stituted for thymidine in virion DNA was determined by equilibrium centrifugation in CsCl (Fig. 3). The distributions of BUdR-substituted virion and cellular DNA's in these gradients are virtually identical. This observation supports our previous suggestion that virion DNA is derived from the normal pool of cellular DNA without special selection. The densities of the once-replicated (HL, $\rho = 1.74$–1.75) and twice-replicated (HH, $\rho = 1.78$–1.79) DNA's are indicative of maximum substitution by BUdR (*16*).

We conclude that the DNA associated with purified virions of RSV consists of a random sample of cellular DNA in a low-molecular-weight form. The possibility that the DNA is enclosed in "pseudovirions", rather than in biologically active virions, is still undetermined. However, the presence of small amounts of ribosomal RNA in RSV (*1*, *6*) and other RNA tumor viruses (*1*) does point to the inclusion of normal cellular elements in virus preparations, either as adventitious contaminants or as virion constituents. A report that DNA is associated with the plasma membrane of human diploid cells (*17*) raises the possibility that the DNA found in oncornaviruses is an envelope constituent, acquired when virions are released from the host cell. Examination of purified viral nucleoids (*18*) for the presence of DNA should provide a test of this suggestion. Whatever its source, the virion DNA of RNA tumor viruses must be taken into account in any study of the RNA-directed DNA polymerase present in these viruses. We have evidence that as much as 5 to 10 percent of the total double-stranded DNA synthesized in vitro by enzyme-active virions consists of transcripts of the avian DNA associated with RSV virions. Other investigators have suggested that virion DNA might serve as the primer for initiation of RNA-directed synthesis within the virion (*19*), but more recent observations indicate that this function is served by a ribopolynucleotide (*20*). For the present, we consider the most reasonable conclusion to be that the DNA present in preparations of RSV is probably devoid of any special function in the life cycle of the virus.

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Systemic Activity of a Juvenile Hormone Analog

Abstract. *The peptidic analog of insect juvenile hormone, ethyl pivaloyl-L-alanyl-p-aminobenzoate with enormous biological activity on the red cotton bug,* Dysdercus cingulatus, *has pronounced systemic effect in sunflower plants. The compound is absorbed by the plant tissues and appears to be translocated throughout the plant system in active form. There is some evidence that juvenile hormone analogs of other types also have similar systemic effects. The discovery of systemic action should aid in the possible utilization of juvenile hormone analogs in insect control programs.*

Juvenile hormone (JH) inhibits cell differentiation in insect metamorphosis and stimulates growth of the ovaries in adult life. Many natural and synthetic compounds exhibit JH activity (*1*) and are effective in contact applications as well as in the diet. Because of their high biological activity and selective action, the juvenile hormone analogs (JHA's) are considered to be potential pesticides (*2*).

In order to explore all possible methods of application of the JHA, we have studied their systemic activity. By analogy with conventional insecticides we realized that such a systemic JHA must be absorbed by the plant system, should be relatively stable, enormously active on insects in peroral application, and at least partly water soluble. With this in mind we selected a few compounds that seemed to have at least some of the above-mentioned qualities. Special attention was paid to the recently discovered (*3*, *4*) group of peptidic JHA which are relatively stable, are highly active on *Dysdercus cingulatus*, and are active when administered in solution in drinking water. We selected the ethyl ester of pivaloyl-L-alanyl-*p*-aminobenzoic acid since as little as 0.00004 μg topically applied to freshly molted last instar larvae will cause formation of half-larval adultoids which are unable to reproduce. When administered in drinking water, adultoids appear already at the concentration of 0.0001 part per million in water.

Since the terrestrial seed feeding Hemiptera like *Dysdercus* invariably require water for their development, they suck the sap of any available plants, in the absence of other water. We used this phenomenon as an indicator of the presence or absence of the compound in the untreated parts of the plant system. We used sunflower plants that were approximately 20 cm long, cultured under greenhouse conditions. Freshly molted last instar larvae of *Dysdercus cingulatus* reared up to this stage on dry cotton seeds and

Table 1. Systemic activity of the juvenile hormone analog, ethyl pivaloyl-L-alanyl-*p*-aminobenzoate in sunflower plants on *Dysdercus* larvae. The values indicate average scores of hormonal activity as explained in the text.

Treated part	Tested part	Time to release (days)	Activity at concentrations of (micrograms per plant)					
			1000	500	250	125	62.5	None
Stem	Upper leaves	1	5	5	4.5	3.6	1.2	0
Stem	Upper leaves	7	5	4.6	3.8	1.4	0.4	0
Stem	Upper leaves	14	3.4	1.8	0.8	0	0	0
Upper leaves	Stem	1	5	4.5	4	3.4	2.3	0

water were used as the test insects.

The compound was applied on the surface in 100 μl of water emulsions containing 0.01 percent Tween 80, 1 percent paraffin oil, and graded amounts of the compound from 0 to 1000 μg per 100 μl of the emulsion. Immediately after application, the treated parts of the plant were isolated in an inverted plastic cup. Larvae were released on the untreated part of the plant 24 hours after application of the emulsion and were enclosed in a small plastic cup containing a few cotton seeds as food.

The evaluation of juvenile hormone activity was made according to the degree of metamorphosis inhibition determined by morphological criteria after the next molt; the usual 0 to 5 scoring system was used (*4*). Maximum activity (score 5) requires appearance of perfect supernumerary larvae; score 3 indicates formation of half-larval adultoids; and score 0 indicates formation of morphologically perfect adults. Each experiment was replicated twice with six to ten insect specimens per replication. The values in Table 1 are an average activity score of all insects tested in each concentration.

Initially, excessive amounts of the compound showed systemic activity even when applied in pure acetone or pure mineral oil, but this crystalline compound formed a solid deposit or unabsorbed film of oil on the treated plant surface, which was inconvenient for quantitative measurements. Therefore, in later experiments, water emulsions with Tween 80 were applied on the stem or upper leaves of the sunflower plant. The results presented in Table 1 show that the compound, at about 100 μg per plant, is able to enter the plant system and exhibit systemic juvenile hormone activity per plant. The data indicate that the compound is transported from the lower part to the upper part of the plant and vice versa, which indicates translocation and distribution throughout the whole plant system. We also found that the compound can enter the plant through the roots for translocation to the stem and leaves. For these experiments we used sunflower plants potted in 50 to 100 g of soil per pot. The soil in the pot was treated once with 5 ml of the above emulsion containing 12.5 to 200 μg of JHA per milliliter. The insects were released on the untreated parts 24 hours after the soil treatment. The results revealed almost maximum activity at the concentrations of 500 to 1000 μg per plant. Little activity has been observed in concentrations of 125 to 250 μg per plant, while no activity was recorded at lower doses.

Table 1 shows that the compound remains relatively active in the plant system for 1 week after application, although there is a remarkable decrease of systemic JH activity after 14 days, except at the highest concentration tested.

In our preliminary experiments with other chemical types we found lower systemic activity in certain terpenoid compounds. We expect that the observed systemic effect of JHA may enhance the potential utilization of these substances in insect control.

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Thyroid State: Effects on Pre- and Postsynaptic Central Noradrenergic Mechanisms

Abstract. *For hypothyroid rats, spontaneous motor activity was less than that in matched normal controls, and the specific activity of tyrosine hydroxylase in the midbrain was significantly greater than that in controls. Rats made hyperthyroid with thyroxine became hyperactive and showed increased sensitivity to the behaviorally activating effects of norepinephrine administered intraventricularly. In hyperthyroid rats, the specific activity of tyrosine hydroxylase in the midbrain remained within the normal range. These results are consonant with studies that suggested both receptor "tuning" and feedback regulation of activity of enzymes involved in biosynthesis of presynaptic neurotransmitter as methods of regulation of the central catecholamine synapse. These results may also help explain the reported potentiation by thyroid hormone of the antidepressant effects of imipramine.*

Some of the physiological manifestations of altered thyroid states may be mediated through peripheral and central adrenergic systems (*1*). For example, thyroxine treatment produces a state resembling hyperthyroidism; this state is characterized by hyperthermia, tachycardia, sweating, and elevated blood pressure. These symptoms are all manifestations of increased adrenergic activity (*2*). Conversely, hypothyroid animals generally show signs of decreased peripheral adrenergic function (*3, 4*). In addition, affects of norepinephrine (NE) and epinephrine on basal metabolic rate, glycogenolysis, and lipase activation are markedly increased in thyroxine-treated subjects (*4*). Finally, in heart and other adrenergically innervated peripheral tissues, turnover of NE is decreased in thyroxine-treated animals, while an accelerated turnover is associated with a hypothyroid state (*5*).

A similar relationship between thyroxine and catecholamines (CA's) may exist in the central nervous system (CNS). Similarities between the symptomatology of thyroid deficiency and of certain psychiatric illnesses, particularly "endogenous" depressions, are well known (*6*). Since much evidence supports the notion that central CA's play an important role in the patho-