Subsequent tests showed that the foregoing reaction was inhibited by the same substances as prevented the softening of shredded cabbage in acetic acid and that boiling the cabbage extract prior to its use, to inactivate any enzymes present, had no effect on the rate of the reaction.

It is concluded from this initial investigation that a factor exists in red cabbage which is non-enzymatic and which is capable of directly or indirectly carrying out an oxidative degradation of cellulose *in situ* under acid conditions. The change of protopectin to soluble pectin which apparently accompanies the loss of cellulose from the tissue could be explained either on the assumption of a similar oxidative factor affecting protopectin but not pectin, or alternatively that the insolubility of the protopectin present derived from its association with cellulose, a hypothesis most recently reviewed by Joslyn[5].

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PHYSIOLOGY

Reducing Agents and Absorption of Iron

Iron absorption has been shown to be increased by a variety of substances. Ascorbic acid is perhaps the best known of these[1]. The activities of inosine[2], succinic acid[3], fructose[4] and cysteine[5,6] have been discovered more recently. Several different hypotheses have been offered in explanation of the action of these substances. (1) That ferrous iron is more readily absorbed than ferric iron and that the ferrous form is maintained by an intra-luminal intestinal reducing agent (ascorbic acid); (2) that iron transport is linked with oxidative metabolism (succinic acid); (3) that iron transport depends on a carrier or chelating agent (fructose, cysteine).

It has not been generally appreciated that all these substances are reducing agents, either of themselves or in the milieu of the cell's interior. Succinic acid can reduce the prosthetic group of succinic dehydrogenase; fructose can reduce DPN when it enters glycolysis as glyceraldehyde-3-phosphate; inosine can reduce the prosthetic group of xanthine oxidase. All these substances can change the redox balance of the absorbing cell. A common action is also suggested by the quantitatively similar effect each exerts on iron absorption, a 1·5–2-fold increase.

We suggested that when the redox balance within the absorptive epithelium is tipped in the direction of reduction, iron absorption is increased. As a preliminary test of this hypothesis we administered by mouth iron plus hydroquinone, a reducing agent without known or postulated metabolic function. Iron absorption was examined in Walter Reed strain rats, weighing 250–400 g, utilizing repeated total-body counting to estimate absorption[7]. Control rats were dosed with 250 μg of iron as ferric chloride with iron-59 as tracer and with 1·6 mg of ascorbic acid, all dissolved in 1 ml. of distilled water. Experimental rats were dosed with the identical solution except for the addition of hydroquinone in varying doses. The small excess of ascorbic acid was included to preserve the ferrous state of intraduodenal iron in both control and experimental groups so that differential formation and absorption of ferric iron would not be in question.

Table 1. Iron Absorption in Control and Experimental Rats

Group	No. of rats	Hydroquinone dose (mg)	Percentage absorption (mean)
I	8	25	21·0
I (control)	8		12·7
II	8	45	21·0
II (control)	10		13·8

Absorption was increased in the hydroquinone-dosed rats (Table 1). The different doses of hydroquinone were similar in their effects on iron absorption. A *t*-test comparison between the combined hydroquinone-dosed rats and the controls showed the difference to be significant, with $P < 0{\cdot}005$. Separate experiments showed that the ascorbic acid used was sufficient to maintain the ferrous state of intraduodenal iron for at least 2 h. Hydroquinone, if it indeed functions as a reducing agent in increasing iron absorption, appears to work within the absorbing cell.

The 50 per cent increase in iron absorption obtained with hydroquinone is similar to that obtained with other compounds. This increase is relatively small compared with the increase which may occur with iron deficiency. The role which the intracellular redox potential may play in the absorption of iron in normal and abnormal states requires further definition.

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A Theoretical Model for the Computation of Decompression Tables for Divers

Discussions on the mechanism of decompression sickness have been focused mainly on the saturation and desaturation of the tissues with inert gases. To assess the capacity of the organism to free itself of an abundance of inert gas, experimental methods have usually been applied. Thus it has been possible to define a limited number of nitrogen-elimination rates which are spoken of as representing different types of tissues. Such information is, however, of limited value for the estimation of the relative importance of a certain tissue fraction (in terms of half-saturation time) for the genesis of decompression sickness. In measurements of nitrogen elimination the volumetrically large tissue fractions dominate the picture in a degree which is not necessarily related to their importance as bends tissue.

The conception of different histological types of tissues as representing different elimination rates is an oversimplification. Obviously, cells which are located at a larger distance from capillaries in a tissue will have slower nitrogen-elimination rates than those close to the walls of perfused vessels.

A more fruitful approach to the problems of inert-gas exchange in the organism seems to be to consider the body as composed of an unlimited or very large number of fractions as regards nitrogen-elimination rates. The ex-