sulphate were significantly lower. In the thoracic aorta hypophysectomy increased the DNA content from 6.0 ± 0.1 to 7.4 ± 0.6 mg/g; growth hormone treatment had no effect. Collagen content was not changed by hypophysectomy, but the elastin content increased from 357 ± 13 to 400 ± 14 mg/g; growth hormone administration returned the elastin content towards normal. Removal of the hypophysis had no effect on the hyaluronic acid content, but all of the sulphated glycosaminoglycans were markedly decreased: heparan sulphate from 2.3 ± 0.1 to 1.6 ± 0.1 μmol uronic acid/g, dermatan sulphate from 3.4 ± 0.2 to 2.7 ± 0.1 μmol uronic acid/g and chondroitin sulphate from 6.9 ± 0.6 to 3.7 ± 0.3 μmol uronic acid/g. Growth hormone treatment returned the concentrations of all three sulphated glycosaminoglycans to control values.

The significance of these findings in relation to vascular diseases will be discussed.

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Comparative Metabolism of Nicotinamide and Nicotinic Acid in Mice

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It is suggested that, in the mouse, metabolism is designed for the utilization of nicotinamide as a precursor of NAD and that the Preiss–Handler pathway (Preiss & Handler, 1958) functions primarily for the purpose of converting nicotinic acid into nicotinamide. It also appears that deamidation of nicotinamide to nicotinic acid is not a prerequisite to either its absorption from the gut or to its utilization as a precursor of NAD.

Adult female C3HeB/FeJ mice were injected intraperitoneally with [7-^{14}C]nicotinamide or [7-^{14}C]nicotinic acid at a dose of 0.9 μmol/animal. Mice fed on Purina chow *ad libitum* ingest approximately this same quantity of the vitamin per day. At intervals from 5 to 90 min after injection animals were killed and tissues were analysed for labelled nicotinamide coenzymes and precursors by paper chromatography.

In all the tissues examined more label was found in NAD after [7-^{14}C]nicotinamide injection than after [7-^{14}C]nicotinic injection. Nicotinic acid was rapidly cleared from all tissues, whereas nicotinamide persisted for 90 min. This finding agrees with observations by Chaykin, Dagani, Johnson & Samli (1965) and Chaykin, Dagani, Johnson, Samli & Battaile (1965). These authors found that nicotinamide is ordinarily the only form of the vitamin found in mice. In liver, after [7-^{14}C]-nicotinic acid injection, label in nicotinic acid rapidly disappeared. At 5 min label appeared primarily in desamido-NAD and NAD. By 10 min the concentrations of these compounds had fallen with a concomitant increase in labelled nicotinamide. By 20 min 90% of the total liver radioactivity was in the form of nicotinamide.

With regard to the metabolism of the vitamin in mice, a further question remained: namely, in what form does the vitamin pass the gut? When nicotinamide was administered, either by stomach tube or by direct injection into the intestine, the only form of the vitamin detectable in blood was nicotinamide. By contrast, 5 min after [7-^{14}C]nicotinic acid administration by the same routes, 30% of the radioactivity added to the blood during passage through the intestine was in the form of nicotinamide. Subsequent passage through the liver resulted in the removal of nicotinic acid from the blood and its replacement by nicotinamide.

These findings suggest that metabolism in mice is adapted to the utilization of nicotinamide and that nicotinic acid is converted into nicotinamide for this purpose.

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The Chemical Nature of Bound Nicotinic Acid

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It has been reported that the bound nicotinic acid of cereals is incorporated in a polysaccharide (Kodicek & Wilson, 1960) or in a polypeptide (Das & Guha, 1960). To investigate further the nature of bound nicotinic acid, experiments were carried out in which bound nicotinic acid was extracted in high yield from wheat bran by a mild chemical procedure. When wheat bran was extracted with aq. 50% (v/v) ethanol (after the method of Christianson, Wall, Dimler & Booth, 1968) at 0°C for 3 h, 60% of the total nicotinic acid was brought into solution. On dialysis 90% of this nicotinic acid