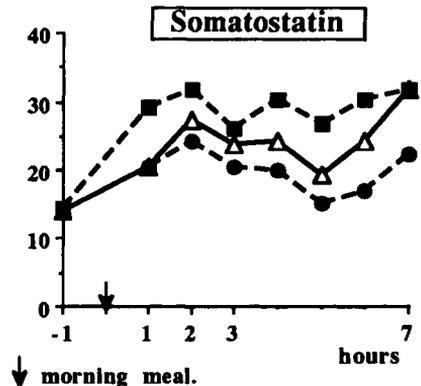
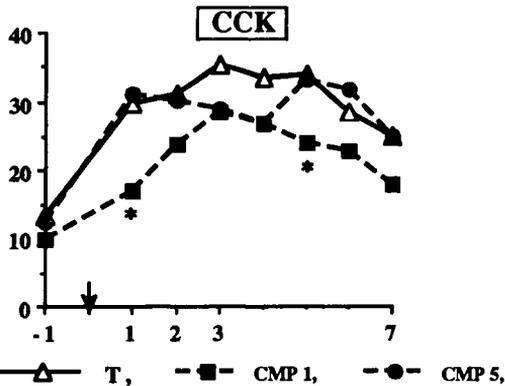
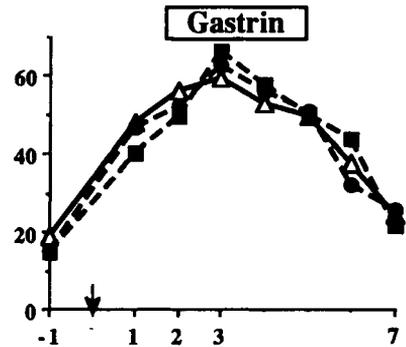
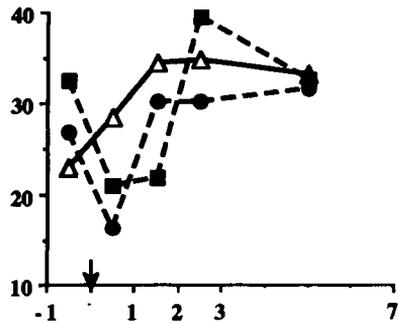


**Quantities of secreted gastric juice**

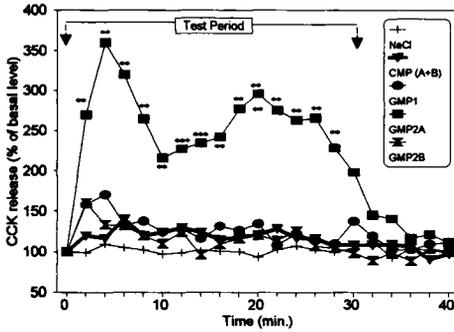


**Fig 1.** Effect of CMP ingestion on quantities of secreted gastric juices (g/h/100 kg LW) and the plasma levels of 3 digestive regulatory peptides (pmol/l). \* Significant differences from the T diet ( $P < 0.05$ ). (P Guillotteau *et al*)

**Effect of caseinomacropptide (CMP) on cholecystinin (CCK) release in rat.**  
 S Beucher<sup>1</sup>, F Levenez<sup>2</sup>, M Yvon<sup>1</sup>, T Corring<sup>2</sup> (1 INRA, Recherches Laitières; 2 INRA, Laboratoire d'Écologie et de Physiologie du Système Digestif, Domaine de Vilvert, 78350 Jouy-en-Josas, France)

*In vivo*, CMP, which is a  $\kappa$ -casein fragment, is the first hydrolysis product emptied from the stomach after milk or casein ingestion. Several authors have reported that CMP acted on gastrointestinal tract functions, probably through hormonal regulation. This study evaluates the effect of CMP on the release of the intestinal hormone CCK.

CMP is a heterogeneous fraction because it contains all post-translational modifications of  $\kappa$ -casein (phosphorylation and glycosylation) and mutations of the major genetic variants A and B. Four fractions were isolated from CMP by HPLC. They differ by their carbohydrate contents and by genetic variants. We have studied the effect of these CMP fractions on CCK release using an isolated duodenojejenum model of rat developed by Cuber *et al* [(1989) *Am J Physiol* 256, G989-G996]. Fractions were infused into the lumen of the isolated intestine at physiological doses for 30 min. For every fraction 6 trials were performed. Venous effluent was collected as 2-min fractions for 30 min, and CCK8 was determined by RIA. NaCl perfusion was used as control.



**Fig 1.** Effects due to luminal infusion of different fractions of CMP on CCK release. Results are in percent increase above basal level. Asterisks represent the significant differences from basal level (\*  $p < 0.05$ ; \*\*  $p < 0.01$ ; \*\*\*  $p < 0.001$ ). (S Beucher *et al*)

Only the fraction containing the slightly glycosylated forms of variant A (GMP2A) strongly stimulated CCK release (fig 1). The carbohydrates, and especially sialic acid, played a major role in activity, because the carbohydrate-free fraction of CMP, variant A and B (CMP(A + B)) was inactive and desialylation of the active fraction suppressed its effect. However, sialic acid solution induced only a very short and low response, consequently the peptide chain also appears to be involved in the peptide activity. The carbohydrate content and the amino-acid replacement of genetic variant A are also supposed to

play a major role. Indeed, highly glycosylated forms of variants A and B (GMP1) and slightly glycosylated forms of variant B (GMP2B) were not potent CCK secretagogues.

Therefore, the peptide activity is due to a definite structure involving sialic acid, which is always located at the end of sugar chains, and replaced amino-acid residues of variant A. The threonine 31 residue of this variant may be particularly involved, because it is a glycosylation site.

These results show that this casein peptide is able to stimulate the release of a hormone involved in the regulation of gastrointestinal functions, probably through binding to intestinal receptors.

### Effects of sorghum tannins on the activity of peptidases in the small intestine of the weaned piglet.

R Lizardo <sup>1</sup>, J Peinau <sup>1</sup>, JM Rouanet <sup>2</sup>, P Besançon <sup>2</sup>, A Aumaitre <sup>1</sup> (<sup>1</sup> INRA, Station de Recherches Porcines, 35590 Saint-Gilles; <sup>2</sup> Université de Montpellier II, 34095 Montpellier Cedex 05, France)

The presence of peptidases, *N*-aminopeptidase (NAP), dipeptidyl peptidase IV (DPP IV) and  $\gamma$ -glutamyl transferase ( $\gamma$ GT), in the membrane of the brush border enterocytes of the pig small intestine has been demonstrated [Kenny and Maroux (1982) *Physiol Rev* 62, 91-128]. The deleterious effect of sorghum tannins on the intestinal

**Table I.** Intestinal peptidases, alkaline phosphatase and maltase specific activities <sup>1</sup> in the weaned piglet: effects of site and dietary tannins (R Lizardo *et al*).

Peptidase	Site of measurement S <sup>2</sup>			Diet D <sup>3</sup>			Statistical analysis <sup>4</sup>		
	Duodenum	Jejunum	Ileum	Maize	Sorghum 0.03%	Sorghum 1.36% <sup>5</sup>	S	D	RSD
<i>N</i> -Aminopeptidase	24.0 <sup>b</sup>	38.7 <sup>a</sup>	44.2 <sup>a</sup>	31.2	37.5	36.9	**	ns	19.5
Dipeptidyl peptidase IV	43.7 <sup>b</sup>	61.5 <sup>a</sup>	66.8 <sup>a</sup>	66.8	48.8	53.2	*	ns	33.1
$\gamma$ -Glutamyl transferase	16.8 <sup>b</sup>	22.6 <sup>ab</sup>	28.6 <sup>a</sup>	29.7 <sup>b</sup>	15.6 <sup>a</sup>	14.5 <sup>a</sup>	*	**	13.7
Alkaline phosphatase	365.2 <sup>b</sup>	403.6 <sup>ab</sup>	481.0 <sup>a</sup>	458.7	413.4	370.9	*	ns	182.9

<sup>1</sup> International units per mg protein; <sup>2</sup> average value for the 3 sites of measurements; <sup>3</sup> average value for the 3 diets; <sup>4</sup> \*, \*\*  $P < 0.05$  and  $0.01$  respectively; RSD: residual standard deviation; <sup>a, b</sup> are significantly different; <sup>5</sup> tannins as % of catechin in the diet.