547	0.0509
2nd crop	0.1064	0.0956	0.0893	0.0769	0.0951	0.0712	0.0787	0.0716	0.0953	0.0998	0.1005

The total alkaloid content of seeds from the first and second crop in GA-treated and untreated plants is shown in the Table. Alkaloid values in seeds obtained from the first crop of plants subjected to GA [1], [2] and [3] treatment were quite comparable to those obtained from the controls. However, in seeds obtained from GA [4]-treated plants alkaloid content was markedly lower. Figure 1 also illustrates this phenomenon. Seeds from the second crop of all plants were richer in total alkaloids than those of the first crop (Table). The average increase observed for the control plants and GA [1]-, [2]- and [3]-treated plants was 33%, and it amounted to 90% for plants which had been subjected to GA [4] treatment. Seeds from the latter plants had practically the same alkaloidal content as seeds obtained from the controls of this series. Evidently, the effects of the gibberellic acid treatment had been outgrown. Analysis of the alkaloidal pattern (Figure 2) revealed that the decrease in lysergic acid amide content of GA [4]-treated plants was largely responsible for the reduction of total alkaloids observed in the first crop. Patterns reflecting the behaviour of the second crop show that after GA [4] treatment the lysergic acid amide had again reached normal values whereas the clavine alkaloid concentration had somewhat increased.

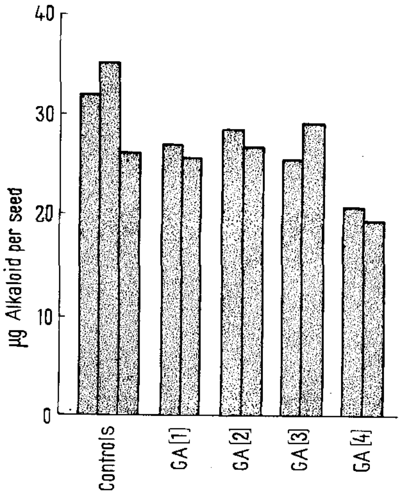

Fig. 1. Total alkaloids in µg per average seed of plants of *Ipomoea violacea* L. after spray treatment with: 100 ppm GA for 2 weeks, GA [1]; 500 ppm GA for 2 weeks, GA [2]; 100 ppm GA for 9 weeks, GA [3]; 500 ppm GA for 9 weeks, GA [4].

It should be noted also that the GA [4]-treated plants showed a reduced production of flowers and seeds and yielded only 6.5 and 6.8 mg of total alkaloids per plant in their seeds or 34.1% of the average production of the control plants[12].

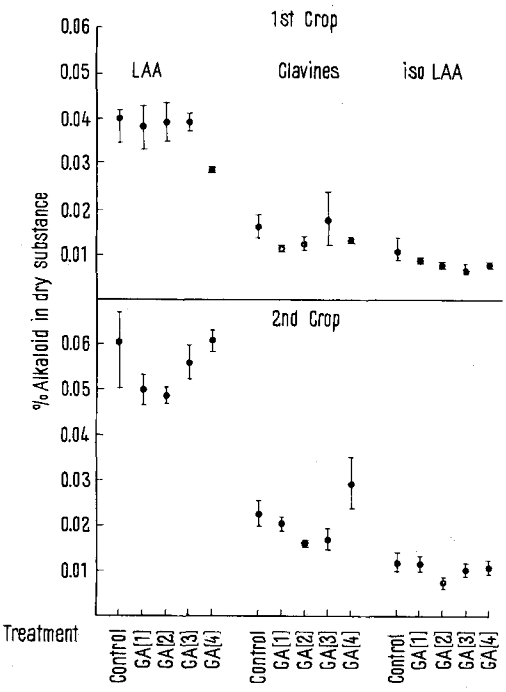

Fig. 2. % of lysergic acid amide (LAA), clavines and *iso*lysergic amide (isoLAA) found in first and second crop in dried seeds of untreated and GA-treated plants. Both the average values and the ranges are represented schematically.

Résumé. Une étude sur des plantes *Ipomoea violacea* (L.) dans une serre a démontré qu'un traitement prolongé de 500 ppm d'acide gibbérellique pendant 9 semaines a réduit le contenu d'alkaloïdes des graines de la plante. Un traitement semblable de 100 ppm et un court traitement de 500 ppm a produit très peu d'effets.

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