urinary excretion of calcium even when differences in glomerular filtration rates were disregarded. As possible explanations for their observation of maximum excretion during the period May-September, with a zenith in July or August, they mentioned changes in dietary calcium intake and the seasonal variations which the serum level of 25-hydroxycholecalciferol (25-OHCC) is known to undergo, or possibly a combination of both, since the zenith of serum 25-OHCC occurs later than that of the urinary calcium excretion.

We have studied the same problem by performing simultaneous determinations of the 24-hour urinary calcium excretion and the 24-hour clearance of creatinine in 26 healthy volunteers taking a standard diet. The estimated contents of this diet were 800 mg of calcium, 900-1100 mg of phosphorus, and 60-140 mmol (mEq) of sodium per day and 1 g of protein per kg body weight per day. There were 17 male subjects aged 21-29 (average 24) years and nine females aged 20-54 (average 34) years. Twelve, including five females, were studied from May to September inclusive and 14 (four females) from October to April inclusive. Assessment was undertaken as well of the 24-hour urinary calcium excretion, both uncorrected (UCaV (mg/24 h)), and corrected for variations in the clearance of creatinine (Ccr (ml/min)) according to the formula $(UCaV \times 100)/Ccr$. The importance of relating UCaV to Ccr in one way or another has been stressed repeatedly.¹⁻⁴ As evaluated by Student's *t* test the clearance-corrected excretion was found to be significantly higher during summer, the mean ($\pm$ S.D.) being 225 ± 61 mg/24 h as against 165 ± 37 mg/24 h during the winter ($t = 3.115$, $P < 0.01$). The figures for uncorrected excretion were 230 ± 66 mg/24 h and 199 ± 47 mg/24 h respectively ($t = 1.404$, $P > 0.10$). Despite the small number of observations the monthly averages displayed a pattern (see fig.) very similar to that so nicely presented by Dr. Robertson and his colleagues.

This pattern was recognizable even when males and females were considered separately.

Our observations indicate that seasonal variations in the daily urinary excretion of

calcium may take place independently of dietary changes.—We are, etc.,

I. TRANSBØL
F. SCHØNAU JØRGENSEN

Medical Department F,
Gentofte Hospital,
Hellerup,

B. LUND
O. H. SØRENSEN

Medical Department E,
Frederiksberg Hospital,
Copenhagen,
Denmark

- 1 Nordin, B. E. C., Hodgkinson, A., and Peacock, M., *Clinical Orthopedics*, 1967, 52, 293.
- 2 Transbøl, I., et al., in *La Sarcoidose*, Rapp iv Conf. Internat., p. 618, eds. J. Turiaf, and J. Chabot. Paris: Masson, 1967.
- 3 Transbøl, I., Hahnemann, S., and Hornum, I., *Acta Medica Scandinavica*, 1968, 184, 33.
- 4 Peacock, M., Robertson, G. W., and Nordin, B. E. C., *Lancet*, 1969, 1, 384.

Liver Flukes: a Warning

SIR,—I would like to correct a point in Dr. A. T. Hunt's letter (1 March, p. 510) dealing with liver flukes. The Tidenham epidemic¹ in fact occurred in the winter of 1968-9 and not in June 1972 as he states.—I am, etc.,

A. H. DAVIES

Chepstow, Gwent

- 1 Hardman, E. W., Jones, R. L. H. and Davies, A. H., *British Medical Journal*, 1970, 3, 502.

** The mistake was ours, not Dr. Hunt's, and was inadvertently introduced in transferring a reference from the text to the foot of his letter. The relevant passage should have read: "In my description of the Tidenham epidemic, published in June 1972. . . ." We apologize for any misunderstanding that this may have caused.—ED., B.M.J.

Schistosome Antigen in Transplanted Kidney

SIR,—I was enlightened to read of the demonstration of circulating soluble antigens and antibody in schistosomiasis by Drs. M. A. Madwar and A. Voller (22 February, p. 435). I have recently treated a patient with a long history of schistosomiasis (*Schistosoma mansoni*), nephrotic syndrome, and ultimate renal failure. Renal biopsy and nephrectomy studies revealed the glomerular hallmarks of immune-complex nephropathy. The patient received a renal transplant and subsequently underwent antischistosome therapy with niridazole. He developed a transient "one-shot" immune complex nephritis. With the use of specific anti-schistosomal adult worm antiserum the antigen was revealed in the glomerular mesangium of the transplant biopsy. Thus that "prerequisite for immune-complex disease" has been found in the kidney. These studies will soon be reported in full elsewhere.—I am, etc.,

DANIEL B. GOULD

Huntingdon Valley,
Pennsylvania, U.S.A.

Abuse of Fenfluramine

SIR,—Fenfluramine has been considered to be not a drug of abuse, because of initial unpleasant side effects.¹ Hawks² found no evidence of its abuse among addicts asso-

ciated with the addiction research unit. Götestam and Gunne³ showed that amphetamine-dependent subjects had no preference for fenfluramine against placebo. However, Levin⁴ described 60 drug-dependent young males who claimed to have used fenfluramine as a drug of abuse. Euphoria, derealization, and perceptual changes were reported effects. I wish to report a case of fenfluramine abuse.

The patient, a 28-year-old woman, was referred because she had been forging prescriptions for fenfluramine for the past six months. She had begun taking fenfluramine four years previously when her weight was about 170 lb (77 kg) and her height 5 ft 5 in (1.62 m). Initially she noticed nausea, vomiting, dizziness, and drowsiness on dosages of 80 mg a day. Weight loss was quite rapid, and for the past year she had remained at about 115 lb (52 kg). Her longest period of abstinence was six months. In the past seven months while taking on average 240 mg of fenfluramine daily she had noticed feelings of euphoria, excess energy, and little need for sleep. There was increased appetite and libido. There were no obsessional-compulsive symptoms. She found alcohol potentiated the effect of fenfluramine-induced euphoria. During this seven-month period she noticed increasing tolerance to the dosage. She had forged prescriptions on three or four occasions. She did not appear to have been abusing any other drug.

The patient's desire to lose weight was sufficient to overcome the initial unpleasant side effects. This pattern has been observed with more classical drugs of abuse—for example, morphine. There is little reference in the literature to the very obvious stimulant effect of fenfluramine found in this case.—I am, etc.,

H. P. ROSENVINGE

Knowle Hospital,
Fareham, Hants

- 1 Oswald, I., et al., *British Medical Journal*, 1971, 3, 70.
- 2 Hawks, D. V., *British Medical Journal*, 1970, 1, 238.
- 3 Götestam, K. G., and Gunne, L. M., *British Journal of Addiction*, 1972, 67, 39.
- 4 Levin, A., *British Medical Journal*, 1973, 2, 49.

Serum Muramidase Activity in Untreated Cancer

SIR,—We read with interest the paper by Professor E. H. Cooper and others (14 September, p. 662) on raised serum muramidase levels in colorectal cancer. They consider that the raised muramidase activity results from stimulation of the reticuloendothelial system. It is accepted, however, that muramidase is mostly derived from disintegrating granulocytes and that serum muramidase levels are an index of granulocyte destruction.^{1,2} When granulocyte production equals destruction muramidase levels are also an index of granulocyte bone marrow production,¹ and they have been used for monitoring recovery of granulopoiesis in patients with neutropenia.^{2,4} The results, however, were unreliable in patients with fever and bacterial infection,² since in cases of bacterial infection serum muramidase levels are raised owing to increased destruction.^{2,5}

We measured by the turbidometric method of Litwack⁶ the serum muramidase levels in 120 patients with various untreated malignant tumours, including 20 cases of colorectal cancer, and compared them with the levels in 50 controls. All were within the normal range and there was no significant difference between the patients and the controls. This agrees with the findings of Jollès et al.⁷ Our patients with colorectal