this study is that the tests used did not pick up any latent abnormality of anorectal function in the continent diabetics.

In these studies the only objective finding was that of reduced internal sphincter pressure in the anal canal measured by manometer. Conscious appreciation of rectal distension is a highly unreliable test.[6] In our study, we found that thresholds of rectal distension could not detect differences between normal subjects, diabetics, or patients with faecal incontinence. None of the above papers have helped in defining the pathogenesis of faecal incontinence in diabetic subjects. They suggest that the cause is either internal anal sphincter dysfunction, external anal sphincter dysfunction or decreased awareness to rectal distension, or perhaps combinations of the above. In our study we have used precise, repeatable[7] and objective measurement of sensory and motor function of anorectal physiology, which has delineated previously unknown asymptomatic abnormalities in the pelvic floor of patients with diabetic neuropathy. This combined with the fact that the diabetic population are at risk of faecal incontinence[1] and that idiopathic faecal incontinence is commonly associated with pelvic floor neuropathy suggests that progression of pelvic floor neuropathy in diabetic subjects may lead to faecal incontinence. We still maintain, however, that this view will only be confirmed by a further study of a group of incontinent diabetics using the same techniques.

J ROGERS, D M LEVY, M M HENRY, AND J J MISIEWICZ

Department of Gastroenterology and Nutrition, Central Middlesex Hospital, Acton Lane, Park Royal, London NW10 7NS.

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Are individuals born as twins at a higher risk of developing Crohn's disease?

SIR,—In a recent survey of the Swedish twin registry including 25 000 pairs of twins, Tysk *et al*[1] found that the prevalence of ulcerative colitis among twins was almost similar to its prevalence in the general population in Stockholm (74×10^5 *v* 78×10^5, respectively). The prevalence of Crohn's disease among twins, however, was almost twice the prevalence of this disease in the general population in the same area (106×10^5 *v* 54×10^5, respectively). Unfortunately, the above mentioned authors did not relate to the difference in the prevalence of both diseases in twins, nor did they suggest any explanation for the increased incidence of Crohn's disease in twins.

It is anticipated that the prevalence of ulcerative colitis and Crohn's disease would be higher in a population of twins than in the general population because of the genetic aetiologic factor in both diseases, particularly in Crohn's disease. When one twin is affected with Crohn's disease, the other twin is at increased risk of being affected, especially if both are monozygotic. When the prevalence of Crohn's disease in the twin population is calculated, however, even if both twins are affected, the prevalence is 88×10^5 – much higher than in the general population.

Another method of determining whether twins are at increased risk of Crohn's disease is to calculate how many patients affected with the disease have twins. We are currently completing a study of the familial incidence of Crohn's disease among Jewish patients in Israel. In a group of 154 patients, four were found to have an unidentical twin who was not affected. In this group, one in 39 Crohn's patients has a twin as opposed to the incidence of twins in the general population, which is approximately one in 89.[2]

It, seems, therefore, that there is an increased risk for an individual born as a twin to be affected with Crohn's disease. Additional data are needed to confirm this observation. A better understanding of this tendency may help in the understanding of the aetiology and transmission of the disease.

J ZLOTOGORA AND D RACHMILEWITZ

Department of Medical Genetics and Department of Medicine, Mt Scopus Hadassah University Hospital, Jerusalem, Israel.

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Reply

SIR,—We are grateful for the interest that Drs Zlotogora and Rachmilewitz have shown our twin study. They suggest that there is an increased risk for an individual born as a twin to be affected by Crohn's disease. This is based on their findings of four pairs of dizygotic twins within a population of 154 patients with Crohn's disease. None of these four pairs was concordant for the disease. Thus, in their study one of 39 Crohn's disease patients was a twin in contrast with one of 89 in the general population. These figures must, however, be interpreted with caution as their Crohn's disease population is small and risk of errors produced by chance must be fairly great.

Obviously Drs Zlotogora and Rachmilewitz find support for their hypothesis in our results as they comment on the high prevalence of Crohn's disease in the twin population in comparison with epidemiological data from Stockholm. We are presently performing an epidemiological study on inflammatory bowel disease within our catchment area. The preliminary data show that the prevalence for both ulcerative colitis and Crohn's disease is considerably higher than in Stockholm. Furthermore, the Stockholm data do not include ulcerative proctitis cases. Thus, epidemiological comparisons of this kind are like comparing apples and pears. In fact the prevalence data in our twin study were not included in the original version but were added on request by the referees in order to get an idea about selection bias. In our study only hospitalised patients could be traced which means that there is a good chance of identifying patients with Crohn's disease more easily while patients with ulcerative colitis can be lost as they are more often treated as outpatients. Our findings did indicate, however, that the selection bias was reasonable when comparing published prevalence figures from Sweden.

CURT TYSK, EVA LINDBERG, GUNNAR JÄRNEROT, AND BIRGITTA FLODERUS-MYRHED

Regionsjukhuset,
S-70185 ÖREBRO,
Sweden.

Possible target for cystic fibrosis in the small intestinal epithelium

SIR,—We read with interest the paper by Taylor *et al*[1] reporting the failure of jejunal biopsies from children with cystic fibrosis to respond to secretagogues. Taylor and coworkers used the short circuit current approach to evaluate the ability of cAMP- and Ca^{2+}-dependent substances to promote secretion. Secretagogues failed to have an effect in cystic fibrosis epithlium but the Ca^{2+}-ionophore A23187 was reported to have a small effect. This led the authors to suggest that the defect lies beyond the step for cAMP generation and that a mechanism for raising intracellular Ca^{2+} might also be impaired. We have recently been studying the ionic channels present in small intestinal epithelial cells by the patch clamp technique and would like to comment on Taylor and colleagues' data in the light of our own data.

Necturus small intestine, is an established model for intestinal ion transport studies, with which direct evidence that the apical membrane of enterocytes possesses a cAMP-activated Cl^- conductance has been obtained.[2] Chloride channels identified in isolated enterocytes by the patch clamp technique[3] share many of the properties documented for the Cl^- channels defectively regulated in cystic fibrosis airway epithelium.[4–6] In cell attached membrane patches the channel is activated by an increase in cellular cAMP. Upon excision of the membrane patch silent channels could be irreversibly activated by strong (+120 mV) depolarisation. In excised, inside-out membrane patches bathed in symmetrical Cl^--rich solutions the chloride channels exhibit outward rectification, that is outward currents (chloride flowing from the extracellular to the cytoplasmic side) observed at depolarised potentials are significantly greater than inward currents recorded at corresponding hyperpolarising potentials. The small intestine Cl^- channels are highly selective for anions (Br^-, F^-. I^- and SCN^-, but not HCO_3^- or gluconate, are permeant) over cations. Moreover, changing the Ca^{2+} concentration bathing the cytosolic patch surface was without effect upon channel behaviour. This is again consistent with results for Cl^- channels originating from tracheal epithelium.[4]

The homology between the Necturus enterocyte Cl^- channel and those of mammalian origin suggests that this channel may be the agent responsible for intestinal secretion reported by Tayor *et al.* If this were indeed the case then the conclusions drawn from their experiments with the calcium ionophore should be re-examined in view of the Ca^{2+}-insensitivity of this channel. The possibility must be considered that the ionophore induced elevation of intracellular Ca^{2+} activates basoateral K^+ channels, which are known to be Ca^{2+}-activated.[7–9] The result-