Letter to the Editor

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It has recently been suggested that an abnormal anion exchange mechanism operates in the sweat glands of cystic fibrosis (CF) patients (1, 2). According to this suggestion, a defect in the CF sweat gland results in abnormally low permeability of the sweat duct to chloride ion. The normal exchange of chloride for bicarbonate cannot then occur, so that in CF sweat there is an abnormally low level of bicarbonate as well as an abnormally high level of chloride.

There is now well established evidence that pancreatic bicarbonate secretion is abnormal in all CF patients (3, 4, 5, 6) and, therefore, it seemed sensible to look at chloride levels in these secretions. We examined duodenal aspirates obtained during pancreatic function testing previously described (5), and obtained the data shown in Table 1. Bicarbonate values were obtained on the samples at the time of collection. Chloride values were obtained on samples stored at −70°C.

Chloride output from CF patients is lower than that of controls so at first glance Quinton's hypothesis of defective chloride-bicarbonate exchange would not appear to be true for secretin-pancreozymin stimulated pancreatic secretions. Interestingly, however, the ratio of chloride output to bicarbonate output is much higher for CF patients than controls ($P < 0.001$). This is true for all the CF patients regardless of their trypsin output although the patient with highest trypsin output has the lowest chloride/bicarbonate ratio.

It may be, therefore, that both the pancreas and sweat gland in CF exhibit abnormalities in ion exchange involving chloride and bicarbonate. The hypothesis of Johansen *et al.* (8) and Hadorn *et al.* (7), suggesting a generalized disturbance of water and electrolyte movement in exocrine tissue warrants re-examination to take into account this added information. We urge other investigators to collect more data on total electrolyte output of pancreatic secretions to see if our findings can be substantiated and explained.

REFERENCES AND NOTES

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Response

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We find the above report and data very encouraging with regard to the possibility of a generalized abnormality in Cl permeability in cystic fibrosis (1, 2). As the authors point out, the production of both Cl^- and HCO_3^- are significantly lower in CF patients than in control subjects. Although this result is probably, at least in part, due to secondary loss of pancreatic parenchyma, the ratio of Cl^- to HCO_3^- production is always >1.0. In control subjects, the same ratio is always <1.0. It is perhaps more than coincidental that these same differences are found with respect to the ratio of Cl to Na production in sweat from CF and control subjects (3).

In the CF sweat gland, we now have evidence that Na is absorbed in excess of Cl^- due to the fact that the duct tissue is abnormally impermeable to Cl. That is, while the mechanism for Na absorption may be normal, net Na uptake is impeded by

Table 1. *Output of bicarbonate, chloride, and trypsin in pancreatic secretions over a time period of 0–15 min after secretin-pancreozymin stimulation*

Patient	Diagnosis	Age (sex)	Bicarbonate mmol/kg ($\times 10^{-2}$)	Chloride mmol/kg ($\times 10^{-2}$)	Trypsin (I.U.)	Chloride to bicarbonate ratio
E.D.	C.F.	8.0 (F)	1.0	3.4	0	3.4
C.B.	C.F.	6.0 (F)	0.5	2.8	0	5.6
L.A.	C.F.	17.0 (M)	0.1	0.8	0	8.0
M.A.	C.F.	14.0 (F)	0.1	0.4	0.54	4.0
S.H.	C.F.	6.0 (F)	0.3	1.2	2.61	4.0
W.H.	C.F.	18.5 (M)	1.6	3.4	12.88	2.1
C.P.	C.F.	11.5 (F)	0.7	4.8	24.70	6.8
T.A.	C.F.	8.5 (F)	6.1	6.3	33.41	1.03
A.H.	Malabsorption	11.5 (M)	21.2	9.2	49.15	0.43
S.H.	Pancreatitis	11.0 (M)	12.6	7.5	18.13	0.6
J.M.	Failure to thrive, diarrhea	8.0 (M)	17.3	17.0	52.37	0.96
R.N.	Hepatitis	7.5 (M)	14.2	10.8	47.27	0.76
K.O.	Pancreatitis	11.0 (F)	9.6	5.2	53.79	0.54
B.P.	Gastrointestinal reflux	6.0 (F)	12.0	7.7	28.77	0.64
			* $P < 0.05$	* $P < 0.05$	* $P < 0.25$	* $P < 0.001$

* P values refer to comparisons of data between the CF patients and controls.