

## DOES GASTRIN CROSS THE SHEEP PLACENTA?

A.W. BELL\*, P.D. CRANWELL\* and J. HANSKY\*\*

Gastrin is thought to influence early gastric development in the neonate (Cranwell and Hansky 1980). What role gastrin has in gastrointestinal development in the fetus and whether circulating gastrin is of maternal or endogenous fetal origin is not known. The following two studies were performed in an attempt to answer the latter question.

Twelve late-pregnant ewes with single fetuses had catheters placed in a carotid artery, a uterine artery and vein, and the fetal aorta. Study 1: During the last 30 d of gestation, blood was sampled simultaneously from the maternal carotid and the fetal aorta at 3-5 d intervals. Study 2: In 3 experiments with 2 of the animals, human gastrin G17-I was infused via the uterine artery at a rate of  $160 \text{ pmol min}^{-1}$  for 30 min, preceded and followed by 30 min periods of saline infusion; blood was sampled at 10 min intervals from the uterine vein and maternal carotid artery and the fetal aorta. Plasma gastrin was measured by radioimmunoassay using antiserum 74 which measures G17 and G34 equally (Hansky *et al.* 1971). In the first study, average maternal gastrin was  $58.1 \pm 2.6 \text{ fmol ml}^{-1}$  and average fetal gastrin  $65.1 \pm 3.9 \text{ fmol ml}^{-1}$  (mean  $\pm$  SE of 74 samples). These were not significantly different. In each of the gastrin infusion studies maternal uterine vein and carotid artery gastrin rose significantly but fetal gastrin remained unchanged (Fig. 1).

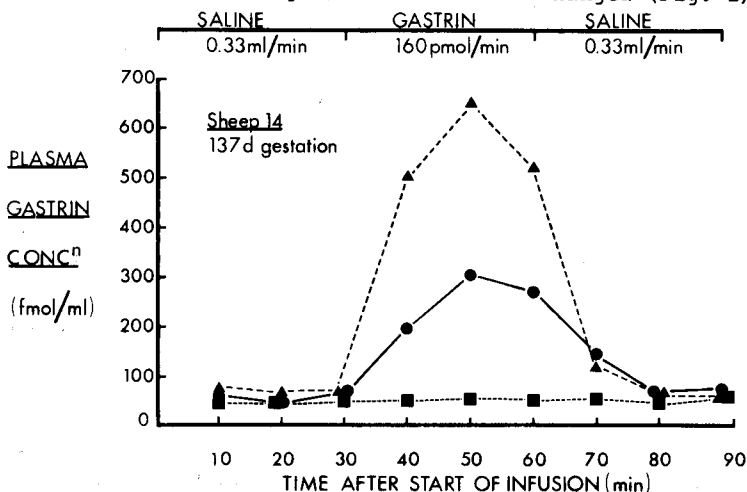


Fig. 1. Plasma gastrin concentration in uterine venous (▲), maternal carotid (●) and fetal aortic (■) blood during infusion of saline or gastrin into the uterine artery.

These studies suggest 1. Human gastrin does not cross the sheep placenta; 2. The fetal lamb produces its own gastrin; 3. If gastrin is a growth factor, then endogenous fetal gastrin may be the hormone which promotes growth and development of fetal gastric mucosa.

CRANWELL, P.D. and HANSKY, J. (1980) *Res. vet. Sci.* (In the press)

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\* School of Agriculture, La Trobe University, Bundoora, Vic. 3083.

\*\* Department of Medicine, Monash University, Vic. 3004.