hypertransfused rats, which, unlike the plasma iron in normal animals, is not further elevated following radiation (Gurney, C. W., and Taylor, K. T., unpublished results). It must be stressed that only effective erythropoiesis is being measured, and future investigation must also include a study of the blood-forming organs to assess the degree to which stem cells damaged by irradiation undergo differentiation and then succumb while still erythrocyte precursors.

In addition to affording a means for the simple quantitative determination of irradiation damage and recovery of a population of cells, we believe this model offers possibilities for investigation into the nature and kinetics of the stem-cell compartment, and it is for these reasons that these preliminary observations are reported.

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Development of the Islets of Langerhans in Man

Very little has been published about the differentiation of the α and β cells in the development of the pancreatic islets in man. The only work since the introduction of the modern granule stains is that of Ferner and Stoeckenius[1] (based on twelve fœtal pancreases), and Schultze Jena[2] (based on six fœtal pancreases).

The material for the present work was 36 pancreases from human fœtuses with weights ranging from 34–3,000 gm. (10 wk. gestation to full term), stained by Gomori's chrome-alum hæmatoxylin phloxin method[3] and/or by chrome-alum hæmatoxylin with Gomori's one-stage trichrome counterstain. Eleven pancreases were from fœtuses between 10–20 wk. gestation. It is the islets in these early months of gestation, in particular, which have not been adequately described before.

Four stages are observed in the development of the islets. These overlap so that any one pancreas will show islets in more than one stage :

(1) State of budding islets. In the third gestational month, clusters of islet-cells bud off from the ducts. Differentiated α and β cells are already present. The β cells form a central cluster, around which ungranulated islet-cells and occasional α cells are ranged.

(2) Bipolar stage. As the islets grow, the β cells become grouped at the tip of the islet farthest from the duct, while the α cells form a calyx at the base of the islet. The proportion of ungranulated cells diminishes. Islets in this bipolar phase are observed from the fifth to the eighth gestational month.

(3) 'Mantle-islet' stage. The α cells at the base of the islets proliferate and grow right round the β cells. The islet now has a kernel of β cells and a shell of α cells. Some ungranulated cells are still present among the α cells.

There is a good deal of overlapping between the bipolar and mantle-islet stages. Mantle-islets are seen as early as the fifth gestational month, and dominate the picture from about the sixth gestational month.

(4) Stage of mature islets. From the eighth gestational month onward, a few islets can be found where the α and β cells have the more-or-less haphazard distribution characteristic of the mature adult islet, although the majority are 'mantle-islets' right up to term and beyond.

A detailed account of this work, with photomicrographs, will be reported elsewhere.

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PATHOLOGY

Serological Tests for Toxoplasmosis

Interest in various serological tests for toxoplasmosis has increased considerably in recent years. Siim and Lind[1], in a preliminary communication, have recently described a flocculation test using toxoplasma antigen adsorbed on to acrylic particles. They stated that preliminary results of absorption experiments showed the existence of more than one toxoplasma antigen. The following experiments carried out in this Laboratory may be of interest.

A high-titre human antitoxoplasma serum was absorbed with toxoplasma-infected mouse peritoneal exudate at room temperature (20° C.). This removed almost all the hæmagglutinating, complement-fixing and dye-test antibodies. Absorption of the same serum overnight with human red cells coated with toxoplasma antigen (as used in the hæmagglutination test of Jacobs and Lunde[2]) removed the hæmagglutinating antibody but the dye test and complement-fixation antibodies were unaffected.

Control tests were set up using a normal human serum, absorbed with toxoplasma suspension, and a normal human serum absorbed with red cells coated with toxoplasma antigen. This experiment excluded effects due to elution of soluble toxoplasma antigen. Absorption of the antitoxoplasma serum with human unsensitized cells did not alter the titres of the dye, hæmagglutinating or complement-fixing antibodies. The results are summarized in Table 1.

The results of this experiment were substantiated by using a HeLa cell tissue culture toxoplasma antigen to absorb a hyperimmune serum from another patient. The effect of using a tissue culture antigen

Table 1. ABSORPTION OF ANTITOXOPLASMA SERUM

	Dye test	Hæmagglutination test	Complement-fixation test
Antitoxoplasma serum unabsorbed	Positive at 1/1,280	Positive at 1/1,280	Positive at 1/640
Antitoxoplasma serum absorbed with toxoplasma suspension	Positive at 1/40	Negative at 1/20	Negative at 1/20
Antitoxoplasma serum absorbed with sensitized human red cells	Positive at 1/1,280	Negative at 1/20	Positive at 1/1,280
Antitoxoplasma serum absorbed with unsensitized human red cells	Positive at 1/1,280	Positive at 1/1,280	Positive at 1/1,280
Normal human serum control absorbed with toxoplasma suspension and then mixed with antitoxoplasma serum	Positive at 1/640	Positive at 1/320	Positive at 1/640
Normal human serum control absorbed with sensitized human red cells and then mixed with antitoxoplasma serum	Positive at 1/640	Positive at 1/640	Positive at 1/640

as an absorbent was essentially the same as that with the mouse exudate antigen.

These experiments demonstrate that the antibody measured in the hæmagglutination test differs from that in either the dye or complement-fixation tests. Therefore the antigen or antigens responsible for the hæmagglutination test differ from those responsible for the dye and complement-fixation tests.

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Effect of a Magnetic Field on Sarcoma 37 Ascites Tumour Cells

THIS communication deals with work on the effect of magnetic field on living ascites tumour cells *in vitro*. The hanging-drop culture was prepared by diluting a 7-day-old transplanted ascites mouse tumour with suitable aliquots of Tyrode solution. The culture slide to be exposed to the field was then placed between the poles of an Alnico permanent magnet in a horizontal position at a constant temperature of 37° C. The field strength used during the experiment was about 4,000 gauss and was determined independently by the susceptibility technique. The control slides not exposed to the field were treated identically. The microscopic examination of the slides was carried out periodically.

The control tumour cells showed normal growth and active differentiation, even after 18 hr., whereas most tumour cells exposed to the field showed complete degeneration at the end of this period. These experiments were repeated several times with reproducible results. The slides made after 18 hr. were stained with acetocarmine and photomicrographs were made.

Although it is not possible to explain these results clearly at the present time, the following postulates and assumptions are considered in working out a hypothesis for this purpose. (1) The 'chemical components' in the cells have varying magnitudes of magnetic susceptibility representing the diamagnetic and the permanent and any transient paramagnetic contributions. Hence, the permanent and any transient components will experience varying forces under the influence of the magnetic field. (2) Although these forces are small from a macroscopic point of view, they may be enough to upset the delicate balance (chemical and other equilibria) inside and outside the cell. (3) Such changes are assumed, at least in part, to produce the degeneration of the cells.

We wish to thank Dr. G. S. Sperti for his encouragement and Messrs. Albert Gerth and Robert Neagele for their valuable assistance. Further work on the effect of a magnetic field on living cells will be published elsewhere.

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Association of Detectable Antibodies produced by *Haemonchus* spp. Infections in Cattle, with Serum Globulin Fractions

EARLIER investigations have shown that cattle which exhibit a marked degree of natural resistance to infection with *Haemonchus* spp. also develop higher complement-fixation titres than susceptible cattle, and that rises in the complement-fixation antibody titres often occurred at the same time as rises in the gamma one fraction of the serum globulin. This resistance has been shown to be heritable[1,2]. These observations were made under field conditions where extraneous stimuli were likely to produce changes in the globulin fractions, so that the correlation between complement-fixation titres and the γ-1-globulin fraction was not always consistent and definite conclusions were not possible. To clarify this situation, cattle sera known to be positive by the complement-fixation test were fractionated and the fractions identified and tested for antibody content. The following is a brief account of the procedure and results obtained.

The β- and γ-globulins were first separated from the serum by precipitation with 16 per cent sodium sulphate at 37° C. The globulin was further fractionated on a diethylaminoethyl cellulose chromatographic column with gradient potassium acid phosphate buffer solutions ranging from *p*H 7·0, 0·005 *M* to *p*H 5·8, 0·1 *M*, after the technique in refs. 3 and 4. Three antibody positive fractions were isolated in this manner. The protein content of these fractions was estimated by the biuret colorimetric method[2,5]. The relative concentration of the individual globulins in these fractions was ascertained by adding them to a known standard serum and observing the alteration in the globulin fraction content of the serum by electrophoresis in an Antweiler microelectrophoresis apparatus. Complement-fixation test techniques were carried out as described in ref. 1, and hæmagglutination tests following the method in refs. 6 and 7.

Considerable variation occurred in the relative amount of total protein in the samples, and antibody contents have therefore been expressed as antibody potency per gm. of protein. Complement-fixation test results have been expressed as 'index ratios' (refs. 2 and 8), which is the ratio of the amount of complement fixed by the serum in the presence of antigen to the amount fixed in the absence of antigen, after corrections, if necessary, have been made for anti-complementary effects of the antigen. Details of the results are shown in Table 1.