containing the D-Ala-D-Ala sequence.[3] Therefore the apparent discrepancy between our results and theirs in RA sera is not surprising. Furthermore we are not alone in failing to note an association between serum antibody to bacterial PG and RA. Pope *et al.* have recently made a similar observation.[4]

There is, however, one point on which all studies of anti-PG concur; namely, that the humoral immune response to bacterial PG in humans is diverse. Until the basis for this diversity is more clearly understood, the relationship of anti-PG to rheumatic diseases, such as AS, RS, and RA, will probably remain a matter of speculation.

Rheumatology-Immunology Center, Veterans Administration Medical Center, Philadelphia, PA, USA — H PARK, H R SCHUMACHER

Department of Biochemistry, Thomas Jefferson University, Philadelphia, PA, USA — A R ZEIGER

Kuzell Institute for Arthritis Research, Medical Research Institute, San Francisco, CA, USA — J T ROSENBAUM

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Blood dyscrasia associated with azapropazone therapy

SIR, The pyrazolon derivatives phenylbutazone and oxyphenbutazone are potent causes of blood dyscrasias,[1 2] but azapropazone, a pyrazolon derivative[1] in regular usage, has been considered to be free of haematological side effects.[2] In May 1984 an 80-year-old female was admitted for investigation of leucopenia and anaemia which had developed five weeks after commencing azapropazone 300 mg four times a day. Her other medications consisted of thyroxine 0·15 mg in the morning, ferrous sulphate 200 mg three times daily, and lactulose 15 ml twice daily. Significant medical history consisted of primary hypothyroidism (diagnosed in 1977), adenocarcinoma of the colon treated by anterior resection in 1979 and, in 1980, investigation of mild asymptomatic splenomegaly. Bone marrow smears analysed in 1980 were consistent with involvement by early chronic lymphatic leukaemia with satisfactory granulocyte maturation. At a follow up in March 1984 haemoglobin was 9·9 g/dl, red cells $3·52 \times 10^{12}$/l, white cells $4·1 \times 10^{9}$/l, and platelets 185×10^{9}/l. In May 1984 haemoglobin was 6·9 g/dl, red cells $2·49 \times 10^{12}$/l, white cells $0·6 \times 10^{9}$/l (neutrophils 79%, eosinophils 0%, basophils 0%, lymphocytes 20%, monocytes 1%), and platelets 121×10^{9}/l. The blood film showed mature white cells and a normochromic anaemia with marked anisopoikilocytosis; the bone marrow showed dyserythroblastic and dysmyeloblastic features with no evidence of carcinoma, and the granulocyte series was arrested at the myelocyte stage. Chest *x*-ray, serum B12 and folate, repeated faecal occult blood tests, and blood and urine cultures were unremarkable. Blood urea was 9·3 mmol/l and creatinine 151 μmol/l. Azapropazone was withdrawn, and by 12 days postadmission white cells were $4·1 \times 10^{9}$/l (neutrophils 81%, eosinophils 2%, basophils 1%, lymphocytes 10%, monocytes 6%), haemoglobin 9·0 g/dl, and platelets 187×10^{9}/l. She was discharged, and in November 1984 haemoglobin was 12·1 g/dl, red cells $3·93 \times 10^{12}$/l, white cells $6·1 \times 10^{9}$/l, and platelets 213×10^{9}/l.

This patient's underlying, albeit quiescent, haemopoietic problem may have rendered her more susceptible to a drug-induced toxic effect, suggesting that it may be prudent to avoid azapropazone in such cases.

Division of Geriatric Medicine, Stobhill General Hospital, Glasgow G21 3UW — STEPHEN T GREEN, HUGH MCMILLAN, LINDSAY ERWIN

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Notes

EULAR symposium 1986

A symposium on 'seronegative polyarthritis' will be held in Rome on 13–15 October 1986. Details from Professor Raffaele Numo, Cattedra di Reumatologia, Policlinico, 70124 Bari, Italy.

XVth Symposium of the European Society of Osteoarthrology

The symposium on 'Articular cartilage and other joint structures in relation to loading and movement' will be held on 25–27 June 1986 in Kuopio, Finland. Details from Heikki J Helminen, ScD(Med.), Managing Chairman, University of Kuopio, Department of Anatomy, PO Box 6, SF–70211 Kuopio, Finland.