results upon regenerating worms confirm those of other workers. Oxygen consumption remains above normal during regeneration. Oxygen consumption of both normal and regenerating worms is markedly decreased by heavy exposures with X-rays. The profound effect of this treatment on the oxygen consumption may be seen from Table II.

TABLE II.

Oxygen Consumption (cc. per hr.) of Individual Planarians Before and After X-ray Treatment.

Animals	Normals before irradiation	24 hrs. after irradiation
A	0.01472	0.00114
B	0.00924	0.00027
C	0.00876	0.00110
D	0.00559	0.00037
E	0.01186	0.00570

Determinations by a micro-modification of the Winkler method.

In view of the stimulating action of glutathione upon cell division and the high concentration of this compound in embryonic cells, the destruction of glutathione by X-rays or at least its inactivation offers an explanation of the selective action of X-rays upon cells undergoing mitotic division and upon embryonic cells of planarians and other forms.

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Action of Adrenalin on the Metabolism of Peripheral Tissues.

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It has been found impossible to affect the carbon dioxide or total acid production of excised frog muscles by placing them in adrenalin solutions.[1] The absence of a calorigenic effect[2] under such unphysiological conditions was not surprising; and more recent work has indicated that the absence of the viscera[3] and particularly of the liver[4] may have been as responsible for the negative result as the

[1] Griffith, *Am. J. Physiol.*, 1923, lxv, 15.

[2] Boothby and Sandiford, *Am. J. Physiol.*, 1923, lxvi, 93.

[3] Soskin, *Am. J. Physiol.*, 1927, lxxxiii, 162.

[4] Casky, *Am. J. Physiol.*, 1927, lxxx, 381.

lack of proper circulation or other abnormal conditions to which the muscles themselves were immediately exposed.

It is not known in what manner the viscera cooperate in the calorigenic action of adrenalin; whether by serving as the locus of its action, or by keeping the peripheral tissues in the proper physiological condition to respond to the adrenalin itself. The following experiments were undertaken to determine some of the effects that might be produced in one of the hind legs of an anesthetized cat upon the addition of a known amount of adrenalin directly to its arterial blood supply.

All experiments were done on cats under chloralose anesthesia. The animal was always prepared so that simultaneous samples could be taken of the arterial blood going to and venous blood coming from the left hind leg. At the time of collection of the venous sample the rate of flow was also determined in a manner somewhat similar to that described by Himwich and Castle.[5] The usual precautions were taken to prevent alteration in the gas content of the blood; and this was determined by the manometric method of Van Slyke and Neill, using 0.2 cc. samples in duplicate.

In large cats that could stand the additional loss of blood, 2 normal sets of samples, 10 or 15 minutes apart, were taken before the adrenalin injection; these served to establish the average spontaneous variation to be expected under the conditions of these experiments. In the remaining experiments the adrenalin was injected immediately after taking a single normal pair of samples.

The adrenalin was made up in 0.9% NaCl or mammalian Ringer from 1:1,000 Parke-Davis adrenalin chloride solution, neutralized and warmed before injection; and this was made directly into the iliac artery so that the adrenalin acted first, and we believe, in most cases, only on the tissues of the leg under observation.

Results: As controls we have 18 pairs of normal samples taken 10-15 minutes apart before injecting adrenalin; the average of these gives for the rate of blood flow and oxygen consumption of the first and second samples, respectively, 30.9 and 1.77; and 30.1 and 1.74 (all are cc. per minute). In other words in the 10 to 15 minutes elapsing between the first and second blood samples the rate of blood flow decreased only 0.8 cc. per minute and the oxygen utilization only 0.03 cc. per minute.

The effect of injecting adrenalin at the rate of 0.0004 mg. per cc. per minute for 5 minutes; we give the average of 10 experiments; in these the rates of blood flow and of oxygen consumption just

[5] Himwich and Castle, *Am. J. Physiol.*, 1927, lxxxiii, 92.

before the injection were, in cc. per min., 29.0 and 2.05; just after the injection they were, 26.0 and 1.93, *i. e.*, the adrenalin decreased the blood flow 3.0 cc. and the oxygen utilization 0.12 cc. per minute. We have another set of 9 experiments in which the adrenalin was given at just twice the rate used above, *i. e.*, 0.0008 mg. per cc. per minute for 5 minutes; the average rate of flow and of oxygen consumption just before these injections was 33.8 and 2.44, respectively; just after, the figures are, 27.6 and 2.14; *i. e.*, this dose of adrenalin slowed the rate of blood flow by 6.2 cc. per minute and the oxygen utilization 0.30 cc. per minute.

Although these decreases of oxygen utilization are not large they are 4 and 10 times greater than the change observed to occur spontaneously during an interval 2 or 3 times as long between normal samples; but even more significant, perhaps, is their constancy. Of the 18 pairs of normal samples, the oxygen consumption of the second was less than the first in 11 and greater in 7; of the 19 adrenalin experiments the oxygen consumption following the injection was less than the normal in 14, unchanged in 1, and greater in only 4.

These figures are derived from uncorrected oxygen contents; the determination of the oxygen capacity of 33 pairs of arterial-venous pairs gave an average venous capacity 0.46 vol. %, less than the arterial; this is not only opposite in sign to what has previously been reported (see 5) but also cannot be due to changes in cell volume, which, as an average of 27 determinations, was 55.07 for arterial, and 54.98 for the venous samples.

The effect of adrenalin on the carbon dioxide production was too variable and uncertain to justify speaking of it until further work is done.

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Dynamics of Insulin Secretion by the Pancreas and Epinephrine Secretion by the Suprarenal Gland.*

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Many authors have tried to prove that the pancreatico-duodenal vein is the carrier of the pancreatic hormone but nobody was able

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