Concentrations of Radioactive Materials in the Air during 1957

The present concern over the possible hazards associated with the introduction of man-made radioactive materials into the air has been occasioned, in part, by a lack of awareness of the levels of activity of the naturally occurring radioactive materials in the air. It is thought appropriate at this time to present the most recent information obtained from a continuing study conducted by the U.S. Naval Research Laboratory on the radioactivity of the air.

The concentration of radon, thoron, and fission products in the air is obtained from the changes in the rate of decay, over a 16-hour period, of radioactive particulate matter collected on efficient filters (with 98-percent retention of particles as small as 0.3 μ in diameter) through which 900 to 1300 m^3 of air have passed during the previous 24 hours (1). Measurements were made daily on identical equipment located at Washington, D.C.; Yokosuka, Japan; Kodiak, Alaska; and Little America, Antarctica. Calibrated radioactivity standards were counted daily in each unit. The average of the daily measurements covering the full year of 1957, with the exception of short periods when the equipment was undergoing repair, is presented in Table 1. The radon concentration is that occurring in the early afternoon at each site and generally represents the minimum concentration during the 24-hour collection period.

As may be seen, the bulk of the radioactivity is due to the ever-present radon and its decay products, which result from radioactive decay of radium in the soil and the consequent diffusion of the gaseous radon daughter into the air. The concentration of radon and thoron in the air is thus dependent on the location of land masses relative to the prevailing winds passing over the collecting site (*1*). In spite of the fact that the concentration of air-borne fission products in the Washington area was unusually high during 1957, due to the extensive nuclear test series in Nevada, this man-made material amounted to only 1.2 percent of the radon concentration. The other collection sites show similar values for the fission-product–radon ratios. The concentration of thoron is roughly equal to that of the fission products in every case. When one takes into account the series of radioactive products associated with each radon decay, the additional external dose due to the fission products in the air is found to be inconsequential.

Table 1. Geographical distribution of atmospheric radioactivity during 1957 (activity in micromicrocuries per cubic meter).

	Radon	Thoron	Fission Products
Washington, D.C.	172	2.3	2.1
Yokosuka, Japan	54	0.48	0.66
Kodiak, Alaska	7.3	0.042	0.16
Little America, Antarctica	1.5	0.01	0.019

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Reference

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On the Effect of Inorganic Phosphate on Hexose Phosphate Metabolism

In 1935 Theorell (*1*) showed that inorganic phosphate (P_i) (*2*) inhibited glucose-6-phosphate dehydrogenase, the enzyme catalyzing the first reaction of the hexose monophosphate shunt pathway. It is also well known that P_i plays an essential role in the Embden-Meyerhof glycolytic scheme. These two observations suggest the possibility that the local intracellular P_i level could determine the pathway of glucose metabolism. A high P_i concentration should inhibit the shunt but allow glycolysis to proceed, whereas a limiting P_i concentration should exert the opposite effects. This report provides experimental evidence that the P_i concentration in extracts of ascites tumor cells does produce this effect on hexose phosphate metabolism. The reactions involved are shown diagrammatically in Fig. 1.

Ehrlich mouse ascites tumor cells were removed from the animal, washed two times with 0.1*M* Tris buffer, *p*H 7.4, and then homogenized in 0.01*M* Tris buffer, *p*H 7.4, for 20 seconds with micro glass beads in a Nossal shaker (*3*). The cell debris and mitochondria were removed by centrifugation. The temperature during the preparation of this enzyme extract was maintained at 4°C.

In one series of experiments, 0.2 ml of this extract with added TPN^+ was incubated in the presence or absence of phosphate buffer with (i) G-6-P and (ii) glucose-1-C^{14}. At the end of the incubation period the reaction was stopped by placing the vessels in a boiling water bath or by adding an equivalent volume of 10 percent trichloracetic acid. The G-6-P that remained was determined by treatment of the deproteinized extract with 100-fold purified glucose-6-phosphate dehydrogenase (*4*) and excess TPN^+. In the experiments with glucose-1-C^{14}, the $C^{14}O_2$ produced was collected in KOH in the center well of Warburg vessels and counted as $BaC^{14}O_3$ at infinite thickness. The amount of hexose that was metabolized by way of the shunt pathway was calculated either from the TPN^+ reduction (*5*) or from the $C^{14}O_2$ production. Pentose phosphate formation was not used as a means of estimating the proportion of G-6-P metabolized by the shunt pathway because pentose intermediates are utilized by these enzyme extracts.

A typical experiment is described in the legend of Fig. 2. At the end of a 20-minute incubation period the disappearance of G-6-P was complete in the presence of added TPN^+ whether phosphate was present or not. However, as can be seen on the basis of the increase in absorption at 340 mμ (Fig. 2), the percentage of G-6-P metabolized by way of the shunt in the presence of 0.05*M* phosphate was only 55 percent of that in the presence of Tris. Also recorded in Fig. 2 are the parallel results obtained on the basis of $C^{14}O_2$ production from glucose-1-C^{14}.

A second series of experiments was carried out in a glycolysis medium (*6*) in the presence of either 0.02*M* Tris or 0.02*M* potassium phosphate buffer. Oxidized glutathione was added as an electron acceptor for TPNH (*7*). Under these conditions it was not necessary to add TPN^+ to the incubation mixtures and there was an 85 percent inhibition of $C^{14}O_2$ formation from 0.5 μmole of glucose-1-C^{14} in the phosphate buffer. Lactate was formed in both the Tris and phosphate buffers but was radioactive only in the latter. With glucose-6-C^{14} the lactate was radioactive in both buffers and no $C^{14}O_2$ was formed. These results are in agreement with the view that the glucose is being degraded through both the glycolytic and shunt pathways.

The relative contributions of these two pathways of glucose metabolism in various tissues have been the subject of numerous publications (*8*). Certainly the availability of TPN^+ in the cell may limit glucose oxidation through the

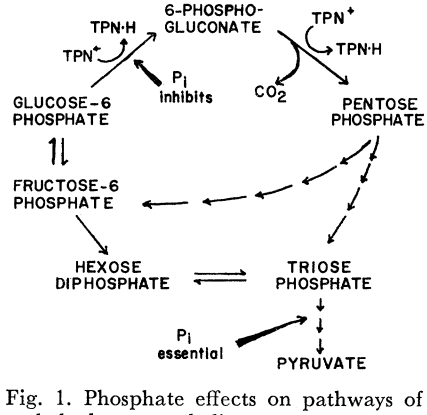

Fig. 1. Phosphate effects on pathways of carbohydrate metabolism.