pletely suppressed. These seeds, if thoroughly washed with water and transferred to distilled water for an additional 4 days, failed to develop roots. At lower concentrations partial inhibition of root elongation occurred (Table 1). It was established from this and another experiment that  $1.5 \times 10^{-5} M$  (2.6 µg/ml) would result in 50-percent inhibition of elongation In similar but less extensive experiments with barley and flax, elongation of the roots of the former was 50 percent inhibited by approximately  $2.5 \times 10^{-5}M$ azaserine, and of the latter at the low concentration of approximately  $4 \times 10^{-6} M$  (0.7 µg/ml).

In aerated water culture the growth of barley roots. var. Moore, was repressed by comparable concentrations of azaserine. The criterion in such tests is the dry weight of roots produced in 5 days at 25°C (Table 2). In this system the concentration producing 50-percent inhibition would be  $1.3 \times 10^{-5}M$  (2.25)  $\mu g/ml$ ).

Shoot growth is not affected at levels causing substantial repression of root development. Azaserine has been applied in the following ways to 8-day bean seedlings grown in vermiculite: (i) 50 µg in 0.02 ml 50-percent ethanol to the base of the unifoliate leaf;

Table 1. Azaserine repression of root elongation of germinating cucumber seed (96 hr at 25°C).

$\begin{array}{c} \text{Concentration} \\ (\mu \mathcal{M}) \end{array}$	Inhibition in root length (%)	
200	100	
100	84.1	
40	69.4	
10 .	38.7	
3.3	14.2	

Table 2. Azaserine repression of root growth of barley seedlings (5 days at 25°C in the dark).

Concentration $(\mu M)$	Reduction in root dry weight (%)		
30	73.2		
10	43.0		
3	15.4		

(ii) 100 µg in water, similarly; (iii) unifoliate leaf dipped in  $1 \times 10^{-5}M$  solution for 48 hr; (iv) same as (iii) using  $1 \times 10^{-4}M$ ; (v) 25 ml  $1 \times 10^{-4}M$  solution applied to the surface of the root medium. None of these treatments produced any apparent inhibition or stimulation of growth of the shoot or of any part thereof, nor were there morphologic changes or telemorphic responses as occur with growth regulators.

These studies indicate that root growth of cucumber, barley, and flax seedlings is repressed at azaserine concentrations substantially lower than those which cause inhibition of many microorganisms, and of the same order as those reported to be inhibitory to the most sensitive microorganisms. The possibility of employing a plant system for azaserine assay is suggested.

#### **References** and Notes

- 1. J. Ehrlich et al., Nature 173, 72 (1954).
- G. R. Bartz et al., Nature 173, 72 (1954).
  G. L. Coffey et al., Antibiotics & Chemotherapy 4, 775 3. (1954)
- Available through the courtesy of George Rieveschl, Parke, 4. Davis and Co., Detroit, Mich.
- Paper No. 2 from the Plant Nutrition Laboratory, Michi-5. gan Memorial Phoenix project No. 32, of the University of Michigan.
- 6. D. Ready and V. O. Grant, Botan. Gaz. 109, 39 (1947).

27 October 1954.

# Communications

# Wastes and Nutrients

In a recent article by Paul B. Sears [Science 120, 959 (1954)] appears the remarkable statement on page 960, line 7, "Nature long ago discarded the nonsense of carrying poisonous wastes and nutrients in the same vessels." To paraphrase Herbert Dingle's recent delightful article [Science 120, 513 (1954)] "This statement sounds like nonsense, because it is nonsense."

The blood supply must not only convey materials destined for the retail consumers but also remove their garbage, for, apart from incidental losses occasioned by the operation of externally secreting glands, there are no other means available. Even the idea that arterial blood is a supply system, and venous blood a garbage-removal system, is entirely incorrect, both factually and philosophically. The urgent necessity of keeping the individual consumer, or tissue cell, in

a reasonably ordered and stable environment-the well-known homeostasis of Cannon-is such that sudden and marked changes of all kinds are undesirable. (This matter might in itself appear to warrant serious ecological investigation.) In reality it is found that the system of supply, as well as the system of garbage removal, work on percentage rather than on absolute changes, and in this way the shocks of both deliveries and pick-ups are minimized. In this matter, the presence of garbage, especially some kinds like urea, plays a very useful role indeed, and we are in consequence led to a second criticism that the common and thoughtless use of such phrases as "poisonous wastes" on occasion may also equally become "nonsense."

O. S. GIBBS

Jefferson Medical College of Philadelphia

13 December 1954.

SCIENCE, VOL. 121

In my paper on "Human ecology" [Science 120, 962 (1954)] the reference to "the late Otto Glaser" should read "Otto Glasser." Otto Glaser, professor emeritus of biology at Amherst, died in 1951. I am happy to report that Otto Glasser of the Department of Biophysics, Western Reserve Medical School, and editor of the excellent handbook of Medical Physics is still very much alive.

To O. S. Gibbs of Jefferson Medical College I am indebted for a vigorous objection to my somewhat unguarded statement that "Nature long ago discarded the nonsense of carrying poisonous wastes and nutrients in the same vessels." This is of course not literally true, except as mass effects are concerned, and they are what I had in mind. In both "pure" rivers and "pure" arterial blood there are materials with a considerable range of physiological properties, their effects being regulated by what amount to homeostatic processes. These processes break down in streams overcharged with human waste, industrial and domestic, and since we often depend on such streams for public water supply, a situation amounting to biological nonsense does exist.

Perhaps I should have used the analogy of upper and lower alimentary canal rather than artery and vein. But since the bulk of correspondence concerning the article has come from medical men, none of whom have raised the issue, I assume that my intent was clear and the analogy was not wholly inappropriate. PAUL B. SEARS

Conservation Program, Yale University, New Haven, Connecticut

6 January 1955.

## C<sup>14</sup>-Labeled Ergot Alkaloids

To provide detectable material for *in vivo* studies with the alkaloids and their derivatives and to enable an extension of our investigations on their biosynthesis, we have produced  $C^{14}$ -labeled ergot alkaloids (1).

Rye was grown in crocks and, prior to flowering, sealed in bell jars of 40-lit capacity. Approximately 160 ml of  $CO_2$ , containing 0.1 mc C<sup>14</sup>, was introduced each day for 9 days. Illumination at an intensity of 300 ft-ca was continuous for 12 days. The plants, after removal, were infected with a culture of *Claviceps purpurea* and maintained in a normal environment until the sclerotia were mature. Fourteen sclerotia, of total weight 192 mg, were obtained.

The defatted sclerotia were extracted and the alkaloids isolated by a column partition chromatographic procedure (2). Further purification was effected by converting the alkaloid to the lactate and shaking with ether to remove nonalkaloid contaminants. The base, generated in aqueous solution, was recovered by shaking with ether-chloroform (2:1) mixture.

The ether-chloroform solution was evaporated on a planchet, and 20 1-min counts were made with a thin mica end-window G-M tube. The residue was redissolved in ether and again counted. The alkaloid was

Table 1. Results of analysis.

Alkaloid	counts/min		Amt. (µg)	count/min mg	
Ergotoxine Ergotamine Ergonovine	$\begin{array}{c} 144.8 \pm 1.9 \\ 334.3 \pm 4.5 \\ 42.1 \pm 2.0 \end{array}$	$\begin{array}{c} 151.8 \pm 3.3 \\ 358.1 \pm 3.7 \\ 50.0 \pm 1.6 \end{array}$	$68.5 \\ 240.4 \\ 6.0$	2115 1390 7016	2216 1489 8333

quantitatively removed, converted to the lactate, and assayed spectrophotometrically using p-dimethylaminobenzaldehyde test solution (U.S.P.). The results of analysis are reported in Table 1. Production of a larger quantity of labeled alkaloids is planned.

Ara G. Paul

ARTHUR E. SCHWARTING

School of Pharmacy,

University of Connecticut, Storrs

#### **References** and Notes

1. Aided by grants from the U.S. Atomic Energy Commission, contract No. AT (30-1) 1666.

2. J. E. Carless, J. Pharm. Pharmacol. 5, 883 (1953).

8 December 1954.

### Diffusion Constant and Diffusion Coefficient

In his treatise, *The Anatomy and Physiology of Capillaries*, Krogh (1, p. 268) defines the diffusion constant as

. . . the number of cc of gas which will in one minute diffuse through an area of 1 cm<sup>2</sup>, when the pressure gradient is one atmosphere per  $\mu$  (0.001 mm).

Krogh uses this definition in his discussion of diffusion within tissues. The term diffusion constant has usually been regarded as analogous to the diffusivity (2), but Krogh's diffusion constant differs from the diffusivity, or diffusion coefficient, in two ways: in defining the gradient it employs the micron instead of the centimeter, and it employs "tension" units instead of concentration units. ("Tension" is partial pressure of diffusing substance in the gas phase at equilibrium between gas and liquid phases.) The first difference is unimportant because it simply introduces a constant factor of  $10^4$ ; but the second is important because it makes the diffusion constant a composite of two variables, the diffusivity and the solubility of diffusing substance in the liquid medium. For example, Krogh lists a value of 0.34 for the diffusion constant of  $O_2$  in water at 20° C. The diffusivity of  $O_2$  in water is 1.607 cm<sup>2</sup>/day (Spoehr, 3), or  $1.12 \times$  $10^{-3}$  cm<sup>2</sup>/min. The solubility of  $O_2$  in water is 0.031 at 20°C. Thus the magnitude of the diffusion constant of Krogh can be computed as  $1.12 \times 10^{-3} \times$  $0.031 \times 10^4 = 0.346.$ 

Obviously this diffusion constant is not an index of diffusivity because it is so importantly influenced by solubility; yet it appears that physiologists have made the error of assuming that it is such an index.

Prosser et al. (4) reproduce Krogh's definition and