EFFECT OF HYPOXIA ON THE RATE OF ABSORPTION AND INCORPORATION OF GLUCOSE-C^{14} INTO ORGANS AND TISSUES

K. V. Smirnov and O. I. Babkina

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Acute hypoxia increases the rate of glucose absorption in the alimentary tract.

The effect of hypoxia on gastrointestinal functions has been investigated by Razenkov [5] and his pupils [2-4, 6], who studied changes in the secretory activity of the salivary, gastric, and intestinal glands, the pancreas, and liver, and also changes in intestinal movements. However, only isolated studies have so far been made of the absorptive function of the intestine during hypoxia [1, 7, 8, 10].

The object of the present investigation was to study the rate of absorption of glucose 1-6C^{14} and the distribution of C^{14} among organs and tissues in a normal atmosphere and after exposure to acute hypoxia.

EXPERIMENTAL METHOD

Experiments were carried out on male albino rats weighing 180-220 g. The animals received their last meal 18-20 h before the experiment. Hypoxia was produced in a 35-liter pressure chamber in which the air temperature was 21 ± 0.5° and relative humidity 70-90%. The animals were "raised" at the rate of 2 m/sec to an altitude of 12,000 m, at which they remained for 15-20 min before being brought back to "ground level" within 30-40 sec. Immediately after the descent, the rats received 2 ml of an isotonic solution (5.6%) of C^{14}-labeled glucose, with an activity of 25 μCi, by gastric tube. After a known time interval (0.5, 1, 3, 24, and 48 h) the animals were decapitated. The liver, lungs, kidneys, spleen, brain, and muscles were removed and weighed and their radioactivity was determined. Pieces of gastrointestinal tract (stomach and small intestine) were ligated at both ends and their weight was determined with and without contents. The contents and the walls of different parts of the gastrointestinal tract were then subjected to radiometric analysis. Radioactivity of the blood, urine, and feces also was determined. Measurements were made in thick specimens by means of an end-window T-25-BFL counter with a B-2 apparatus.

To obtain comparable values, the ratio between the discovered activity per gram tissue tested and the activity injected per gram body weight was calculated in percent. Experiments were carried out on 124 animals.

EXPERIMENTAL RESULTS

The concentrations of glucose 1-6C^{14} in the gastric contents of the rats after exposure to acute hypoxia for 30 min (16.81 ± 0.97%), 1 h (9.12 ± 1.46%), and 3 h (7.56 ± 0.29%) were higher ($P < 0.001$) than in the control animals (11.90±1.29%, 3.28±0.50%, and 0.16±0.01% respectively). The glucose concentration in the small intestine was lower ($P < 0.001$) only 30 min after its administration (1.92 ± 0.12% compared with the control 3.20 ± 0.30%); at all other times no significant difference was observed. Transient inhibition of the evacuation of glucose from the stomach could be attributed to vagopyloric spasm [9].

Investigations of the incorporation of glucose-C^{14} into the organs and tissues showed that, although evacuation of glucose from the stomach into the duodenum after exposure to acute hypoxia was still con-

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siderably retarded (30 min), the radioactivity of the blood during the first 30 min (72.65 ±3.27%), and subsequently after 3 h (40.61 ± 1.51%), 24 h (34.18 ± 1.87%), and 48 h (23.44 ± 0.91%) it was significantly higher ($P < 0.001$) than in the control animals (58.31 ± 2.02%; 33.47 ± 0.99%, 18.5 ± 1.18% and 14.29 ± 0.55% respectively). This can be explained both by an increase in the rate of absorption after exposure to hypoxia and by a decrease in the utilization of C^{14} from the blood by the organs. In these experiments the radioactivity of the internal organs of animals kept in hypoxia was higher or, at least it was not lower, than in the control rats. These results show that after exposure to acute hypoxia the rate of absorption of glucose from the gastrointestinal tract is increased.

Activity of C^{14} per gram liver in animals exposed to acute hypoxia for 30 min (8.94 ± 0.21%) and 1 h (11.03 ± 0.53%) was twice as high, and in those exposed for 3 h (8.97 ± 0.23%) and 24 h (4.39 ± 0.15%) it was three times higher than the radioactivity of the liver of the control animals (4.00 ± 0.21%, 4.67 ± 0.22%, 2.68 ± 0.06%, and 1.25 ± 0.07% respectively). It was not until 48 h after administration that the C^{14} content in the liver in both groups of animals showed a decrease, to 1.56 ± 0.06% after hypoxia and to 1.27 ± 0.08% in the control animals. The differences observed after 48 h were at a lower level of significance ($P < 0.02$).

The increase in the content of glucose-C^{14} in the brain 30 min (0.67 ± 0.03%), 1 h (0.95 ± 0.02%), 3 h (0.78 ± 0.02%), and 24 h (0.44 ± 0.06%) after acute hypoxia compared with the control (0.52 ± 0.04%, 0.71 ± 0.03%, 0.54 ± 0.02%, and 0.24 ± 0.01% respectively) was evidently associated with an increase in the blood supply to the brain under those conditions. However, by 48 h this increase was no longer statistically significant ($P > 0.5$).

The specific radioactivity of the lungs, kidneys, spleen, and muscles was indistinguishable from the control. Comparison of the radioactivity of the walls of the stomach and small intestine in the control and experimental animals showed significant differences only during the first 30 min after adminstration of glucose 1-6C^{14}. Subsequently the incorporation of C^{14} into the walls of the stomach and small intestine of the experimental rats was significantly ($P < 0.001$) higher than in the controls. This difference was particularly marked after 3 h (1.47 ± 0.07% in the experimental animals, 0.44 ± 0.03% in the controls) probably because of delay in the onward movement of the injected glucose and the fact that absorption began in the stomach itself. Normal values were not restored even after 48 h, and the radioactivity of the walls of the stomach and small intestine in the experimental rats (0.25 ± 0.01% and 0.57 ± 0.02%) was significantly higher ($P < 0.001$) than in the controls (0.14 ± 0.01% and 0.18 ± 0.01%).

Under normal conditions, excretion of glucose-C^{14} in the urine and feces was observed 30 min after injection of the glucose (specific radioactivity of the urine after 30 min 24.53 ± 4.47%, after 1 h 64.27 ± 6.52%; in the feces after 30 min 5.03 ± 0.41%, and after 1 h 9.38 ± 1.37%). The elimination of C^{14} with the feces was increased after 3 h (specific activity 309.82 ± 21.09%) to a much greater degree than in the urine (specific activity 75.33 ± 10.43%); subsequently although the elimination of isotope diminished, most of it was excreted with the feces.

Under the influence of hypoxia, the excretion of isotope in the urine was reduced (normal value 64.27 ± 6.52%) 1 h after injection of labeled glucose (29.31 ± 5.39%, $P < 0.001$), and its excretion in the feces was increased after 30 min (29.00 ± 2.89%) and after 1 h (23.22 ± 2.82%) compared with the control (5.03 ± 0.41% and 9.38 ± 1.37% respectively). Maximum excretion of C^{14} with the feces in the experimental animals, because of retention of glucose in the stomach, was delayed from 3 h until 24 h (387.11 ± 17.65%), and its level still remained high even after 48 h (250.31 ± 6.85%, $P < 0.001$); the normal value is 202.02 ± 10.72%.

The results of these investigations thus show that the rate of absorption of glucose in the alimentary tract is increased in animals exposed to acute hypoxia.

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