reducing its average altitude and therefore the time required to complete each circuit. Detailed calculations, based on the equations of satellite motion, then determine the quantitative relation between the reduction in the period and the average air density in the orbit.

Since the density falls off very rapidly with increasing altitude, the average density in the orbit is heavily weighted by the contributions near perigee. Our calculations indicate that for both satellites, as also in the case of Sputnik I (*3*), the rate of change of period actually determines the density at an altitude approximately 50 km above perigee.

Tables 1 and 2 list the anomalistic periods and corresponding rates of change of period for the Explorer I and Vanguard I satellites, as issued by the Vanguard Computing Center. The tables also give the estimated values of the ballistic drag parameter, M/C_dA (M = satellite mass, A = satellite area projected along the direction of motion, and C_d = drag coefficient). We note that the observed rate of change of period determines only the ratio of the density to the ballistic drag parameter, hence a knowledge of this parameter is essential for the density analysis. The large probable error indicated in the value of M/C_dA for Explorer I represents the uncertainty in the cross-sectional area of that satellite. Explorer I has a cylindrical shape with a length of 203 cm and a radius of 7.3 cm, and the maximum and minimum values of its projected area may therefore vary by a factor of 20, depending on the orientation of the cylinder relative to the direction of motion. In our analysis the projected area is estimated by averaging over all orientations of the satellite, but a proper calculation of the effective area is extremely complicated in the present case because the motion of Explorer I about its center of mass cannot be properly described by either a random tumbling or a uniform precession about a fixed spin axis (*4*). We consider our estimate of the projected area to be uncertain by a factor of 2.

Table 3 gives the densities which we obtain from the time-weighted averages of dP/dt in Tables 1 and 2, together with the altitudes to which these densities refer. The probable errors in the average density represent the combined effects of the uncertainty in area and the variations in dP/dt during the period covered by the observations. The densities in Table 3 correspond to a mean scale height of (73 ± 10) km and a mean temperature of $1250° \pm 200°$K for the region between 400 and 700 km.

Although the density value based on Explorer I has a large probable error, this result is still of substantial interest because, when it is combined with the Vanguard I density, it gives us an indication of atmospheric conditions in a latitude region not covered by the results from Sputnik I and the earlier rocket flights. The perigee of Sputnik I was located at a latitude of $39° \pm 6°$N during the period on which the density analysis of that satellite was based, and the rocket data which we combined with the sputnik value were obtained at latitude 33°N. On the other hand, the orbits of the Explorer and Vanguard satellites are confined to the region centered on the equator and lying between 33°N and 33°S. Since perigee rotates at the rate of 6° per day in the orbital planes of these satellites, the average densities for the intervals covered in Tables 2 and 3 constitute a thorough sampling of all latitudes in this region. Thus the combination of rocket and Sputnik I data describes a temperate-zone atmosphere, while the Explorer and Vanguard results refer to a band of latitudes centered about the equator.

According to recent results of LaGow at 200 km (*5*), the summer day-time density at 59°N is 8 times the corresponding density at 33°N. We expect comparable differences between the present results and our earlier model for temperate latitudes, but in fact the densities of Table 3 are only 30 to 50 percent less than the lower limit of probable error in the temperate zone atmosphere (model *a* of reference *2*). Presumably the comparison must also allow for diurnal and seasonal variations, which are as yet very poorly determined.

As a corollary to the preceding remarks, it is interesting to note that because of the strong latitude dependence of upper atmosphere densities, we cannot compare density determinations based on the orbits of the present United States satellites with those obtained from U.S.S.R. satellites.

Table 2. Orbital periods for Vanguard I, derived by the Vanguard Computing Center from Minitrack data. The third column gives the average value of dP/dt, obtained from the tabular differences in the first and second columns.

Date	P (min)	dP/dt (min/day)
1 May	134.277	
		$(3.0 \pm 0.5) \times 10^{-4}$
7 June	134.266	
		$(2.8 \pm 0.8) \times 10^{-4}$
25 June	134.261	
Weighted av. (min/day)		$(3.0 \pm 0.6) \times 10^{-4}$
M/C_dA		(24 ± 8) kg/m^2

Table 3. Densities derived from satellite data.

Altitude (km)	Density (g/cm^3)
405 (Explorer I)	$9^{+6}_{-4} \times 10^{-15}$
720 (Vanguard I)	$(1.2 \pm 0.3) \times 10^{-16}$

I. Harris
R. Jastrow
Nucleonics Division, U.S. Naval Research Laboratory, Washington, D.C.

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1. We are very much indebted to W. F. Cahill and C. Wade, Jr. for their cooperation in carrying out the necessary computations on the IBM 704 computer at the National Bureau of Standards.
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Growth Promotion in Pea Epicotyl Sections by Fatty Acid Esters

The growth of excised pea epicotyl sections is less than that of a similar section left on the intact plant, even if optimum concentrations of indole acetic acid (IAA), gibberellic acid (GA_3) (*1*), sucrose, and cobalt (*2*) are supplied. In an investigation (*3*) of this failure of excised sections to grow optimally, fatty acid esters have been found to bring about section growth promotions much larger than any previously reported in this standard auxin bioassay material.

The peas used were the customary bioassay variety, Alaska, and the dwarf variety, Laxton's Progress. The technique utilized was essentially that of Christiansen and Thimann (*4*), except that ten 10-mm sections were employed in 20 ml of solution, a rotary shaker was employed, the *p*H was 5.5, and, in the case of the dwarf pea, sections were cut at 6 days of age. Since it is especially necessary that the dwarf pea receive a standard amount of red light during development, a continuous illumination of about 0.3 erg/cm^2/sec was supplied from a 1-watt neon bulb filtered through 1/8-inch thick pieces of No. 2444 red and No. 2082 green Plexiglas.

Pea sections so prepared showed a large increase in growth over that of con-

Table 1. Dependence on auxin of the pea fraction response. Percentage increase in length of 10-mm subapical sections of third internode of Laxton's Progress pea seedlings taken at 6 days of age, after 24 hours in 1.25 percent sucrose + $5 \times 10^{-5}M$ $CoCl_2$ + $5 \times 10^{-3}M$ KH_2PO_4 (*p*H 5.5), and additives as listed. Standard deviations are cited. All solutions initially 0.2*M* in acetonitrile.

Additive and amt. (mg/lit.)	No pea fraction	40-mg/liter pea fraction
Controls	29.7 ± 5.3	16.6 ± 2.5
GA_3 (0.1)	30.6 ± 3.6	25.5 ± 7.0
IAA (0.3)	51.2 ± 6.0	61.5 ± 5.4
GA_3 (0.1) and IAA (0.3)	58.7 ± 17.7	94.3 ± 10.1

trols when boiling ethanol extracts of dwarf or Alaska pea seeds, epicotyls, roots, or tips were added to the bioassay solutions. This growth response (Fig. 1*A*), as large as 130 percent above initial length in some other experiments, was markedly greater than that produced by adenine, uracil, and certain amino acids (*5*). But the growth of the sections was still not entirely restored to that of the intact plant, on which comparable sections grow about 140 percent for the dwarf and some 200 percent for the Alaska pea in the same period.

The ethanol extract of Alaska pea seeds was concentrated by dialysis, extraction of the nondialyzables with benzene, and vacuum distillation of the resulting benzene extract (bp 140° to 160°C at pressure of about 0.1 mm-Hg). This pea fraction is apparently largely a mixture of glycerides, and its further study was hampered by its insolubility in water. Tests of detergents to disperse it led to the surprising discovery that Tweens 20, 80, and 81 (*6*) had activity themselves; this activity was nearly as large as that of the fraction isolated from peas (Fig. 1*B*). To obviate this difficulty, acetonitrile was adopted as a dispersing agent. At concentrations less than 0.3*M*, it was found to be physiologically inert, and its presence had no marked modifying influence on the results reported here.

Some of the Tweens have been shown previously to promote specifically the growth of *Lactobacillus* (*7*) and of a *Venturia* mutant (*8*). In these lower plants, the effect of the Tweens was traced to their content of unsaturated fatty acids, but it was also mimicked by biotin. In peas the growth obtained with methyl linoleate was as great as that obtained with the isolated natural fraction, although the optimum was sharper—that is, high concentrations were more inhibitory; methyl oleate was also active (Fig. 1*C*), and methyl linolenate behaved similarly. But unlike the response in lower plants, the response in peas was not notably *p*H-sensitive, and biotin was completely inactive on peas. It also differed in that saturated fatty acid esters produced a response. The saturated esters are less satisfactorily studied quantitatively because of dispersion problems.

A wide range of other vitamins, yeast extract, kinetin, traumatic acid, diphenyl urea, adenine, uracil, uridine, cytidine, glycerol, triacetin, batyl alcohol, and lecithin failed to produce comparable elongations, nor have any of these compounds yet been shown to enhance significantly the response to the fatty acid esters. It seems likely, therefore, that the activity of the isolated pea fraction can be largely, if not entirely, attributed to its content of fatty acid esters, rather than to a specific glyceride.

Other peas, the dwarfs Progress No. 9 and Little Marvel, were also shown to respond to the pea glyceride fraction. In growth of *Avena* coleoptile sections, on the other hand, no significant effect except inhibition by the pea fraction or by unsaturated acid esters has been noted in any test to date.

The effect of the isolated glyceride fraction and of methyl linoleate is apparently to promote the sensitivity of the pea sections to IAA. The pea fraction alone is ineffective or inhibitory. In the presence of GA_3 alone it has a very small effect, while in the presence of both IAA and GA_3 its maximum effect is exhibited (Table 1). Even larger promotions can be noted in other experiments (Fig. 1).

The quantitative relations suggest that a hormonal rather than a gross nutritive role is played by the acid esters. Sections from Laxton's Progress peas give optimal growth in $2 \times 10^{-6}M$ IAA and $7 \times 10^{-5}M$ methyl linoleate or oleate. Sections from Alaska peas at this IAA level (which is suboptimal for them) respond optimally to methyl linoleate at $3 \times 10^{-5}M$. The effective ester concentrations must actually be less since it has been necessary to supply these compounds as an acetonitrile dispersion and not as a true solution. The esters are thus effective in quantities at most only 20 to 35 times larger than that of the hormone, IAA. This suggests a close relationship of auxin activity to lipid metabolism, a conclusion fortified by the results of Christiansen and Thimann (*4*), who found that inhibition of lipid utilization in pea sections paralleled inhibition of growth, and which has also been advanced on less direct grounds by Bennet-Clark (*9*).

These experiments indicate a need for a reassessment of the conditions for the pea-section auxin bioassay. They also confirm that dwarf peas are similar in auxin response to standard peas (*10*).

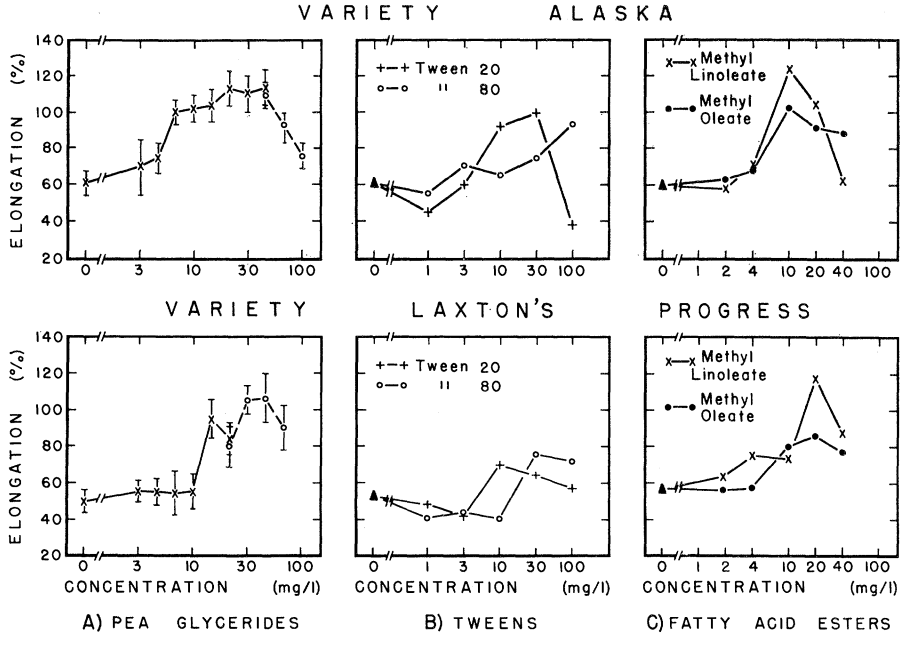

Fig. 1. Growth of two varieties of pea epicotyl sections in various fatty acid esters. All sections were grown in 1.25 percent sucrose + $5 \times 10^{-5}M$ $CoCl_2$ + $5 \times 10^{-3}M$ KH_2PO_4 (*p*H 5.5) + 0.3 mg of IAA per liter + 0.1 mg of GA_3 per liter; these conditions provide maximum growth of Laxton's Progress peas. Percentage increase in length over the initial 10-mm section after 24 hours is plotted against concentration in milligrams per liter, the latter being a log scale. *A*, Pea fraction isolated as in text. Standard deviations indicated by bars. Crosses and circles indicate separate experiments. *B*, Plus marks, Tween 20. Circles, Tween 80. *C*, Crosses, methyl linoleate. Dots, methyl oleate. In *A* and *C*, the solutions also initially contained 0.25*M* acetonitrile.

However, in the case of gibberellic acid, which has so large an effect on intact peas and only a small effect on excised sections (*11*), this investigation has failed to show any specific promotion of GA_3 action by any extract or additive tested. These facts suggest that the analysis of accessory growth factors, such as the "calines" proposed by Went (*12*), has continuing pertinence in the study of plant growth (*13*).

BRUCE B. STOWE
Biological Laboratories, Harvard University, Cambridge, Massachusetts

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Action of Salicylate on Metabolism of Acetate-2-C^{14} in the Rat

Salicylates have been shown to uncouple oxidative phosphorylation reactions in mitochondrial preparations (*1*), and this action may explain the increased oxygen consumption observed after the administration of salicylates to man (*2*) and to the rat (*3*). It may also be related to some of the effects of salicylates on carbohydrate metabolism in animals and isolated tissues. Thus, although an increased rate of glycogenolysis due to release of adrenaline is an important factor (*4*) in the depletion of glycogen caused by salicylate in the normal rat (*5*), impairment of glycogenesis due to an inadequate production of energy-rich phosphate bonds may also be concerned, particularly since salicylate diminishes glycogen synthesis in isolated rat-liver slices (*6*). The hypoglycaemic action of salicylate in the alloxan-diabetic rat (*7*) may also be interpreted as resulting from defective carbohydrate synthesis.

Table 1. Effect of salicylate on the incorporation of C^{14} in the liver glycogen and on the excretion of $C^{14}O_2$ and $C^{12}O_2$ in the breath of rats given acetate-2-C^{14}. Results are expressed as means plus or minus standard deviation. The significance of the differences between the means of the control and salicylate groups has been analyzed by the *t* test, and values of *P* are included.

Rats (No.)	Liver glycogen d.p.m./mg. (disintegration/min mg)	Total CO_2 [(0–60 min) mg/min]	$C^{14}O_2$ Cumulative % injected dose (0–60 min)	Specific activity of total CO_2 (μc/g) C/10 μc injection		
				10 min	30 min	60 min
			Control			
6	5193 ± 1640	7.32 ± 0.45	32.94 ± 2.01	2.43 ± 0.20	3.48 ± 0.37	2.28 ± 0.19
			Salicylate (500 mg/kg)			
4	20 ± 12.5 $P = 0.01$	14.70 ± 1.52 $P = 0.01$	52.27 ± 3.93 $P = 0.01$	1.87 ± 0.24 $P = 0.2$	3.50 ± 0.35 $P = 0.9$	1.42 ± 0.17 $P = 0.05$
			Salicylate (250 mg/kg)			
4	60 ± 18 $P = 0.01$	9.25 ± 0.40 $P = 0.02$	48.22 ± 4.33 $P = 0.02$	2.05 ± 0.28 $P = 0.4$	4.26 ± 0.41 $P = 0.3$	2.17 ± 0.13 $P = 0.8$

The effect of salicylate on the appearance of C^{14} in the liver glycogen and expired CO_2 of rats given acetate-2-C^{14} has been studied (*8*). Male rats of the Long-Evans strain, weight 200 to 250 g, were fasted for 24 hours and given 3 millimoles of sodium lactate by stomach tube at the beginning of the experiment. Thirty minutes later they received sodium salicylate by intraperitoneal injection; acetate-2-C^{14}, approximately 10 μc per rat, was administered by the same route after a further 30 minute interval. The radioactivity and CO_2 content of the breath were measured for 1 hour by the continuous recording equipment described by Tolbert, Kirk, and Baker (*9*). The rats were then killed by an intraperitoneal injection of Nembutal, the liver was excised, and the liver glycogen was isolated by the method of Marks and Feigelson (*10*) and purified to constant specific activity according to the directions of Stetten and Boxer (*11*).

The results, given in Table 1, show that salicylate, in a dose of either 250 mg or 500 mg/kg of body weight, inhibited the incorporation of C^{14} into liver glycogen after the injection of the labeled acetate. The higher dose of salicylate caused significant increases in both the $C^{12}O_2$ and the $C^{14}O_2$ but did not change the specific activity of the total CO_2. A similar but less marked pattern was observed with the lower salicylate dose.

The major pathway by which acetate carbons are incorporated into liver glycogen is via acetyl-coenzyme A, the Krebs cycle, decarboxylation of oxaloacetate to give phosphopyruvate, and the modified reversal of the Embden-Meyerhof scheme of glycolysis (*12*) and energy-rich phosphate bonds are necessary at various intermediate steps. The virtual absence of radioactivity in the liver glycogen of the salicylated animals, therefore, is consistent with the view that salicylate impairs carbohydrate synthesis by interfering with the production of energy-rich phosphate bonds. It has been emphasized by Weinman, Strisower, and Chaikoff (*12*) that the mere demonstration of incorporation of isotope into glycogen does not necessarily mean that a net synthesis of glycogen from a labeled fatty acid has occurred. These workers consider that glycogen synthesis from acetate is made possible only by an additional (that is, nonacetate) influx of Krebs cycle intermediates into the cycle. The administration of lactate to the animals in the present work could provide such an influx and make possible the net increase of glycogen via the synthetic reactions outlined above.

The increased excretion of $C^{12}O_2$ and $C^{14}O_2$ in the breath of the salicylated rats may be a direct result of the well-known action of salicylate in causing hyperventilation by stimulation of the respiratory center. However, a contributory factor may be an increased substrate breakdown as a consequence of inefficient phosphorylation mechanisms.

M. J. H. SMITH
Donner Laboratory for Medical Physics, University of California, Berkeley

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