sia ($P < .001$) and a significant polyuria ($P < .001$) compared with the polydipsia and polyuria of the nontreated controls or of the irradiated, Pitressin-injected animals. The urinary output values of the irradiated Pitressin-injected animals were not significantly different from the outputs of the nontreated control animals, but the water intakes were significantly higher than the intakes of the nontreated controls ($.02 < P < .05$). Water intakes and urine outputs within experimental groups were not significantly different in the case of the nontreated controls or the irradiated peanut-oil injected animals. However, water intakes were significantly greater than urine outputs ($P < .001$) in the irradiated, Pitressin-injected group.

These results show that postirradiation polyuria is not due to an obligatory renal tubular water loss, but they do not offer proof for any of the postulated mechanisms. If the phenomenon is the result of an imbalance between adrenal cortical diuretic and posterior pituitary antidiuretic factors as proposed by Smith and Tyree (*2*), the evidence in this paper would suggest that the imbalance can be corrected by the injection of Pitressin.

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Quantitative Roentgenography of Skeletal Mineralization in Malnourished Quechua Indian Boys

Roentgenograms of the hand suitable for densitometric analysis as well as assessment of skeletal age were taken on 91 Quechua Indian boys from Hacienda Vicos in the North Central Peruvian Sierra. These boys were from a biologically isolated inbreeding population of over 1800 Indians almost wholly unmixed with Europeans (*1*). This Indian population lives in a narrow upland valley 10,000 to 12,000 feet in altitude, off the Callejon de Huaylas 250 miles north of Lima. Until 1957 the Vicos Indians were economically depressed serfs exploited by a long series of absentee landlords, but then they were freed by government proclamation.

The principal Vicos food crops are corn and potatoes; intake of animal proteins is negligible. Dietary surveys in 1952 and 1953 (*2*) and a check study in 1956, when the roentgenograms were taken, revealed that the average daily intake of vitamins A and B_2 and calcium were respectively 3, 56, and 14 percent of adjusted INCAP recommended allowances (*3*). Preliminary studies suggest that Vicos soils are calcium-deficient. Calorie and protein intake were about 70 percent of the recommendations, and fats represented only 7 percent of the daily caloric intake. Superposed on this inadequate nutrition was a heavy infestation of intestinal parasites, particularly *Ascaris* (*4*).

The posteroanterior roentgenograms of the left hand were taken by Humberto Mesones, through the courtesy of Ramon Vallenas, subdirector of Peru's Department of Industrial Hygiene. The roentgenographic technique, the developing procedure, and the photodensitometric apparatus used in the analysis have been described elsewhere (*5*) and need only brief mention. Eight- by ten-inch No-Screen film in cardboard holders was used, and an aluminum-zinc alloy film calibration wedge was simultaneously exposed with the subject's hand. The x-ray apparatus was a mobile Phillips 15 M A, manufactured in the Netherlands. Electric current was provided by a 220-v 60-cy/sec gasoline generator with a 3500-watt output. Exposure time was lengthened slightly to compensate for drawdown. The developed film was placed in the photodensitometric apparatus, and the middle phalanx of the fifth finger was evaluated for bone density coefficients at three sites or trace paths: the proximal and distal ends, whose values were averaged, and the centers. In this analysis the bone density coefficient is the number of grams of the aluminum-zinc alloy which, in a similar geometric shape, would absorb the same amount of x-radiation as the bone, divided by the volume of bone in cubic centimeters.

The Quechua Indian means, grouped by age (Table 1), stand in decided contrast to a series of normal white boys drawn from various parts of the United States (*6*). The means for the 7–9- and 10–12-year-olds of both Indians and whites are similar, but then the 13–15- and 16–20-year-olds from the United States show increases statistically significant at the .001 level, or better, for both the phalanx end and center trace paths. No such increase is present in the Indian boys in the age groups studied. The mean differences between Indians and whites in the 13–15- and 16–20-year groups are also statistically significant at the .001 level for each trace path. Accordingly, bone mineralization shows steady mean increases through the teens in United States whites but none at all in the Indians.

Table 1. Mean density coefficients of the phalanx center and phalanx end trace paths of Vicos Indian boys and United States white boys.

Age group	Cases (No.)	Density coefficient (mean ± standard deviation)	
		Phalanx center	Phalanx end
	91 Vicos boys		
7–9	18	0.98 ± 0.30	0.59 ± 0.16
10–12	29	1.06 ± 0.23	0.65 ± 0.15
13–15	31	1.02 ± 0.21	0.64 ± 0.14
16–20	8	0.99 ± 0.40	0.66 ± 0.25
	624 United States white boys		
7–9	77	1.02 ± 0.23	0.64 ± 0.11
10–12	101	1.03 ± 0.28	0.66 ± 0.16
13–15	225	1.32 ± 0.30	0.89 ± 0.21
16–20	221	1.43 ± 0.24	1.03 ± 0.19

The lack of increased bone density through the late teens in the Vicos Indians is probably due to dietary inadequacies. This view is supported by Williams and associates (*7*), who observed that bone-density measurements reflected the nutritional history of an individual. In addition, radical decreases in dietary calcium in rats depress bone-density values of their femurs (*8*) and caudal vertebrae (*7*). While this suggests that the depressed bone density coefficients of the Vicos Indians may be a result of their low calcium intake, the marked deficiencies in other nutrients and the extreme parasite infestation must also be considered. At Vicos, augmented bone mineralization, indicated by higher density values, may occur in the early 20's although we have no roentgenograms of this age group. The higher standard deviations in the numerically inadequate 16–20-year-old group may presage such an increase in bone mineralization. Indeed, the chewing of coca by almost all the Vicos Indian men (but never by the women) may result in increased calcium ingestion, since release of the alkaloid in coca is accomplished by mixing *cal* with it. *Cal* is thought to contain considerable quantities of calcium and is the Spanish word for lime.

The depressed bone mineralization in the Vicos boys is paralleled by an average lag of several years in skeletal ma-

turation (*9*) when assessments of the same roentgenograms are compared with Greulich-Pyle (*10*) standards. This is the greatest retardation in skeletal maturation reported for a number of preadult Peruvian series (*11*). Furthermore, the Vicos Indian boys show a mean deceleration of growth from about 10 to 15 years of age compared with other series from the Peruvian Sierra. After 15 years of age, the Vicos boys show a belated growth spurt that may relate to puberty. The slow and retarded growth, maturation, and phalangeal mineralization of the Vicos boys and the ultimately small body size of the adult men (*12*) probably reflect body economy in utilizing the meager amount of available calcium, as well as an inadequate total nutritional environment.

Clearly, the photodensitometric analysis of bone mineralization fits in closely with the growth and maturational data and provides revealing information on the relationship of food to physique. After several years on a diet raised to adequate levels, the Vicos Indian boys will be restudied with respect to any alterations in their developmental patterns (*13*).

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Increased Ascorbic Oxidase Activity Induced by the Fungal Toxin, Victorin

The toxin theory of disease has been an intriguing concept ever since the discovery of diphtheria toxin in 1888, by Emile Roux and Alexandre Yersin. In the field of animal pathology, the theory has led to brilliant advances in the understanding and control of disease. The application of this concept to the field of plant pathology, although admittedly less fruitful, has in the last decade yielded important results.

The studies of Braun (*1*), Dimond and Waggoner (*2*), Gäumann (*3*), and Gottlieb (*4*), to mention a few, clearly demonstrate that plant pathogens are capable of producing metabolites in culture which, when they are applied to the host, produce injurious effects in some cases similar to those encountered in natural infection. However, proof that plant pathogens produce toxins which are directly and solely responsible for the symptoms found in infected plants is still lacking.

Victorin, the toxin produced by the fungus *Helminthosporium victoriae* M. and M., first described by Meehan and Murphy (*5*), offers a unique tool for investigation of the toxin theory in relation to plant disease. These workers, as well as Litzenburger (*6*) and Luke and Wheeler (*7*), observed that, unlike other phytotoxins which generally lack the host specificity exhibited by the pathogen involved, victorin affected only those varieties of oats that were susceptible to the fungus (hybrids derived from victoria). Luke and Wheeler (*7*) also demonstrated that high yields of toxin could be obtained from highly pathogenic isolates of the fungus, whereas nonpathogenic strains failed to produce toxin.

Studies by Romanko (*8*) disclosed that victorin caused three- to five-fold increases in respiration of oat tissues of varieties susceptible to *H. victoriae* but that it failed to produce any appreciable effect on the respiration of Camellia, a resistant variety. This was apparently the first report of a toxin's producing such an effect only in plants susceptible to the pathogen. Further studies carried out by me (*9*) have shown that this increase in respiration is directly proportional to the concentration of toxin applied (original culture filtrate, containing 1000 units per milliliter as measured by the method of Luke and Wheeler, diluted 2.5×10^{-2}) (Fig. 1).

When cuttings were placed in toxin for 4 hours and exposed to light, and the enzymatic activity was measured 12 hours later, ascorbic oxidase, the major terminal oxidase system found to be in operation in susceptible oats, was found to be four times as high as that in comparable tissues (Fig. 2). The possible reasons for this increase in enzyme activity are at present not known, but in view of Newcomb's (*10*) findings that auxin (indoleacetic acid) greatly increases ascorbic oxidase activity in tobacco pith cells grown in culture, the results obtained appear to be significant. Tests with oats of resistant varieties showed that treatment with the same concentration of victorin failed to produce any detectable effect on ascorbic oxidase activity.

Romanko's studies (*8*) suggested that the increase in respiration of susceptible tissues caused by victorin may be due to uncoupling of phosphorylation from oxidation. Studies with 2,4-dinitrophenol (DNP), a proven respiratory uncoupler,

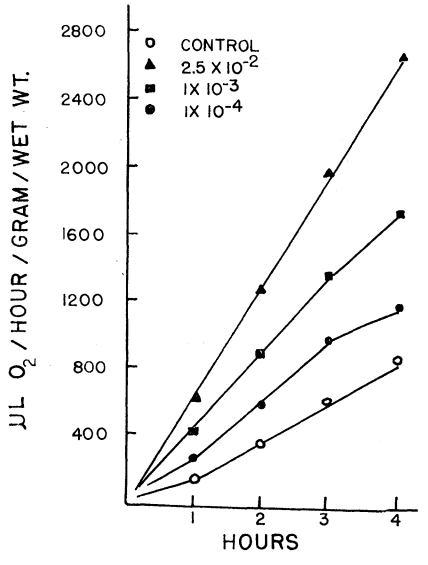

Fig. 1. Increase in respiration of susceptible oat tissue as a function of toxin concentration.

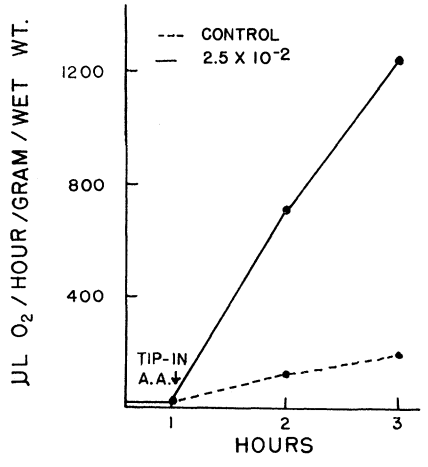

Fig. 2. Increase in ascorbic acid oxidase activity of homogenates from susceptible oat plants treated with victoria.