exocrine secretion and induced several modifications of the microstructure of the upper gut. The structural changes in the proximal part of the small intestine are not surprising, since the expression of CCK-A receptors has been demonstrated in several animal species. In contrast, no CCK-A receptor mRNA has been found in the ileum. In conclusion, in neonatal calves the influence of CCK on upper gut development has been shown. It could act both directly via CCK-A receptors expressed on the upper part of the small intestine and indirectly by the modulation of pancreatic secretion.

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Effect of nitrite on electrical activity of rat gastric fundus and duodenum in vivo. M. Ceregrzyn, M. Wiechetek (Department of Pharmacology and Toxicology, Faculty of Veterinary Medicine, Warsaw Agricultural University, Nowoursynowska 166, 02-787 Warsaw, Poland)

In addition to methemoglobin formation and vasodilatation, nitrites influence the motility of the gastrointestinal tract. Our recent observations show that nitrite markedly decreases gastrointestinal motility in vitro and it should be pointed out that the in vitro effects of nitrite are elicited by doses that do not cause classic toxic signs. Thus, the aim of the present study was to determine if doses considered non-toxic might induce changes in gastrointestinal electrical activity in rats in vivo.

The experiment was performed on four female Wistar rats. Two bipolar electrodes were surgically implanted, one in the stomach (the border between fundus and corpus, along the greater curvature) and the second in the duodenum. The intragastric cannula was placed into the stomach near the oesophagus on the lesser curvature. The experiment was performed 10 days after surgery on freely moving animals, which had free access to water and food until the experiment began. The electrical activity of the gastrointestinal tract was recorded 30 min before and 90 min after the 0.9 % NaCl or sodium nitrite (SN) administration (single dosage of 0.5 mL of $NaNO_2$ solution through an intragastric cannula reaching doses of 20, 30 or 65 $mg \cdot kg^{-1}$). The recording of the electrical activity was performed using an analogue digital recording system (MacLab, ADInstrments). The frequency of sampling was 100 samples/s, the signal was filtered at 50 Hz (high frequency cut) and 0.3 Hz (low frequency cut).

SN caused a change in the spiking activity of the gastrointestinal tract, whereas the administration of the same volume of 0.9 % NaCl did not cause any changes in the electrical activity of rat stomach and duodenum. The frequency of spikes during the control period of observation was 48.35 ± 13 and 27.2 ± 5 spikes/min (± SEM) in the stomach and in the duodenum, respectively. Twenty $mg \cdot kg^{-1}$ of SN was the lowest effective dose decreasing the frequency of spikes in the stomach and in the duodenum. The decrease in the frequency was observed for 10 min after the SN administration and the inhibition of electrical activity lasted about 60 min. The frequency of spikes recorded 10 min after the administration of 20 $mg \cdot kg^{-1}$ SN reached 26.6 ± 1 and 22.1 ± 1 spikes/min in the stomach and in the duodenum, respectively. The amplitude of spikes remained unchanged except during the short interval of the lowest frequency observed after the administration of the highest dose of SN (65 $mg \cdot kg^{-1}$). Clinical signs of methemoglobinemia (cyanosis and increased breath rate/min from 40 ± 5 in the control period to 65 ± 4) appeared 80 min after nitrite administration only in the case of the highest dose of SN.

The results obtained indicate that nitrites in doses not producing clinical signs of methemoglobinemia induce changes in the electrical activity of the stomach and the duodenum of rats.