the present studies the contents of all cystic stomach implants were at pH 7-8. If acid were being produced by the implant, then buffering by proteins in the cyst contents or leakage of hydrogen ions across a faulty gastric mucosal barrier as postulated by Davenport[2] might be responsible for the neutral pH. It would be worthwhile to study acid-secretion in stomach implants using more sophisticated techniques and the use of gastrointestinal hormones and pharmacologically active substances.

In some implantation experiments we used very young fetal stomachs of age 13-14 days, when no canalicular parietal cell antigen can be demonstrated. The appearance of this antigen after 5-10 days implantation confirms the finding of other authors[12,14] that parietal cells can develop out of immature undifferentiated mucosal cells.

The decrease in expression of the canalicular parietal antigen in allogeneic implantations and the observed inflammatory and atrophic changes in the mucosa could be compared with the loss of parietal cells and humoral and cellular immune response to parietal cell antigen in chronic atrophic gastritis in humans.

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Correction

Structure of three isomeric host-specific toxins from *Helminthosporium sacchari*, Experientia *39*/4 (1983) 343-347.

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We regret a misprinting in table 3 of this article. The table should have read as follows:

Table 3. ^{1}H-NMR data[a] for aglycone moieties

Carbon No.	Isomer A H_α	H_β	Isomer B H_α	H_β	Isomer C H_α	H_β
C-1	1.40	1.80	1.28	1.8	1.34	1.88
C-2	-	?	-	3.88	-	4.20
C-3	2.04	2.40	1.97	2.77	5.50	
C-5	-	-	1.92	-	2.10	-
C-6	2.73	2.24	1.8	1.63	1.99	1.52
C-7	-	2.61	-	2.62	-	2.64
C-8	1.64	2.00	1.8	1.8	1.83	1.93
C-9	1.37	1.37	1.43	1.32	1.45	1.29
C-12	5.11; 5.20		5.19; 5.30		5.24; 5.33	
C-13	4.03; 4.23		~4.07; 4.22		4.05; 4.23	
C-14	1.70		4.64; 4.95		1.72	
C-15	1.15		0.78		0.92	

[a] δ-Values in ppm relative to internal DSS = 0.