devoid of sensory receptors in the dog.

It is concluded from this histological study that the nerve endings in the cat's pulmonary artery are most concentrated at the bifurcation and are also found to a significant extent in the adjacent region of the main pulmonary artery and in initial parts of its right and left branches.

<div style="text-align:right">

M. ANTHONY VERITY
JOHN A. BEVAN

</div>

Departments of Pathology and *Pharmacology, University of California School of Medicine, Los Angeles*

References

1. R. Bianconi and J. H. Green, *Arch. ital. biol.* **97**, 305 (1959).
2. J. C. G. Coleridge and C. Kidd, *J. Physiol. (London)* **150**, 319 (1960).
3. J. C. G. Coleridge, C. Kidd, J. A. Sharp, *ibid.* **156**, 591 (1961).
4. J. A. Bevan, *Pharmacologist* **3**, 64 (1961).
5. O. Larsell and R. S. Dow, *Am. J. Anat.* **52**, 125 (1933); M. Takino and S. Watnabe, *Arch. Kreislaufforsch.* **11**, 18 (1937); J. F. Nonidez, *Am. J. Anat.* **68**, 151 (1941).
6. N. D. Levine, *Stain Technol.* **14**, 29 (1939).
7. De Castro, *Trabajos Lab. invest. biol. Univ. Madrid* **25**, 331 (1928).
8. J. Boss and J. H. Green, *Circulation Research* **4**, 12 (1956).
9. J. A. Bevan, *ibid.*, in press.

7 December 1961

Beryllium and the Growth of Bush Beans

Abstract. Beryllium in nutrient solution inhibited the growth of bush beans. The initial symptom was retarded root development. Although severe stunting of plants occurred, the foliage retained normal color. Roots accumulated beryllium. Increased beryllium concentrations decreased calcium in roots, stems, leaves, and fruits, and also decreased magnesium in roots and stems. Phosphorus was slightly increased in stems, leaves, and fruit.

Because of its unique properties, beryllium is a suitable constituent of power sources for nuclear- and chemical-powered propulsion devices. This element is toxic to animals (*1*). Our interest in Be developed upon considering whether or not its dispersion into the natural environment might also have injurious effects on higher plants. Observations of beneficial and detrimental effects of Be on plant growth processes have been reported (*2*).

Ten-day-old bush bean seedlings (*Phaseolus vulgaris*, "Tendergreen") were transferred from sand culture to aerated, nutrient solutions containing 0, 0.5, 1.0, 2.0, 3.0, or 5.0 ppm Be. The base nutrient solution also contained $2.25 \times 10^{-3}M$ KNO_3, KH_2PO_4, and $MgSO_4$, $1 \times 10^{-3}M$ NH_4NO_3, and

$3.75 \times 10^{-3}M$ $Ca(NO_3)_2$. Micronutrient levels were 0.05 ppm B, 0.01 ppm Mo, 0.5 ppm Mn, 0.05 ppm Zn, 0.02 ppm Cu, and 2.5 ppm Fe(EDDHA). The *p*H of this nutrient solution was buffered at 5.3 ± 0.5. All of the Be treatments had four replicates. Each replicate consisted of two plants grown in 3.6 liters of nutrient solution that was renewed five times during 48 days of plant growth on the Be treatments. At harvest the plants were divided into roots, stems, leaves, and fruit.

An inhibiting effect of Be on the growth of bush beans was evident from the dry weights of the plant parts (Fig. 1). The mean total dry plant weights were 60.2, 40.2, 35.5, 20.6, 14.5, and 7.3 g from the 0, 0.5, 1.0, 2.0, 3.0, and 5.0 ppm Be treatments, respectively. Visual symptoms of Be inhibition first were observed on the roots of bean seedlings transferred to nutrient solution containing 3.0 and 5.0 ppm Be. The roots turned brown within 5 days after Be treatment was started, and the roots failed to resume normal elongation. More than normal numbers of stubby rootlets developed from pudgy, secondary roots. Stunting of plant foliage became apparent within 10 days of exposure to the higher Be treatments; however, the foliage continued to retain natural color at all Be concentrations during the 48-day treatment period. These abnormal symptoms became progressively more severe as the Be concentration in the nutrient solution increased from 0.5 to 5.0 ppm. Earlier flowering occurred for plants grown at the higher Be treatments, which was reflected in the dry weights of fruits (Fig. 1). Although the bush beans grown at 0 and 0.5 ppm Be set more fruit pods, these pods were much less mature when harvested than were the fruit pods produced at the higher Be treatments.

Table 1 shows the concentrations of Be, Ca, Mg, and P in parts of bush beans. Concentrations of Be, Ca, and Mg were measured by emission spectrograph. Phosphorus was determined by the method of Allen (*2*). Beryllium accumulated in the root tissues. Among the aerial parts, the leaves accumulated the highest concentrations of Be; relatively small levels of Be accumulated in the bean fruits. Uptake of Be was linear with respect to the concentration of Be in the nutrient solution. Calcium was decreased in roots, stems, leaves, and fruits as the Be concentration was increased in these plant parts. Increased
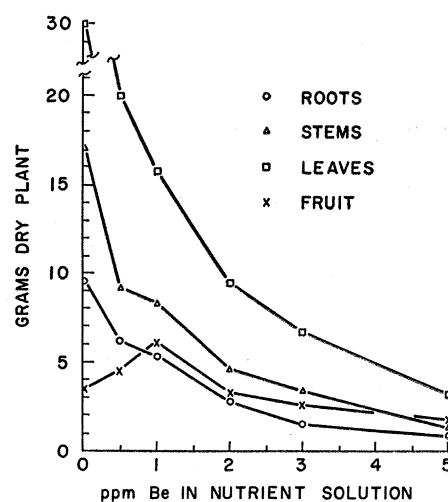

Fig. 1. Effect of increasing concentrations of Be in nutrient solution on the dry weights of parts of bush beans grown 48 days on Be treatments (mean of four replicates).

Be concentrations decreased the Mg concentration in roots and stems but did not alter Mg in leaves and fruits. The concentration of P in roots was reduced at the 5-ppm Be level but was not affected at the lower treatment levels. In other plant parts, the P concentration tended to increase as the Be concentration increased. The percent of total mineral ash in dry plant tissues

Table 1. Concentrations of Be, Ca, Mg, and P in parts of bush beans grown on Be-treated nutrient solution (mean of four replications).

Be in solution (ppm)	Conc. of element in dry plant tissue (mg/g)			
	Be	Ca	Mg	P
Roots				
0	0	5.2	5.6	18.0
0.5	0.271	3.5	6.5	18.4
1.0	0.431	4.5	5.8	20.2
2.0	0.668	4.3	3.6	18.6
3.0	0.978	3.7	2.3	19.4
5.0	1.076	2.6	2.0	13.1
Stems				
0	0	8.5	1.9	6.8
0.5	0.004	6.8	1.9	7.2
1.0	0.006	5.5	2.1	7.2
2.0	0.015	4.0	1.8	9.2
3.0	0.018	3.5	1.7	8.7
5.0	0.024	3.4	1.8	8.4
Leaves				
0	0	22.1	4.2	10.8
0.5	0.008	31.4	6.1	11.1
1.0	0.016	30.1	6.3	10.9
2.0	0.034	19.7	6.7	15.2
3.0	0.042	18.0	6.7	16.0
5.0	0.070	17.9	6.8	15.4
Fruit				
0	0	4.8	2.6	6.3
0.5	0.001	5.1	2.7	6.9
1.0	0.002	4.6	2.6	6.9
2.0	0.004	3.9	2.6	7.8
3.0	0.005	2.8	2.5	8.8
5.0	0.006	2.2	2.7	10.2

was decreased in roots and leaves but was not altered in stems and fruits when the concentration of Be increased in these plant parts.

Beryllium might become injurious to higher plants if high levels were dispersed onto the soil or into ground and irrigation waters where plant roots could come in contact with Be concentrations in excess of 1 ppm in the soil solution. Inhibiting effects of Be on plant growth appear to be centered primarily in the root tissues, and the evidence (3) now indicates that Be may inhibit the normal functions of the plant phosphatase enzyme systems. An inhibition of the normal metabolism of inorganic P which the plant attempts to overcome by increased P uptake might account for the increased P concentrations at the higher Be treatments. Apparently Be does not inhibit the formation and functions of chlorophyll (4).

EVAN M. ROMNEY
JAMES D. CHILDRESS
GEORGE V. ALEXANDER
Laboratory of Nuclear Medicine and Radiation Biology, School of Medicine, University of California, Los Angeles

References and Notes

1. J. Schubert, *Sci. American* **199**, 27 (Aug. 1958); E. D. Hutchinson, R. D. Armstrong, E. A. Maynard, H. C. Hodge, *AEC Biol. and Med. Report TID-4500; Symposium, A.M.A. Arch. Ind. Health* **19**, 9 (1959).
2. R. J. L. Allen, *Biochem. J.* **343**, 858 (1940).
3. C. Lepierre, *Compt. rend.* **156**, 409 (1913); F. Lehr, *Biochem. Z.* **168**, 166 (1926); P. Maze and P. J. Maze, Jr., *Compt. rend. soc. biol.* **132**; 373 (1939); R. A. Steinberg, *Am. J. Botany* **33**, 210 (1946); M. B. Hoagland, *Arch. Biochem. Biophys.* **35**, 249 (1952).
4. These studies are supported by contract AT(04-1)GEN-12 between the Atomic Energy Commission and the University of California at Los Angeles.

16 October 1961

Plasminogen Activity of Plasma and Serum

Abstract. Human plasma and serum were obtained from blood of resting persons. Plasminogen was determined by activation with the optimal amount of streptokinase. As in the majority of cases no significant difference was found between the proteolytic activity of plasma and serum, a blood clot cannot contain more plasminogen than that present in the volume of serum included.

According to current views, a precursor (profibrinolysin, plasminogen) present in plasma reacts with an activator or kinase to produce an enzyme (fibrinolysin, plasmin). The enzyme is inactivated by antifibrinolysins (1). The

Table 1. Comparison of plasminogen activity of human plasma and serum. One volume of inhibitor-free and neutralized plasma or serum, 1 volume of 3-percent casein solution in borate buffer (pH 7.3) and 0.1 volume of 0.154M sodium chloride containing between 430 and 830 units of streptokinase (Varidase) per milliliter of plasma or serum were incubated at 37°C for 30 min. The reaction was stopped by chilling the tubes to 2°C and addition of 2 volumes of 20-percent trichloroacetic acid (wt/vol). After centrifugation the optical density of the solution was determined at 280 mµ. For each reaction mixture a corresponding blank was prepared at 2°C and its optical density was subtracted from that of the incubated mixture. Human serum of known protein content was used as standard. Figures represent averages of trichloroacetic acid-soluble material expressed as milligrams of protein per milliliter of original plasma or serum.

Number of determinations	Plasminogen activity		$B \times 100 \over A$
	Plasma (A)	Serum (B)	
1	2.16	1.96	90.7
14	2.50 (2.16 to 3.27)	2.46 (2.09 to 3.10)	98.4 (94.8 to 99.1)
6	1.73 (1.01 to 2.40)	1.79 (1.04 to 2.45)	103.4 (102 to 105.5)
6	1.78 (1.00 to 2.98)	1.96 (1.07 to 3.20)	110.1 (107 to 115)

concentration of plasminogen in serum will be further reduced by any capacity of the blood clot to adsorb plasminogen. Therefore, the concentration of plasminogen in plasma should be higher than in serum if these conditions prevail.

In order to obtain information about the difference of plasminogen activity in the two fluids, blood specimens were taken at rest 2 hours after a fat-free breakfast from 14 healthy laboratory personnel who are accustomed to handling blood and do not suffer from any apprehension which might enhance fibrinolysis (2). In addition, blood from five patients who were suspected of deficient hemostasis was tested. Some subjects were examined twice.

The first 20 ml of venous blood was placed in two tubes, which were then stoppered and incubated at 37°C for 2 hours. To each milliliter of serum obtained after centrifugation was added 0.02 ml of 1.3M tertiary sodium citrate. A further 20 ml of blood was mixed with 0.2 ml of 1.3M tertiary sodium citrate in tubes chilled to 2°C.

Plasmin antagonists in cell-free plasma and serum were destroyed by adjusting pH to 2.0 and allowing to stand at room temperature for 15 to 30 min (3). After neutralization, plasminogen was activated with optimal amounts of streptokinase (Varidase, Lederle). Proteolytic activity was measured after addition of casein (4). The results are given in Table 1.

It was calculated from 17 duplicate estimations that the results of plasminogen determinations in plasma and serum are not significantly different, at the .05 confidence level, if they lie between 94.6 and 105.7 percent (5). A linear relationship was established between proteolytic activity and concentration of plasma and serum down to 50-percent concentration.

The potential proteolytic activities of plasma and serum were not significantly different (P < .05) in 20 instances. In six instances serum contained a higher plasminogen activity than plasma and in one case serum activity was lower than the plasma activity. No correlation was found between the plasminogen activity and sex or age (17 to 61 years). From these results it is seen that the concentration of plasminogen in serum was in the majority of cases not less than 95 percent of that in plasma and therefore little adsorption of plasminogen had taken place on the blood clot. Plasminogen determinations carried out with normal human plasma before and after defibrination with bovine thrombin (Parke-Davis) gave results similar to those tabulated.

There is justification to assume from experimental evidence that no loss of plasminogen occurred in the chilled plasma during the time of blood collection and determination as a result of plasmin formation due to the possible presence of plasminogen activators. It follows that no fibrinolytic activity was present in the tested specimens.

The tabulated results have a bearing on the treatment of thrombosis by fibrinolytic agents. In mixtures of purified plasminogen and iodinated fibrin (6) it has been observed that some plasminogen is adsorbed by fibrin, and from this finding it has been assumed that fibrinolysis in the circulation results from the activation of plasminogen supposedly present in a thrombus. Also, it has been estimated that approximately 30 percent of the available plasminogen in plasma is adsorbed onto fibrin during clot formation (7).

The data presented, however, indicate that a clot prepared from normal blood taken during resting contains no more plasminogen than is present in the