Table II. Effect of bethamethasone on gastric mucosal mast cell population (MCP)

Region of stomach	Mean MCP ± SE			p-Values		
	Group-I Control (10)	Group-III Adrenal intact rats with steroid (10)	Group IV Adrenalectomized rats with steroid (10)	I–III	I–IV	III–IV
Glandular	255±7	138±6	228±7	<0.001	0.01	<0.001
Pyloric	118±4	82±4	114±6	<0.001	0.6	<0.001
Rumen	56±3	43±3	47±4	0.01	0.1	0.6

Figures in parentheses denote the number of rats

Results. Table I shows that bilateral adrenalectomy has produced a highly significant (p 0.001 or less) increase in the gastric mucosal mast cell population, irrespective of the region of the stomach and the interval after adrenalectomy – 5 or 15 days. Extension of the post-operative period to 15 days has not increased the number of mast cells in any part of the stomach (p 0.02 or more). Table II shows that betamethasone has reduced the mast cell population in adrenal intact rats in comparison with the control rats; the decrease being significant in all the 3 portions of stomach. On the other hand, the mast cell population in bilaterally adrenalectomized group, which received betamethasone, is almost the same as that of the control group.

Discussion. A good correlation has been shown between histamine, mast cells and parietal cells in all the zones of the stomach of rats, normal and cortisone treated, by FOLEY and GLICK[8]. They concluded that histamine was probably liberated from the mast cells when they underwent degranulation in response to appropriate stimuli. The increase in number of mast cells in adrenalectomized animals indicates that in the absence of the circulating steroids the histamine stays bound within the mast cells. Adrenalectomy may thus remove a potent stimulus for degranulation of mast cells as compared with adrenal-intact rats. The consequent lack of histamine release in the gastric mucosa can well explain the gastric anacidity reported in Addison's disease and adrenalectomized animals. The lack of a significant increase in the mast cell population on extension of the post-operative period shows that adrenals have a definite time-limited influence on the turn-over of gastric mucosal mast cells. The significant decrease in the number of mast cells in the betamethasone-treated normal rats is probably due to degranulation of mast cells by the steroid. The results also indicate that this effect is inhibited in adrenalectomized rats.

It is logical to conclude from these observations that betamethasone and likewise adrenal glucocorticoids can liberate histamine from the gastric mucosal mast cells and histamine in its turn stimulates the gastric glands, particularly the parietal cells.

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Effect of Exogenous Application of Nucleic Acids and Auxin on the Rooting of Hypocotyl Cuttings of *Impatiens balsamina*. Evidence for the Uptake of Information Molecules

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Summary. Exogenously supplied DNA and RNA hastened root initiation and also increased the formation of roots on hypocotyl cuttings of *Impatiens balsamina* with intact apex and cotyledons. IAA appreciably increased the nucleic acid-caused enhancement in root formation. In combination with lower concentrations of nucleic acids, it even stimulated the growth of roots as well as of hypocotyls. Higher concentrations of nucleic acids were, however, toxic.

JAIN and NANDA[2] and NANDA et al.[3] have shown that the auxin-caused increase in the production of adventitious roots involves the synthesis of proteins, and that this effect is mediated through the multiplication of either DNA or RNAs or both. Auxins are also reported to induce the formation of new species of m- or t-RNAs during the initiation and development of roots[4] (also unpublished data). Investigations were undertaken in this laboratory to study the interaction effect of auxin with nitrogen bases and also with their nucleosides and nucleotides on the formation of adventitious roots. It was considered that it will shed some light on the molecular basis of auxin effect on this morphogenetic event. This paper deals with the effect of nucleic acids on the rooting of hypocotyl cuttings of *Impatiens balsamina*.

Seedlings of *Impatiens balsamina* var. Rose were raised from uniform seeds. Four cm long cuttings were made from the seedlings by excizing about the lower 1.0 cm rooted part and leaving behind 3.0 cm hypocotyl, the cotyledons and the apex intact. 240 such cuttings were

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Effect of DNA, RNA and IAA each alone and in combination, on the period to root initiation, the number of hypocotyl cuttings of *Impatiens balsamina* that rooted out of 10 and also on the number (figures within parenthesis) and length of roots and hypocotyls

Treatment (mg/l)	Period to root initiation (h)	No. of rooted cuttings and roots (within parenthesis)	Length of root (cm)	Length of hypocotyl (cm)
Water (control)	96	10 (8.2±0.37)	2.0	4.5
IAA 1.0	96	8 (14.8±0.62)	0.8	5.5
DNA 0.01	40	8 (7.8±0.34)	2.0	4.5
DNA 0.10	40	8 (10.6±1.22)	1.8	6.2
DNA 1.00	40	10 (15.2±0.36)	0.5	6.0
DNA 5.00	–	–	–	–
RNA 0.01	40	10 (11.8±1.02)	1.0	6.0
RNA 0.10	40	9 (14.0±1.00)	0.5	5.6
RNA 1.00	–	–	–	–
RNA 5.00	–	–	–	–
DNA 0.01+RNA 0.01	60	10 (10.0±0.97)	3.0	6.5
DNA 0.10+RNA 0.10	60	8 (10.7±0.93)	2.2	5.0
DNA 1.00+RNA 1.00	–	–	–	–
DNA 0.01+IAA 1.0	72	9 (13.4±0.44)	2.6	5.2
DNA 0.10+IAA 1.0	20	10 (16.3±1.41)	2.4	7.2
DNA 1.00+IAA 1.0	48	10 (25.3±0.93)	0.3	6.0
DNA 5.00+IAA 1.0	–	–	–	–
RNA 0.01+IAA 1.0	72	10 (18.0±1.72)	5.6	6.0
RNA 0.10+IAA 1.0	72	10 (26.0±1.43)	0.8	5.4
RNA 1.00+IAA 1.0	–	–	–	–
RNA 5.00+IAA 1.0	–	–	–	–
DNA 0.01+RNA 0.01+IAA 1.0	72	10 (24.8±2.20)	1.4	7.0
DNA 0.10+RNA 0.10+IAA 1.0	72	10 (26.6±2.00)	0.4	6.0
DNA 1.00+RNA 1.00+IAA 1.0	–	–	–	–

–, Died after 24 h; ±, SE.

divided into 24 equal groups and were planted vertically in holes on polythene sheets stretched over Petri-dishes (10 cm ⌀) containing the test solutions with varying concentrations of DNA and RNA each alone or together and with or without IAA.

The nucleic acids used were calf thymus DNA and yeast RNA and these were obtained from Sigma Chemical Co., USA. The test solutions were prepared in 30 μM chloramphenicol to prevent microbial contamination. An equivalent amount was added to water to serve as control. The cultures were maintained in an air-conditioned room at 28±3 °C and were exposed to continuous illumination (3200 Lux). Periodic observations of the number of rooted cuttings and the number and length of roots and hypocotyls were recorded for 14 days. The experiment was repeated 3 times with similar results. The results of 1 experiment, together with the treatments, are presented in the Table.

Number of roots. Both DNA and RNA hastened root initiation, the effect being most pronounced with 1.0 mg/l DNA+1.0 mg/l IAA. Thus, roots were produced within 20 h in 0.1 mg/l DNA+IAA, in 40 h in DNA and RNA each alone, but delayed to 96 h in IAA or water.

Both the nucleic acids enhanced root formation, the effect increasing with concentration and 1.0 and 0.1 mg/l of DNA and RNA respectively being most effective. Higher concentrations proved toxic as cuttings died without rooting. IAA appreciably increased the enhancement caused by both nucleic acids, the effect again being more pronounced in combination with 1.0 mg/l DNA or 0.1 mg/l RNA. It may be noted that these concentrations of the two together in combination with IAA proved toxic, so that the cuttings died within 24 h.

Length of roots. IAA depressed root elongation (Table). The depressing effect was overcome by both DNA and RNA. In fact, in lower concentrations these nucleic acids stimulated root growth, 0.01 mg/l RNA alone being most effective in this regard. However, root elongation was depressed by 1.0 mg/l DNA and 0.1 mg/l RNA.

Hypocotyl length. The lower concentrations of both nucleic acids, each alone or together, and with or without IAA, stimulated hypocotyl elongation. Callus was formed at the basal end of cuttings cultured in DNA+RNA+IAA.

The rooting of hypocotyl cuttings with intact apex and cotyledons in water reported in this experiment lends support to the earlier results that cotyledons and apex serve as sources of nutrition and auxin, respectively, that are needed for the formation of adventitious roots[5]. The more profuse rooting with IAA in the medium indicates that the level of nutrition was adequate and that a proper balance of the two is necessary for optimal production of roots[6]. The hastening and enhancing effect of lower concentrations of both the nucleic acids on rooting is rather interesting and is suggestive that these bio-molecules are either taken in as such or after these are hydrolyzed into nucleotides or nucleosides, and after entry are reconstituted into the system to cause increased level of DNA or RNAs or both, to produce proteins that are needed for

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the differentiation of cambial derivatives into roots. It may not be out of place to mention here that even starch can be used as a source of carbon for the supply of energy required for root initiation, and that it is mobilized into sugar by the enzymes that leach out of the segments into the medium[7].

Another interesting point that emerges from these results is that the auxin enhances the effectiveness of exogenously applied nucleic acids. The results thus lend support to the postulate that auxin probably acts as a triggering agent at the transcription level, and nutrition serves as a source of carbon to regulate translation[8]. The results are of particular significance in the light of evidence which suggests that the exogenously supplied DNA and RNA effectively enter in the intact cells and protoplasts of various eukaryots[9]. The regulated uptake of exogenous DNA molecules in the cells of plant origin and their expression is also suggested by the work of some others[10–14]. Infact, LESHEM and GALSTON[15] showed that RNA which is extracted from tobacco pith cells and is vaccuum-infiltrated into similar receptor cells, alters the pattern of isoperoxidases in the receptor tissue.

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Spread of Cobalt from a Cortical Epileptic Lesion Induced by a Cobalt-Gelatine Implant into the Frontal Cortex of the Rat

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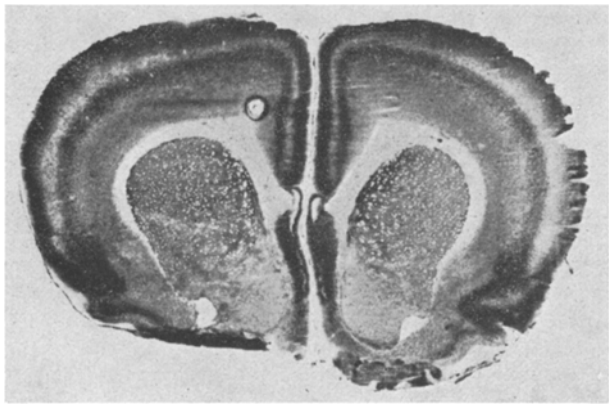
Summary. The spread of cobalt ions from cobalt induced epileptic foci in rats has been investigated. Atomic absorption spectrophotometry and heavy-metal histochemistry reveal cobalt ions spread very widely from the focus. Biochemical and physiological consequences for this model of epilepsy are discussed.

KOPELOFF et al.[2] first reported that the application of powdered cobalt metal to the frontal cortex of the monkey produced epileptiform spikes in the electroencephalogram (EEG). Since KOPELOFF's original observation the application of cobalt powder to the cortex or the insertion of cobalt gelatine pellets into brain has been used to produce reproducible epileptic foci in a variety of animals[3]. In the rat, application of cobalt to the cortex produces a distinct secondary focus in the contralateral cortex[4,5]. Because of the use of cobalt salts to trace axonal pathways[6] we were interested to know if the secondary focus, formed in the contralateral cortex of the rat did contain significant amounts of cobalt. If this were so then the value of this model would be reduced. Previous workers using this model[4,5] have suggested that the secondary focus in this model arises as a response to the spread of electrical signals from the primary focus across the corpus callosum and represents a response similar to the kindling phenomenon described by GODDARD[7]. The presence of significant amounts of cobalt in the secondary focus would mean that it is probably solely caused by the presence of cobalt ions.

Cobalt-gelatine pellets prepared as described by FISCHER et al.[8] of standard size 1 mm diameter and maximally 0.5 mm thick (representing at most 1 mg of cobalt metal in gelatine) were inserted into the right frontal cortex of male PVG rats as described in detail by Dow et al.[4]. The histochemical distribution of cobalt ions after this implant was investigated using the TIMM[9] staining method at 4, 28 and 60 days after implantation. The rats were anaesthetized with an overdose of barbi-

A



A) The distribution of sulphide silver positive material (heavy metals) in the frontal cortex as revealed by the TIMMS method. Note the uneven distribution of staining in the cortical layers.

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