result diagnostic for actin was the presence of 3-methylhistidine in purified surfactant apolipoprotein.

A physiological role for surfactant apolipoprotein/actin can be suggested from the binding of myosin sub-fragment 1 (S_1) (Tsilibary & Williams, 1983). In the type II alveolar cells that secrete surfactant S_1-decorated actin fibres were particularly abundant near lamellar bodies, the intracellular stores of surfactant phospholipid. Taken together with the binding of cytoplasmic apolipoprotein to dipalmitoyl phosphatidylcholine emulsions, this report suggests that apolipoprotein/actin is involved in the intracellular migration and exocytosis of lamellar bodies. The presence of this protein in both immature lung cytosol and immature amniotic fluid could be due to actin derived from non-surfactant sources.

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Placental permeability to metabolites in fed and starved rats

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Foetal growth and oxidative metabolism are supported by the continuous transfer of nutrients from the mother (Battaglia & Meschia, 1978; Munro *et al.*, 1983). The passage of metabolites through the placental barrier may occur by simple diffusion (e.g. for fatty acids), facilitated diffusion (e.g. for glucose) or active transport (e.g. for amino acids) (Enders & Schneider, 1978). The net transfer of a metabolite to the foetus is influenced by the concentration gradient between mother and foetus (Shelley, 1979), blood fluxes at both sides of the placenta (Wilkening *et al.*, 1982), and the actual permeability of the placenta to that metabolite (Dancis & Schneider, 1978). Thus changes in the concentration of a metabolite in the maternal circulation lead to variations in its transfer to the foetus (Boyd *et al.*, 1973). Placental transfer of different substances has been well established in systems which allow simultaneous sampling from both sides of the organ (Battaglia & Meschia, 1978; Faton & Yudilevich, 1981). These studies have confirmed that, independently of the transport mechanism and the net transfer to the foetus, placental permeability differs for each metabolite (Battaglia & Meschia, 1978; Seeds *et al.*, 1980). It is not yet known, however, whether the placenta itself, as an organ, is able to modify its effective permeability to a metabolite in order to modulate its transfer to the foetus. As the foetal/maternal ratio of circulating metabolites varies and is greatly modified by starvation, in the present work we compared the placental permeability of different metabolites in the rat and determined whether maternal starvation affects this parameter. Fed and 48h-starved 21-day-pregnant Wistar rats anaesthetized with sodium pentobarbital (33mg/kg body wt.) were infused for 20min through a cannula placed at the level of the left uterine artery with trace amounts of different ^{14}C-labelled metabolites dissolved in 0.9% NaCl, by using the surgical procedure and other methodological details described by Lasunción *et al.* (1983). In this way, the left uterine horn received the tracer directly, whereas it reached the right horn after dilution in the mother's circulation. The difference between plasma radioactivity in foetuses from the left and the right uterine horn was used as an index of the permeability to the infused metabolite (Lasunción *et al.*, 1983; Palacín *et al.*, 1983). D-[U-^{14}C]Glucose, L-[U-^{14}C]alanine and [U-^{14}C]glycerol were used as tracers because these metabolites are transferred through the placenta by different transport mechanisms (Widdas, 1952; Dancis & Schneider, 1978), and because their concentration in maternal plasma changes with starvation (Herrera *et al.*, 1969; Girard *et al.*, 1977). Two non-metabolizable compounds (3-*O*-methyl [U-^{14}C]glucose and α-amino [1-^{14}C]isobutyric acid) were also used to determine whether placental permeability to them differed from that of their metabolizable analogues. Plasma radioactivity values were much higher in foetuses from the left than from the right horn when [^{14}C]glucose or [^{14}C]alanine was infused, as indicated by the high plasma radioactivity difference value (Table 1), demonstrating the expected high placental permeability to these metabolites. When [^{14}C]glycerol or α-amino[^{14}C]isobutyric acid was infused, however, these difference values were much smaller (Table 1), indicating that placental permeability to these compounds is lower than to glucose and alanine. For α-amino[^{14}C]isobutyric acid, the different results compared with those with L-[^{14}C]alanine may be a consequence of the needs of the placenta to incorporate into its metabolism the amino acid to be transported (Carroll & Young, 1983), although the known difference in the nature of the carrier for α-aminoisobutyric acid and alanine (Enders *et al.*, 1976) could also influence results. With 3-*O*-methyl[^{14}C]glucose, which is fully recognized by the D-glucose carrier (Johnson & Smith, 1980), observed values were very similar to those with D-[U-^{14}C]glucose (Table 1), indicating that glucose metabolism does not affect its placental permeability index. To correct values for a potential unspecific leak, infusion was also performed with L-[1-^{14}C]glucose. As also shown in Table 1, the difference in plasma radioactivity between foetuses from the left and the right uterine horn was very small, which, besides validating the technique used, demonstrates the specificity of placental transport for D-glucose. Experiments in 48h-starved rats were conducted specifically to determine the eventual effects of starvation on placental permeability, but not to quantify the net transfer of metabolites to the foetus. As shown in Table 1, plasma radioactivity differences between foetuses in the left and the right horn were very similar in starved and fed animals for any ^{14}C-labelled compound. This finding indicates that starvation in the rat does not modify placental permeability. Plasma concentration of both glucose and alanine decrease, whereas that of glycerol increases with starvation in the mother (Herrera *et al.*, 1969; Girard *et al.*, 1977), and these changes may secondarily affect the actual metabolite transfer throughout the placenta, even in the presence of unaffected permeability. Therefore the present results suggest that differences in the foetal/maternal ratio of circulating metabolites known to be caused by starvation in the pregnant rat are not due to altered placental permeability to metabolites, but are a

Table 1. *Plasma radioactivity differences between foetuses from the left and the right uterine horn after infusion of L-[U-^{14}C]-alanine, D-[U-^{14}C]glucose, 3-O-methyl[U-^{14}C]glucose, α-amino[1-^{14}C]isobutyric acid, [U-^{14}C]glycerol or L-[1-^{14}C]glucose in the fed and 48h-starved 21-day-pregnant rat*

Infusions were performed for 20min under pentobarbital anaesthesia with the tracers diluted in 0.9% NaCl, at the rate of 12.5 *ml*/min. Values were always corrected by considering 1×10^6 c.p.m. as the total infused radioactivity per rat, and are expressed as means ± S.E.M. Numbers of animals per group are indicated in parentheses. Statistical differences between fed and 48h-starved rats were always not significant ($P > 0.05$).

	Foetal plasma radioactivity differences (left − right)	
	Fed	48h-Starved
L-[U-^{14}C]Alanine	10973 ± 2144 (6)	8800 ± 460 (6)
D-[U-^{14}C]Glucose	6141 ± 713 (6)	9929 ± 1635 (6)
3-*O*-Methyl[U-^{14}C]Glucose	9576 ± 2398 (6)	7681 ± 913 (6)
α-Amino[1-^{14}C]isobutyric acid	1972 ± 195 (4)	2448 ± 648 (4)
[U-^{14}C]Glycerol	1944 ± 447 (6)	2026 ± 300 (6)
L-[1-^{14}C]Glucose	314 ± 65 (3)	—

concurrence of other factors known to affect the transfer, including, among others, availability of metabolites in the maternal side, blood flow, and foetal consumption.

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Studies in humans on the development of the GST1, GST2 and GST3 isoenzymes

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The GST1 are dimeric detoxicating enzymes that in humans are the products of three autosomal loci, GST1, GST2 and GST3 (Board, 1981). These isoenzymes exhibit variability, but only in the case of GST1 does this appear to be genetically determined (Strange *et al.*, 1984). Although studies have reported GST activity in human foetal tissues, no systematic study of the development of these loci has been described and little is known of the details of their expression *in utero*. We now describe experiments to separate, by chromatofocusing, the products of these different gene loci in foetal and neonatal liver cytosol and determine their rela-

Cytosol was prepared from samples of liver obtained with the permission of the Ethics Committee of the Simpson Memorial Maternity Pavilion, Royal Infirmary, Edinburgh, U.K. They were eluted at 4°C from columns containing Polybuffer exchanger 94 (Pharmacia Fine Chemicals, Hounslow, Middlesex, U.K.) previously equilibrated with start buffer (25mM-imidazole buffer, pH 7.3). The pH gradient was formed by using Polybuffer 74 adjusted to pH 4.0 with HCl (5M). Fractions were analysed for enzyme activity and pH. In initial experiments each peak of activity was pooled, concentrated and examined by starch-gel electrophoresis to determine which gene products were present.

Chromatofocusing resulted in the separation of the different GST isoenzymes (Fig. 1). The GST isoenzymes eluted first, usually as two peaks of activity. The first peak was not retained and corresponded to the isoenzyme with fast cathodal mobility, the second peak where present (eluted between pH 6.70 and 6.95) to the isoenzyme with slower cathodal mobility. The GST1 isoenzymes eluted between pH 6.25 and 6.50 (GST1-2 phenotype) or between pH 5.90