hibit the reaction, show a close chemical relation to the substances which are autoxidizable, (*e. g.,* compare *p*-aminoacetanilide with methyl *p*-phenylenediamine) it is not improbable that the latter compounds may also have an inhibitory effect which is masked by their own color reactions.

The tendency of these autoxidizable substances to form colored compounds and finally a black precipitate may be due to a reaction similar to the formation of quinhydrone from quinone and hydroquinone and of phenoquinone from phenol and quinone as described by Karrer.[4] The question arises whether or not the formation of melanin could arise in a similar manner. Raper[5] reports that 3,4-dioxyphenyl alanine is formed upon the oxidation of tyrosine by tyrosinase and that after the oxidation the enzyme can be removed and melanin is still formed. Since the intermediate compound has a quinoid grouping, it seems possible that it might react to form melanin as indicated above.

Summary. The effect of certain split products of some carcinogenic azo dyes and related compounds on melanin formation was studied. It was observed that certain non-autoxidizable compounds such as *p*-aminoacetanilide, *B*-naphthylamine, *p*-aminobenzoic acid and sulfanilamide interfered with this reaction. When chemically related autoxidizable compounds (*p*-aminodimethylaniline, *p*-phenylenediamine, methyl *p*-phenylenediamine and *p*-aminophenol) were used, the color changes in both tyrosine-tyrosinase and control solutions were similar. It is, therefore, difficult to interpret the action of these latter chemicals on melanin formation.

13800 P

Effect of Low Calcium and Vitamin D-Deficient Diet on Bones and Teeth of Mature Rats.*

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Twenty male albino rats which had been raised to an age of 11 months on an adequate stock diet were fed for 5 weeks on a control

[4] Karrer, Paul, *Organic Chemistry,* New York, 1938.

[5] Raper, H. S., *Biochemical J.,* 1927, **21**, 89.

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diet containing 0.462% calcium, 0.458% phosphorus and adequate in vitamin D. Ten of the animals were then transferred to the experimental diet which was deficient in vitamin D and contained 0.007% calcium and 0.450% phosphorus. Calcium and phosphorus balances were determined for each animal over 10-day periods for 220 days. The animals of the control group remained in positive calcium and phosphorus balance throughout the period of observation. The animals fed the deficient diet exhibited a severely negative calcium balance and a positive phosphorus balance.

The humeri and femora and the molar and incisor teeth were dissected free of soft tissue, cleaned, dried and rendered fat-free by continuous extraction with fat solvents. During the removal of the molar teeth from the jaws, it was noted, in the case of experimental rats, that the alveolar bone was soft and friable, the alveolar crest resorbed and the teeth loosened. In the control animals the alveolar bone was well calcified, the crest normal and the teeth firmly fixed in their sockets. The incisor teeth of both groups appeared equally developed and formed. The average weight of the dry fat-free incisor teeth of the two groups was not significantly different. The average percentage of ash in the left femora was not significantly different between the two groups of animals.

The volume and density of the left humerus of each animal was determined, using a specially designed pycnometer and mercury as the suspension medium. The average volume of the humeri of the experimental animals did not differ from that of the controls but the average density of the bones of the former animals was 10.8% less than that of the controls. The calcium and phosphorus contents of the left humeri, in terms of percent of the dry fat-free weight, were not significantly different between the two groups of animals. However, the calcium and phosphorus contents of these bones in terms of unit volume of bone appeared to be significantly lower in the experimental animals than in the animals fed the adequate diet (Table I). These results indicate that determinations reported in terms of weight percent do not serve to distinguish osteoporitic from normal bone, but that the degree of osteoporosis can be described in terms of ash weight or mineral content related to unit volume of bone. These results also indicate that mineral and organic phases are proportionally decreased in the osteoporitic bone.

The enamel and dentin of the incisor and molar teeth were separated, using the method of Manly and Hodge.[1] Calcium and phosphorus analyses made on the dentin indicated that no mineral had

[1] Manly, R. S., and Hodge, H. C., *J. Dent. Res.*, 1939, **18**, 133.

TABLE I.
Calcium and Phosphorus Contents of Dentin and Humeri of Rats.

	Molar teeth dentin		Incisor teeth dentin		Humeri	
	Ca %	P %	Ca %	P %	Ca mg/cc	P mg/cc
Controls	30.24 (±0.60)	13.79 (±0.79)	26.35 (±1.08)	14.84 (±0.61)	301.9 (±37)	139.5 (±15)
Experimentals	30.23 (±0.93)	13.79 (±0.33)	25.51 (±0.84)	15.18 (±0.27)	250.9 (±17)	115.6 (±13)

been lost by the molar teeth of the experimental animals. The composition of the dentin of the incisor teeth, which had been completely reformed several times during the period of observation, was not significantly different between the two groups of animals. The numbers enclosed within parenthesis in Table I are the standard deviations of the respective means.

13801

Functional Reorganization Following Preganglionectomy.

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In the human, preganglionic sympathetic outflow affecting the hand is usually over the ventral roots of thoracic nerves 2 through 9.[1] Preganglionic denervation of the upper extremity in cases of Raynaud's disease has been performed frequently by sectioning the 2nd and 3rd spinal nerves, and cutting the sympathetic trunk between the 3rd and 4th thoracic ganglia, leaving T-1 intact to avoid Horner's syndrome.

Evidence has been obtained that in these cases nervous control of vasoconstriction often returns within a relatively short time.[2] This return has been ascribed to regeneration of the preganglionic fibers.

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1 Ray, B. S., Hinsey, J. C., Hare, Kendrick, and Geohegan, W. A., *Trans. Am. Neurol. Soc.*, 1942.

2 Simmons, H. T., and Sheehan, Donal, *Brit. J. Surg.*, 1939, **27**, 234.