reflects an elevated steady state in the cerebral metabolism of the 5-hydroxyindoles, the decreased [5-hydroxytryptamine]/[5-hydroxyindol-3-ylacetic acid] ratios may indicate an increased bypassing of 5-hydroxytryptamine 'stores'. However, the fact that the total 5-hydroxyindoles ([5-hydroxytryptamine] + [5-hydroxyindol-3-ylacetic acid]) retained, for the most part, the normal pattern of distribution suggests that regional intracerebral hydroxylation was still the controlling factor.

It is of interest that the evidently important change in the metabolism of dopamine caused by tryptophan was detectable only from the study of body fluids, since the metabolite concentrations in the brain were unaltered.

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Isotopic Exchange between Acetylcholine and [*Me*-^{14}C]Choline Catalysed by Human Placental Choline Acetyltransferase

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Choline acetyltransferase catalyses the reversible transfer of acetyl groups between acetyl-CoA and choline. A marked difference in the kinetic behaviour of the partially purified enzymes from rat brain (Potter & Glover, 1968) and from human placenta (Schuberth, 1966) has been reported. Analysis of double-reciprocal plots of initial velocity versus the concentration of one substrate at different fixed concentrations of the second substrate indicate a 'sequential' ternary-complex mechanism (Cleland, 1963) for the brain enzyme; in contrast, a 'ping-pong' mechanism was indicated for the placental enzyme. Although such a fundamental interspecies difference in mechanism is not impossible it must *a priori* be considered unlikely.

Partially purified human placental choline acetyltransferase (Morris, 1966) was incubated with acetylcholine (10μmoles) and [*Me*-^{14}C]choline (0·5μc, 54μc/μmole). The incubation was stopped by addition of 0·2M-$HClO_4$, and unlabelled choline (10μmoles) was added as a carrier. The acetylcholine and choline were isolated via their reineckate salts (Hemsworth & Morris, 1964) and separated by paper electrophoresis (7kv for 1hr.) in 0·1M-pyridine–acetic acid buffer, pH4. The well-separated spots were located with either I_2 vapour or Dragendorff's reagent and cut out and the radioactivity was measured in a scintillation spectrometer. Recovery of radioactivity was between 95 and 100% and acetylcholine recovery, estimated by biological assay, was 90%.

A fairly rapid exchange of radioactivity occurred, 90% of the total being present in the acetylcholine after 30min. incubation. The exchange, however, was absolutely dependent on the presence of CoA; various control experiments gave exchange of 1% or less. This result rules out the 'ping-pong' mechanism and is good presumptive evidence for a ternary-complex mechanism. It is very probable that acetyl-CoA is first formed, and this is then able to incorporate labelled choline into acetylcholine by reversal of the initial step. That acetyl-CoA formation rather than just the presence of CoA is required is rendered more likely by the observation that addition of oxaloacetate and citrate synthase inhibited the exchange to an appreciable extent. Further evidence was provided by the marked inhibition obtained in the presence of chloroacetylcholine; this compound has been shown (Morris & Grewaal, 1969) to be a potent specific inhibitor of acetylcholine synthesis.

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Isolation of a Mucoprotein from Porcine Gastric Mucosa

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Of the various muco-secretions from the gastric mucosa (Glass, 1962), blood-group substances (Kabat, 1956), a sulphated mucoprotein (Pamer, Glass & Horowitz, 1968) and sulphated uronic acid-containing mucopolysaccharides (Kimura, Watanabe & Nagai, 1964) have been well characterized, after isolation by enzymic digestion. To study the major mucoprotein secretion of the surface mucosa without previous enzymic digestion, the gastric mucoproteins were radioactively labelled by incubation of porcine gastric mucosal scrapings with [U-^{14}C]glucose *in vitro* by the method of Allen & Kent (1968). Acid hydrolysis and chromatography of the non-diffusible material showed that radioactivity had been incorporated (14·8% of the total added d.p.m.) into mainly galactose (77%) with smaller amounts in fucose (5%) and the amino sugars, glucosamine and galactosamine (8%).

Studies have been mainly concerned with the undegraded water-soluble mucoprotein fraction, which accounts for 22% of the non-diffusible radioactivity. The water-soluble fraction, on gel filtration, gives three radioactive and u.v.-absorbing peaks that have similar specific radioactivities (1050d.p.m./mg.) and chemical analyses: protein, 20%; hexose, 30%; hexosamine, 32%; fucose, 9%; sulphate, phosphate and uronic acid all less than 0·3%. Except for the higher protein content, this analysis is similar to that of the blood-group substances isolated from peptic digests of porcine gastric mucosa (Kabat, 1956). Further, the water-soluble fraction possesses both A and H blood-group activity. Gel filtration, together with the above analysis, suggests that the faster-moving peak is a polymeric form of the two slower-moving peaks. The radioactivity in the water-soluble fraction is thus in a non-sulphated neutral mucoprotein that shows blood-group activity and comprises 83% by weight of the total water-soluble material.

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Properties of an α-D-Galactosyltransferase in Human Tissues obtained from Blood-Group B Donors

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A particle-bound α-D-galactosyltransferase that transfers D-[^{14}C]galactose from UDP-D-[^{14}C]galactose to low-molecular-weight acceptors containing β-linked D-galactose residues substituted at C-2 with L-fucose was demonstrated in a human submaxillary gland from a blood-group B donor and was not found in glands from donors of group A or O (Race, Ziderman & Watkins, 1968). The presence of this enzyme has now been confirmed in eight group B and two group AB submaxillary-gland specimens and in the mucosal linings from three group B stomachs. The transferase was found in the tissues of both secretor and non-secretor persons belonging to group B, whereas tissues of group A and O controls consistently failed to yield evidence of the enzyme.

The acceptor specificity of the transferase was more clearly defined by examination of a range of low-molecular-weight fucose-containing oligosaccharides isolated from human milk (cf. Kuhn, 1957; Montreuil, 1960). In agreement with the earlier findings, those containing an α-L-fucosyl residue (1→2)-linked to the terminal non-reducing galactose unit, namely 2′-fucosylgalactose, 2′-fucosyllactose and lacto-*N*-fucopentaose I, were good acceptors of galactose in α-linkage. Lacto-*N*-fucopentaose II, lacto-*N*-difucohexaose II and 3′-fucosyl-lactose, which have an L-fucose substituent on the subterminal sugar, and lacto-*N*-difucohexaose 1 and lactodifucotetraose, which are substituted with two L-fucose residues on adjacent sugars, were poor acceptors, or failed to accept, in the same system. The specificity requirements of the α-galactosyltransferase are therefore consistent with those postulated for the product of the blood-group *B* gene (cf. Race *et al.* 1968).

Attempts to solubilize the particle-bound enzyme by ultrasonic treatment or by extraction with 1% sodium deoxycholate resulted in loss of α-galactosyltransferase activity. Treatment with 1% digitonin or Triton X-100 increased the transferase activity of the particles obtained from either submaxillary glands or stomach mucosal linings and released soluble enzyme from the stomach preparations. The most effective method so far investigated for solubilization is extraction with digitonin of an acetone-dried powder prepared from the particles from stomach mucosal lining. The transferase activity of the particles is retained by the insoluble acetone-dried powder and treatment of this preparation with digitonin resulted in the release of about half of the activity in a soluble form.

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Properties of α-Galactosidase I from *Vicia faba*

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Dormant *Vicia faba* seeds contain two molecular forms of α-galactosidase (α-D-galactoside galactohydrolase; EC 3.2.1.22). Form I has a molecular weight of 209000 and appears to be a glycoprotein whereas form II is substantially free of carbohydrate and has a molecular weight of 38000 (Dey & Pridham, 1968, 1969*a*,*b*).

The specificity and mode of action of enzyme I