

ABSTRACTS

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ABSTRACTERS

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IMMUNOHEMATOLOGY

ACQUIRED HAEMOLYTIC ANAEMIA. CLINICAL AND SEROLOGICAL OBSERVATIONS OF TWO CASES. W. Weiner, J. P. Whitehead and W. J. Walkden. From the Blood Transfusion Service, Birmingham, and Hallam Hospital, West Bromwich, England. *Brit. M. J.* 1: 73-77, 1956.

The first patient, a man aged 47, had dyspnea, fatigue, pallor and giddiness. He was known to have auricular fibrillation. The spleen was palpable and the provisional diagnosis was subacute bacterial endocarditis complicating mitral disease. A chest X-ray showed patchy infiltration of the left upper lobe suggesting pneumonia or tuberculosis. However, the hematologic and biochemical features of hemolytic anemia were present and the anemia could be partially controlled with cortisone. The cause of the lung changes was not established. The second patient, a female aged 50, was admitted with jaundice, anemia and purpura, and was found to have hemolytic anemia and thrombocytopenia. Blood transfusion gave only transient benefit, but cortisone therapy caused sustained improvement.

The cells of Case 1 were Group O, Rh-positive and seemed to be of the phenotype CeDee. The serum contained an anti-E antibody and an eluate from the patient's cells showed a strong anti-E activity. Further investigations suggested that the phenotype was in fact Ce DE and that the wrong result in the first investigation was due to an anti-E antibody fixed to the cells blocking the E antigen and preventing agglutination of the cells by the agglutinating serum. The cells of case 2 were group A, Rh-positive, and genotyping was possible only after they had been washed eight times to abolish a certain degree of auto-agglutination. The phenotype was ceDEe. The serum contained an anti-E antibody and the eluate contained not only an anti-E but also an anti-C which was not present in the serum.

It would appear to be essential that cross-matching for the selection of blood for such patients should be performed using not only the serum but also an eluate prepared from the cells of the patients.—R.H.G.

APLASTIC CRISIS IN A CASE OF IMMUNO-HEMOLYTIC ANEMIA. M. Seip. From the Children's Hospital, University of Oslo, Norway. *Acta med. Scandinav.* 153: 137-142, 1956.

A severe case of immuno-hemolytic anemia was observed, in which an aplastic crisis with erythroid aplasia in the bone marrow occurred a few days after bronchography with an iodine-containing contrast medium. Erythrocyte production was temporarily arrested. The patient's condition was critical throughout the crisis, which lasted for about four weeks. The longest duration of an aplastic crisis previously reported is twenty days.—M.S.

A NEW SEROLOGICAL METHOD FOR THE DEMONSTRATION OF HYPERSENSITIVITY TO ALLERGENS. I. METHOD AND FIRST CLINICAL EXPERIENCES. *R. Hoigné, W. Grossmann and H. Storck.* From Medizinische Universitätsklinik, Department of Coagulation Physiology; Dermatologische Universitätsklinik and Privatlaboratorium Dr. Grossmann, Zürich, Switzerland. *Schweiz. med. Wchnschr.* 85: 578-586, 1955.

Thirty-three patients with hypersensitivity to drugs, dust or food were investigated. If the allergen was added to dilutions of serum or plasma obtained from the allergic patients, a characteristic, slight turbidity appeared, which was estimated by a special nephelometer. In 31 of the 33 patients the test proved positive, whereas unsensitized controls always showed no reactions. The turbidity develops as the result of an interaction between the allergen and two factors: I. A factor obtained from the plasma or serum of sensitized patients which is specific for the allergen and is dialyzable. II. A serum factor which is nonspecific and nondialyzable. Eight hundred tests have been performed, and the stability of the factors as well as the effect of serum dilution, temperature and pH changes are reported. The results of this new test are similar to the platelet in-vitro test, (Hoigné), but the new method is said to be more convenient as a routine test.—*M.H.H.*

II. THE ALLERGY FACTORS IN THE SERUM OF SENSITIZED PATIENTS WITH SPECIAL REFERENCE TO A SPECIFIC INHIBITOR OF THE TURBIDITY REACTION. *R. Hoigné.* From Medizinische Universitätsklinik Zürich, Switzerland. *Schweiz. med. Wchnschr.* 85: 1272-1274, 1955.

Further experiences with the new test in 111 patients with hypersensitivity states are reported. The number of false negative results unfortunately is not mentioned. Apart from factors I and II already described, a third factor was found to appear in serum obtained from allergic patients and stored for 6-8 hours. This factor, which inhibits the turbidity reaction, is heat stable and dialyzable. Two components of this factor could be differentiated: component B acted as a specific blocker for factor II, and component N neutralized the allergen. Factors I and II are specific and dialyzable, the latter property being an exception from all antibodies known so far. They probably are not proteins. Factor III resembles the blocking antibody of Cooke et al. which is detectable with a skin test.

The use of the turbidity reaction is not limited by factor III, as it is not found in the fresh serum used for the tests. The communications ought to be looked at critically, and the experiments reported need confirmation. Some basic assertions seem to be in opposition to experiences in immunology hitherto.—*M.H.H.*

ANTIPLATELET PRECIPITATING ANTIBODIES (PLATELET PRECIPITINS) IN THE SERUM OF THROMBOCYTOPENIC SUBJECTS. *P. Ruggieri.* From the Istituto di Clinica Medica Generale, University of Rome, Italy. *Policlinico, sez. prat.* 62: 1157-1161, 1955.

A precipitation reaction was studied in 22 cases of I.T.P., 29 cases of secondary thrombocytopenia and 38 normal subjects, by employing platelet antigen and serum. In seven cases of I.T.P., in two cases of secondary thrombocytopenia, and in none of the controls, the reaction was positive. The two secondary thrombocytopenias were observed in the course of pancytopenia.—*P.d.N.*

PIGMENT METABOLISM

PORPHYRINS IN HEALTH AND DISEASE. *R. Lemberg.* From the Institute of Medical Research, The Royal North Shore Hospital, Sydney. *Australasian Ann. Med.* 4: 5-15, 1955.

This is an excellent review article. It is concerned mainly with the synthesis of porphyrins and with the patterns of porphyrin excretion in the different forms of porphyria.—*G.C.deG.*

AN IN VITRO STUDY OF THE BIOSYNTHESIS OF PORPHYRINS AND HEMOGLOBIN. *L. Eriksen.*

From the Institute of Physiology, University of Oslo, Norway. Akademisk Trykningscentral, Oslo. 53 pp.

This interesting monograph is written in Norwegian with an English summary. The biosynthesis of porphyrins and hemoglobin was studied in vitro in young red cells from rabbits rendered anemic by injections of phenylhydrazine. The author has used radioactive tracer substances and also chromatographic methods. The monograph should be studied in the original by those particularly interested in these problems.—*M.S.*

THE INHERITANCE OF PORPHYRIA. *G. Dean and H. D. Barnes.* From the Provincial Hospital, Port Elizabeth, South Africa, and the Biochemical Department, South African Institute for Medical Research, Johannesburg, South Africa. *Brit. M. J.*, 2: 89-94, 1955.

This is the most extensive study on the mode of inheritance of hepatic porphyria yet published. The authors have collected data on 13 porphyria families of Burgher stock with a total of 236 cases (121 male and 115 female). One of these families, including 479 members in 6 generations, is presented in some detail. Of 125 adult children of affected parents, 60 or 48 per cent have porphyria, namely, 24 males and 36 females. Thus the disease is inherited as a non-sex-linked Mendelian dominant. It is noteworthy that no abnormality in urinary porphyrin excretion was discovered in children under 18 years of age. Thirty-two children, comprising the 6th generation of this family, had all negative urines. Of particular interest is the observation that within the same family the disease became manifest as intermittent acute porphyria, as porphyria cutanea tarda, or as a mixture of both forms. In addition, a number of family members were discovered to have an asymptomatic "latent" form of the disease. In general, women were more prone to develop recurrent acute abdominal symptoms and neurologic complications, whereas in men cutaneous symptoms were seen with greater frequency. None of these families included any congenital (erythropoietic) porphyria.—*R.S.*

USE OF HEMATOPORPHYRIN FLUORESCENCE IN BILIARY AND CANCER SURGERY. *G. C. Peck, H. P. Mack, W. A. Holbrook, and F. H. J. Figge.* From the Departments of Surgery and Anatomy, University of Maryland, School of Medicine, Baltimore, Maryland. *Amer. Surgeon* 21: 181-188, 1955.

Intravenous injection of hematoporphyrin in mice, rabbits, dogs and humans leads to preferential uptake of this fluorescent compound in lymph nodes and neoplastic tissue. Twenty-four to forty-eight hours after the administration lymph nodes and neoplastic tissue can easily be identified by their brilliantly red fluorescence. Most of the hematoporphyrin is excreted with the bile and 1 to 2.5 hours after injection the entire biliary tree is easily visualized by its intense red fluorescence with a Wood lamp. Dosages up to 1000 mg. have been given to humans without apparent ill effects.—*R.S.*

CONGENITAL PORPHYRIA WITH ERYTHRODONTIA, SPLENOMEGALY AND ANEMIA IN TWO SISTERS. *Jan Weremowicz.* From the Internal Medical Service of the Miejskiego Hospital No. 2, Warsaw, Poland. *Polish Medical Weekly*, 9 (18): 550-552 and 9(19): 585-589, 1954.

Two sisters, aged 21 and 3 years, with congenital photosensitive porphyria are described. Both have severe seasonal photodermatitis, erythrodontia and splenomegaly. Excretion of deeply red urine, containing large amounts of uroporphyrin, was said to have been observed shortly after birth. Unusual is the severe alopecia which, each spring and summer, has accompanied the recurrence of the bullous skin eruptions on the exposed parts of the body. Both girls have outspoken anemia with polychromatophilia, occasional circulating normoblasts, marked normoblastic hyperplasia of the bone marrow, elevated serum bilirubin levels and increased urine urobilinogen excretion. In spite of these findings and the associated splenomegaly, the author considers a hemolytic anemia unlikely because of a normal red cell osmotic fragility. (Reviewer's note: It would appear that these two girls most likely had hemolytic anemia which is a feature in practically all cases of congenital photosensitive (erythropoietic) porphyria on record.) The parents of the patients and two other sisters are healthy and have a normal porphyrin excretion.—*R.S.*

EFFECT OF SPLENECTOMY ON PORPHYRIA ERYTHROPOIETICA. *I. M. Rosenthal, E. L. Lipton and G. Asrow.* From the Department of Pediatrics, University of Illinois College of Medicine, Chicago, Illinois. *Pediatrics* 15: 663-675, 1955.

The authors report extended studies of an 8 year old girl with erythropoietic (congenital, photosensitive) porphyria. The patient exhibited a compensated hemolytic disease with hemoglobin and hematocrit values close to normal but with elevated reticulocyte counts and with increased values for fecal urobilinogen and indirect reacting serum bilirubin. The patient's erythrocytes, when transfused into normal recipients were found to have a decreased survival time as determined by the Ashby technic. Removal of the enlarged spleen appeared to result in a temporary improvement of the hemolytic process and in porphyrin excretion; but less than a year postoperatively urinary and fecal porphyrin excretion had again reached preoperative values. Data are given for porphyrin concentration in erythrocytes, serum, spleen, and liver, and the bone marrow has been studied with the fluorescence microscope.—*R.S.*

INTERMEDIATES IN THE BIOSYNTHESIS OF PORPHYRINS FROM PORPHOBILINOGEN. *L. Bogorad.* From the Department of Botany, University of Chicago, Chicago, Illinois. *Science* 121: 878-879, 1955.

An enzyme has been prepared from spinach leaves which, in the presence of 0.1 M cysteine, deaminates porphobilinogen to a colorless intermediate exhibiting no longer a positive Ehrlich reaction. Frozen and thawed chlorella cells are capable of utilizing this intermediate for the synthesis of porphyrins with 3 to 7 carboxyl groups. Since the same chlorella cells are unable to metabolize uroporphyrin, it is considered unlikely that uroporphyrin is an intermediate in the conversion of porphobilinogen to copro- and protoporphyrin.—*R.S.*

EXPERIMENTALLY PRODUCED PORPHYRIA IN ANIMALS. *A. Goldberg and C. Rimington.* From the Nuffield Unit for the Investigation of Pyrrole Pigment Metabolism, Department of Chemical Pathology, University College Hospital Medical School, London, England. *Proc. Royal Soc., B*, 143: 257-280, 1955.

Previously reported studies on Sedormid porphyria in rabbits have been considerably enlarged to include other compounds, which are chemically related to Sedormid. In rabbits and rats, allyl-isopropylacetamid has been found to have a porphyrinuric effect in every respect similar to that observed with Sedormid. However its sedative effect is insignificant as compared to that of Sedormid. Rabbits treated with this new compound showed some gastrointestinal disturbances, but did not exhibit neurologic manifestations or hypertension, in spite of marked porphyrinuria. In the authors' opinion, this finding supports their previously expressed view, that in the human acute porphyria, the neurologic manifestations are not the result of the disturbed pigment metabolism.—*R.S.*

RENAL CLEARANCE OF ENDOGENOUS PORPHOBILINOGEN IN MAN. *A. Goldberg.* From the Nuffield Unit for the Investigation of Pyrrole Pigment Metabolism, Department of Chemical Pathology, University College Hospital Medical School, London, England. *Lancet*, 2: 1095-1097, 1954.

In four patients with acute hepatic porphyria, simultaneous creatinine, urea and porphobilinogen clearance studies were performed. The results indicate that endogenous porphobilinogen is excreted by glomerular filtration without appreciable tubular reabsorption.—*R.S.*

PORPHOBILINOGEN AND δ -AMINO LEVULINIC ACID IN ACUTE PORPHYRIA. *S. Granick and H. G. Van den Schrieck.* From the Laboratories and Hospital of the Rockefeller Institute for Medical Research, New York City. *Proc. Soc. Exper. Biol. & Med.* 88: 270-273, 1955.

Since δ -amino levulinic acid is known to be an obligatory intermediate in the biosynthesis of porphobilinogen, porphyrins and heme, it was reasonable to expect that patients with acute (hepatic) porphyria would excrete this keto-acid in the urine. This has now been

found to be the case. However, this observation contributes little to a better understanding of the underlying defect in acute porphyria since it is not known whether the urinary excretion of δ -amino levulinic acid results from an excessive rate of formation of this compound in the liver, or from a partial block above the porphobilinogen step. On the basis of the latter assumption, the authors have attempted to calculate the liver's capacity for heme formation. Such an attempt would seem to be subject to a number of objections and the significance of the calculated values appears to be open to question.—*R.S.*

LEAD INTOXICATION: I. THE EFFECT OF LEAD ON THE IN VITRO BIOSYNTHESIS OF HEME AND FREE ERYTHROCYTE PORPHYRINS. *L. Eriksen.* From the Institute of Physiology, University of Oslo, Norway. *Scandinav. J. Clin. & Lab. Invest.* 7: 80-85, 1955.

By means of labeled sodium acetate and ferric ammonium citrate the author in *in vitro* experiments with normal duck erythrocytes has shown that lead exercises a strong inhibitory effect on the formation of heme. This effect is probably due to a diminished formation of the porphyrin part of heme. It is suggested that free erythrocyte protoporphyrin is used for the biosynthesis of heme.—*M.S.*

RADIATION EFFECTS

COBALT-INDUCED POLYCYTHEMIA AND SURVIVAL OF X-IRRADIATED ALBINO RATS. *C. F. Gessert and P. H. Phillips.* Department of Biochemistry, College of Agriculture, University of Wisconsin, Madison, Wisconsin. *Proc. Soc. Exper. Biol. & Med.* 89: 651, 1955.

A comparison of life survival following exposure to 750 roentgens whole-body X-irradiation of normal albino rats and cobalt-pretreated rats showed a decrease in the survival rate of the cobalt-treated animals. Six groups of 20 animals each were used in this study. Oral doses of cobalt of 10, 20, 40, 70 and 100 ppm were used in the various groups for periods of 19 weeks prior to and 6 weeks after irradiation. The ratio of the increase in hemoglobin concentration in each cobalt-treated group to the control group after 14 weeks was proportional to the logarithms of the added dietary cobalt.—*W.N.J.*

STIMULATION OF ERYTHROPOIESIS IN SUBLETHALLY IRRADIATED RATS BY A PLASMA FACTOR. *F. Stohman, Jr. and G. Brecher.* National Institute of Arthritis and Metabolic Diseases, National Institutes of Health, Bethesda, Md. *Proc. Soc. Exper. Biol. & Med.* 91: 1, 1956.

The administration of sublethal doses of irradiation to rats results in a decreased rate of erythropoiesis, which may be quantitated by use of the rate and degree of incorporation of radio iron into red cells. In rats subjected to sublethal irradiation, there was a significantly increased rate of erythropoiesis induced by the immediate postirradiation injection of plasma obtained from prephlebotomized rats. The substance responsible for this accelerated rate of erythropoiesis was nondialyzable and not present in the supernatant of heated, acidified plasma.—*W.N.J.*

EFFECT OF WHOLE-BODY X-IRRADIATION ON UPTAKE OF IRON BY DUCK ERYTHROCYTES. *J. R. Klein and R. Cavalieri.* Biology Department, Brookhaven National Laboratory, Upton, New York. *Proc. Soc. Exper. Biol. & Med.* 89: 28, 1955.

Comparisons of the degree of uptake of radioiron by duck erythrocytes in an *in vitro* system before and after total body irradiation were made. Following roentgen irradiation, reticulocytes were decreased, the plasma iron level remained unchanged and there was a decrease of the *in vitro* uptake of radioiron by erythrocytes. The decreased ability of erythrocytes to incorporate radioiron correlated well with the decreased number of immature reticulocytes. There was no evidence that *in vivo* radiation interfered with the ability of the reticulocytes present to incorporate radioiron into hemin. Irradiation of the duck erythrocytes *in vitro*, failed to alter the erythrocyte radioiron uptake.—*W.N.J.*

The Other Journals of Hematology

Revue d'Hematologie, Vol. 11, No. 2, April-May 1956. Editor Marcel Bessis, 6 Rue Alexandre Cabanel, Paris XV^e, France. E. de Harven and P. Dustin, Jr.: Hormonal control of eosinophilia. II. Antagonism between certain mitotic poisons and cortisone on the level of the eosinophilic myelocytes in the rat. A. Schrumpf: Lifespan of red cells in hereditary spherocytosis. R. Latarjet: Biological considerations in the chemotherapy of cancer. J.-P. Thiery and M. Bessis: Mechanism of platelet formation. A study *in vitro* by microcinematography.

British Journal of Haematology, Vol. 2, No. 3, July 1956. Editor J. V. Dacie, Postgraduate Medical School of London. C. C. Booth and D. L. Mollin: Plasma, tissue and urinary radioactivity after oral administration of ⁶⁶Co-labelled vitamin B₁₂. G. Chertkow and J. V. Dacie: Results of splenectomy in autoimmune haemolytic anaemia. W. R. Pitney: The assay of antihæmophilic globulin (AHG) in plasma. T. Iversen and P. Bastrup-Madsen: Congenital familial deficiency of factor V (parahaemophilia) combined with deficiency of antihæmophilic globulin. G. Merskey and N. Sapeika: The anticoagulant action of chlorazol fast pink. J. G. Humble, W. H. W. Jayne and R. J. V. Pulvertaft: Biological interaction between lymphocytes and other cells. P. H. Renton and J. A. Hancock: Variability of the rhesus antigen D. A. S. Wiener and E. B. Gordon: A hitherto undescribed human blood group, A_m. T. P. Telfer, K. W. Denson and D. R. Wright: A 'new' coagulation defect. W. H. Crosby, O. L. Sapp and H. Anderson, Jr.: A sex difference in the response to titrated irradiation therapy (³²P) of patients with chronic granulocytic leukaemia. J. H. Crookston, J. V. Dacie and V. Rossi: Differences in the agglutinability of human red cells by the high-titre cold antibodies of acquired haemolytic anaemia.

Acta Haematologica, Vol. 16, No. 2, August 1956. Secty. H. Lüdin, Burgerspital, Basel. C. Kubota, S. O. Schwarz and F. W. Putnam: Multiple myeloma: correlation of the clinical, the marrow and electrophoretic findings. H. Warnakoff and H. Brücher: Cytologic studies of plasmacytoma (Ger). A. Fieschi, E. Bianchini, G. Cambiaggi, C. Sacchetti and E. Salvidio: Studies on the biological behaviour of the cells of acute leukaemia. W. Künzer, A. Schütz and E. Schütz: Comparative study of spontaneous oxidation of hemoglobin in umbilical cord and adult erythrocytes after inhibition of glycolysis (Ger). R. Gross, J. Heuer and K. Solth.: Quantitative relations between heparin and platelets (Ger). B. Ramot and K. Singer: An unusual circulating anticoagulant in systemic lupus erythematosus.

Vol. 16, No. 3, September 1956, H. B. W. Greig: Myleran in the treatment of chronic myeloid leukaemia. J. Jürgens: Congenital factor VII (SPCA)-deficiency as cause of a hemophiloid hemorrhagic diathesis (Ger). E. Shafir, A. deVries and E. Krejnis: Interaction of Convertin with platelets and with platelet-free and hemophilic plasma. R. J. Oechslin: Osteomyelosclerosis and the skeleton (Ger). S. Otsuka, Y. Koshiishi, K. Naruto and K. Kumai: Pernicious anemia in Japan (statistical study) (Ger).

Sangre, vol. 1, No. 2, 1956. Editor J. Guasch, Copernico, 68, Barcelona. M. Bessis and J.-P. Thiery: Megakaryocytes and platelets examined by electron microscopy. M. Bloch: Morphologic cytology in the diagnosis of diseases of the hematopoietic system. H. H. Hennemann: Hemolytic syndromes in the diseases of the lymphatic system. A. Hittmair: Myeloid metaplasia in the spleen. P. C. Junquera and P. J. Wishart: Distribution of the ABO system among Portuguese people in Rio de Janeiro. V. Maspes, T. Verrastro, E. Coelho and M. Jamra: Platelet agglutinins. I. Nonspecific agglutination by a coagulation factor present in normal serum (factor VII). C. Meza Arrau, A. Nijamkin and R. Valdivieso: Research on anti-erythrocytic antibodies and their role in the hemolytic syndromes. M. Ortega: Determination of potassium in human serum by flame photometry. G. Sansone and A. M. Piga: A regenerative crisis in Cooley's disease. J. P. Soulier and D. Alagille: Sensitivity of various plasmas to thrombin. Study of the "thrombin time" in patients with hepatic disease. A. Lessa: Therapeutic procedures in severe burns. The syndrome "membrane destruction."

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Le Sang, Vol. 27, No. 4, 1956. Editor: F. Emile-Weil, 24 bis, Ave. du President-Wilson, Paris (XVI^e). M. Adant, K. Herman and P. Wissocq: Intravenous injection of amniotic fluid in normal and hepatectomized dogs. A. Aschkenasy: The anemia induced by experimental adrenalectomy. M. Samama and J. Colombani: Quick's prothrombin time in leukemia. J. Hugues and J. Lecomte: Pathogenesis of purpura induced by croton oil. M. Boiron, M. Tubiana, C. Paoletti, A. Dutreiz and J. Bernard: Lesions of the spleen produced by selective irradiation of the spleen and by partial or total body irradiation. A. Guichard, J. Fayloie, R. Alex and A. Revol: Decalcifying aleukemic myelosis implicating platelets and megakaryocytes. Platelet leukemia. J. M. Levy, Mme. Weill-Bousson, Fr. Stephan, L. Fruhling and H. Metzger: Three cases of aleukemic megakaryocytic myelosis. A. Coreos, V. Coreos, S. Coreos and A. Cittanova: Acute erythromyelosis (di Guglielmo's syndrome) with hepatomegaly without splenomegaly in a child. Failure of cortisone.

Blut, Vol. 2, No. 3, July 1956. Editor G. Blumenthal, Föhlerstrasse 2, Berlin N65. T. Lüers, H. Nachtsheim and G. Petzel: Phenocopy of homozygotic Pelger anomaly as a consequence of reactive shift to left in a patient with heterozygotic Pelger anomaly. E. Matsunaga: Selection by ABO incompatibility between mother and fetus. Y. Bayraktar: Adsorption of human and animal hemoglobin on aluminum hydroxide. J. Schröder: Determination of osmotic resistance of leucocytes. G. Walther: Coagulation activity of hemolysed human erythrocytes. A. Windorfer, H. E. Schultze and G. Schwick: Hemophilia with antibody to AHG. E. Koch and H. Winter: Blood and bone marrow in decompensated essential hypertension. G. A. Delijannis: Sickle cell disease in Greece.

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