tion operates uniformly everywhere. However, the increasing proportion of rhesus-negative individuals among male donors (15-1%, in the years 1972-1974 compared with 14-6% among male adults involved in paternity cases in Norway) suggests that a similar self-selection is now operating among male donors as well. The present results therefore suggest that blood-donors are not suitable subjects for studies of population genetics involving investigation of the rhesus system.

I thank Prof. J. Lunderval, Institute of Forensic Medicine, University Hospital, Oslo, for permission to use data from the paternity cases, and Dr M. Fagerhol, Blood Transfusion Centre, Oslo City Hospital, for permission to collect information on new donors.

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LETSEN FEGERSTEN SAUGSTAD.

MANAGEMENT OF MILD HYPERTENSION

Sir,—I would take issue very strongly with your leading article of Feb. 1 (p. 259). It cannot be emphasised too strongly that there is as yet no evidence that substantial reductions in mortality and morbidity can be expected from the vigorous and relentless treatment of mild hypertension.3 Right thinking and a right policy in this matter are of the greatest importance if we are to avoid the lives of vast numbers of healthy people unnecessarily and committing the N.H.S. to much wasteful expenditure.

Dr Shiner and her colleagues (Jan. 18, p. 137) and in two other reports1 find no benefit from the restriction of sodium intake in the management of mild hypertension. The reference you cite does not support your contention. It deals with the risks of hypertension and not the benefits of treatment. The important findings of the Veterans Administration Cooperative Study Group on Antihypertensive Agents,2 though they appear to relate to patients with mild hypertension, do not in fact do so.4 Right thinking and a right policy in this matter is of the greatest importance if we are to avoid the lives of vast numbers of healthy people unnecessarily and committing the N.H.S. to much wasteful expenditure.

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DAVID SHORT.

SMALL INTESTINAL MUCOSA IN COW'S MILK ALLERGY

Sir,—We have made similar observations to those of Dr Shiner and her colleagues (Jan. 18, p. 137) and in two children with cow's milk protein intolerance we have found an increase in IgE plasma cells in the lamina propria of the small intestine after milk challenge. Our method is similar to that described, using tissue fixed in 2-5% buffered formaldehyde and stained with fluorescein-isothiocyanate-conjugated antisera (Hoechst, U.K.).

One child, bottle-fed from birth, was first seen aged 4 months with a history of vomiting, failure to thrive, and passing bulky stools after a gastroenteritis-like illness at 2 months. A small intestinal biopsy showed severe partial villous atrophy (P.v.A.) with patchy increase in eosinophils. The IgE plasma cells in the lamina propria were 44 per sq. mm. The infant was placed on Milk was withdrawn and 30 hours later, that is 4 days after the milk was seen.

2 weeks, having had cow's milk formula included in the diet a milk challenge. In the first child IgM plasma cells also showed an increase, but in the second child IgM plasma cells fell in number after milk challenge. We have also observed an increase of IgE plasma cells in other abnormal biopsies. In a series of biopsies from children with newly diagnosed coeliac disease (not yet proven by gluten challenge) IgE plasma cells have ranged up to 112 per sq. mm. compared with a series of controls with counts from 2-18 cells per sq. mm. IgE plasma cell counts were also raised in a biopsy from a child with mucosal damage following enteropathogenic Escherichia coli gastroenteritis.

It appears that IgE is involved in the immunological response of the lamina propria in several conditions associated with small intestinal mucosal damage. In cow's milk protein intolerance the IgE plasma cells persist for at least 6 days after milk withdrawal.

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ANNE KILBY

VITAMIN E, SELENIUM, AND KNEE PROBLEMS

Sir,—In 1970 I began to experience pain in my knees after sustained strenuous leg work. This condition became worse until in 1973 I was almost unable to complete a hike in nearby mountains because of pain. The problem was identified by an orthopaedic surgeon as ligament irritation on lateral and frontal parts of the knee joint. I was recommended to accept the situation since there was no accepted therapy.

I learned that selenium ingestion had been suggested as a method of relieving some types of arthritis.1 In January, 1974, I began to ingest gelatin capsules, each containing 1 mg. selenium as sodium selenite plus 68 mg. D-α-tocopherol succinate (vitamin E). One capsule was taken regularly with meals every third day. A week before a hike in September, 1974, I ran approximately 1/2 mile each day (as I had done before hikes in previous years), and I increased my selenium and vitamin-E intake into one capsule per day. In so far as I was able to plan the experiment, everything was the same as in previous years, with the exception of my selenium and vitamin-E intake. I hiked 11 miles in one day, ascending and descending 865 m. (2875 ft.) with absolutely no knee discomfort. This contrasted with past hikes, especially the one in August, 1973, in which the distance was identical but I only ascended and descended 506 m. (1685 ft.), and knee pain was nearly unbearable during the last 20% of the hike. The 1974 hike seemed to provide a suitable comparison because of the equal hiking distance and the 70% increase over the 1973 hike in elevation.

I hope that the success of this small personal experiment will encourage further research into vitamin-E and/or selenium therapy of arthritic problems in human knee joints. However, the hazards of selenium supplementation must be borne in mind—1 mg. selenium supplement per day probably approaches adult human toxicity levels. The vitamin-E levels did not exceed those in widespread use.

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