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7. Evidence is available that absorptive or adsorptive diffusion of IAA into the coleoptile sections is negligible and insufficient to account for the disappearance of IAA from the medium. Exhaustive ether extractions and bioassay of the sections for IAA preclude this possibility. Controls on the medium in which the sections had soaked 12 hr previously also showed negligible IAA destruction by possible microorganism contaminants (L. Anker, thesis, p. 27, Univ. of Utrecht, 1953); L. Reinhold, *The New Phytologist* **53**, 219 (1954).
8. This response, although not obtained from enzyme breis preps., would seem to obviate the findings of Goldacre *et al.* (*9*) that the presence of 2,4-dichlorophenol in samples of 2,4-D is largely, if not solely, responsible for the enhanced inactivation *in vitro*. Further, analysis of the sample of 2,4-D used in the present investigations showed less than $3 \times 10^{-6} M$ DCP, as given by the Folin-Ciocalteau reagent.
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Production of Dissecting Aneurysms in Rats Fed *Lathyrus odoratus*

T. E. Bachhuber and J. J. Lalich

Department of Pathology,
University of Wisconsin Medical School, Madison

A toxic factor in sweet peas (*Lathyrus odoratus*) has been shown to exert pronounced effects on the skeletal development of growing rats (*1–5*). The first to record any alteration in the aorta were Ponseti and Baird (*5*), who described aortic aneurysms. Because of this effect which the *Lathyrus* diet apparently exerts on the integrity of the aorta, it seemed important to study further the factors responsible for the arterial changes.

In the initial phase of our investigation (*6*), extracts of sweet pea meal were assayed for toxicity in rats. The casein concentration in the diets that contained the extracts was either 10 or 20 percent. After a crystalline toxic factor had been isolated by E. D. Schilling and F. M. Strong, it was characterized as B-(γ-L-glutamyl) aminopropiononitrile and subsequently synthesized by these workers (*7*). The assays indicated that the crystalline factor from pea meal and the synthetic compound were just as effective in producing skeletal deformities as was crude pea meal (*8*). However, we observed aneurysms in only three of 60 test rats, a much lower percentage than reported by Ponseti and Baird.

Examination of the diets employed suggested that the casein in the basic ration might have exerted an inhibitory effect on the production of arterial lesions in rats fed preparations that contained the *Lathyrus* factor. It was observed that aneurysms developed only in those animals fed a diet with the casein at a 10-percent level. No aneurysms developed when the casein level was 20 percent. Diets in this study were prepared, therefore, with 10-percent casein.

Seventeen control rats were fed aqueous alcohol-extracted pea meal, and 28 test rats were given crude pea meal. The *test diet* follows: 10 percent Borden's crude casein; 10 percent Pabst Brewer's yeast; 50 percent ground pea meal (*9*); 24 percent cerelose; 4 percent Wesson salt; 2 percent olive oil containing 0.21 mg of vitamin-A acetate, 0.26 IU of vitamin D, 10 mg of a-tocopherol, and 0.15 mg of 2-methyl-1-4 naphthoquinone per kilogram of diet. The control diets were similar in all aspects except that aqueous alcohol-extracted pea meal was substituted for the crude material.

Table 1. Effect of feeding crude pea meal on the development of aortic aneurysms. M, male; F, female.

No. and sex	Days on diet	Cause of death	Starting weight (g)	Autopsy weight (g)
Control: aqueous alcohol-extracted sweet pea meal				
126M	40	Killed	41.0	135.5
127M	36	Killed	41.8	134.5
128F	100	Killed	59.1	162.0
129F	100	Killed	54.1	184.0
145M	91	Killed	41.4	163.4
146M	91	Killed	46.5	209.0
147M	49	Died, cause not established	48.6	
148F	32	Killed	50.4	128.5
149M	61	Killed	42.6	143.3
150F	28	Killed	48.3	125.0
Test: crude sweet pea meal				
122M	35	Ruptured aorta and hemothorax	40.8	105.0
124M	37		39.9	119.0
125M	60	Paralysis and malnutrition	36.0	68.0
130F	62	Ruptured aorta and hemothorax	59.0	135.0
135M	68		60.3	169.4
136M	43		59.4	148.0
140M	27		59.0	125.7
141M	48		47.1	95.0
144F	31		51.7	125.0
154F	48		42.3	75.1

The data in the table show the initial weights, days on the diet, weight gains, and cause of death in 20 of the 45 animals that were studied. Neither bony deformities nor ruptured aortic aneurysms occurred in any of the 17 control rats. In 28 test rats, 14 animals died of aortic rupture and massive hemothorax. Two other animals, which died of malnutrition and upper respiratory infection, also had aortic aneurysms. Three test rats were killed, one of which had an aortic aneurysm. Two others died after developing hernias; two others, following a hemorrhage into the urinary bladder and a fractured leg. Five test rats were placed on commercial pellets and are recuperating from malnutrition.

Practically all of the dissections occurred along the arch of the aorta. All of the test rats developed moderate to marked bony deformities. Partial paralysis of the hind limbs, secondary to deformities of the vertebral column, occurred in less than 20 percent of the animals. Microscopic examination of formalin-fixed tissues revealed degeneration and fragmentation of the elastic connective tissue fibers. In addition, there

were medial edema and hemorrhagic dissection of the media in every instance when rupture occurred. In some cases, minimal to marked focal intimal thickening due to fibroblastic proliferation was also evident.

It has been possible to confirm the observation that *Lathyrus* factor will promote the production of aortic aneurysms. When casein was fed at a 10-percent level with the *Lathyrus* factor, aneurysms developed in 17 of 28 rats.

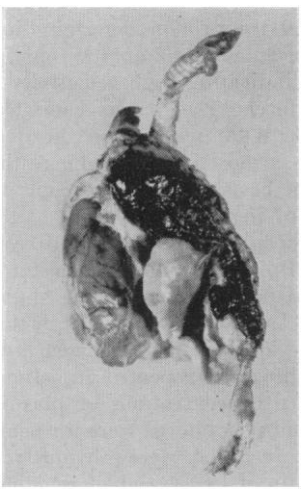

Fig. 1. Rat 135 died of aortic rupture after 68 days on crude pea meal. Left lateral view of heart and lungs with trachea at the top (×1.5). The massive periaortic hemorrhage extends from the arch to the thoracic aorta.

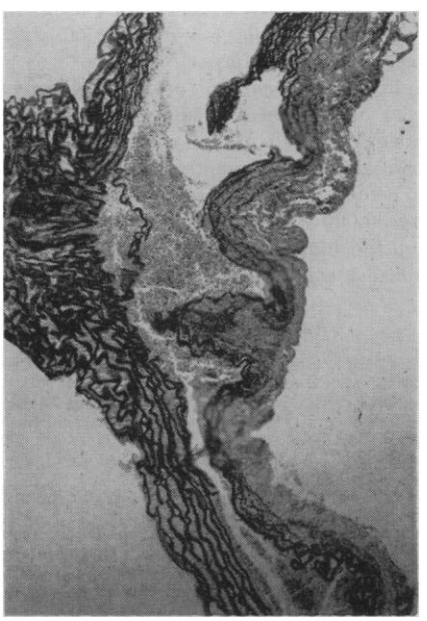

Fig. 2. Rat 136 died of aortic rupture after 43 days on the test diet. Weigert's elastic tissue stain of the aortic arch (×75). The right-hand border illustrates the poorly developed adventitia which contains minimal fat. Note the separation of elastic fibers and the dissecting medical hemorrhage.

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Relationship between Blood Lipids and Radiation Injury in Rabbits

Lawrence J. Milch, Richard A. Yarnell, James V. Stinson, and the Cardiovascular Research Group*

Department of Pharmacology and Biochemistry, USAF School of Aviation Medicine, Randolph Field, Texas

Hewitt (*1*) and his associates reported that the lipoproteins of rabbit serum were markedly increased after exposure to 800 to 900 r of total body x-irradiation and that the degree of hyperlipoproteinemia was related to the survival of the animal. Entenman, Neve, and Olmstead (*2*) noted increased blood phospholipid levels in several species after whole body x-irradiation. It was reported from this laboratory that 24 hr after local exposure of the right hind limb of an otherwise completely shielded rabbit to 30,000 r of x-irradiation, a pronounced hyperlipoproteinemia and hyperlipemia can be observed. The blood lipid changes are accompanied by an increased cholesterol concentration in the irradiated muscle tissue and a significant change in the electrophoretic characteristics of the soluble rabbit muscle protein (*3*). The total data suggest that the extent of blood lipid alteration may be some direct function of the degree of radiation injury in this species. It was decided, therefore, to subject separate groups of rabbits to varying doses of local x-irradiation up to 30,000 r and determine the plasma lipid and lipoprotein concentration at each radiation level (*4*).

Ninety-eight male albino rabbits of the New Zealand strain, each weighing approximately 2 kg, were divided equally into seven groups designated A through G. Each of the rabbits in groups B, C, D, E, F, and G were subjected to local x-irradiation of the distal portion of the right hind limb in dosages of 5000, 10,000, 15,000, 20,000, 25,000 and 30,000 r, respectively, at a rate of 1000 r/min, the animals in group A serving as controls. The irradiation equipment and techniques employed were exactly as have been previously described (*3*). Immediately following exposure the rabbits were weighed, placed in individual cages in an air-conditioned animal room, and maintained on Purina rabbit chow and water, *ad*