

survey. Seventy-three of the organisms were urinary-tract isolates, 14 respiratory tract isolates, and 27 were isolated from other sites. Two-thirds of the 97 patients studied in detail had been in hospital for more than seven days and three-quarters had been on antibiotic therapy before *Serratia marcescens* was first isolated. Details of antibiotics used before the first isolation of *Serratia marcescens*, and of the *in vitro* antibiotic susceptibility of the *Serratia marcescens* isolates would be presented.

#### Factors Affecting Transfer of Antibiotic Resistance between Gram-negative Bacteria in the Human Intestine

J. D. ANDERSON (*University of Bristol*) In the absence of chemotherapy, no transfer of bacterial antibiotic resistance transfer (R) factors could be detected in the faeces of four subjects who swallowed potential donor and recipient organisms even though the plasmids concerned could be freely transferred in broth, both to the ingested potential recipients and to a variety of faecal coliforms. The faeces of these subjects contained such large populations of the relevant organisms that one would have expected transfer to occur if the bacteria had been in a broth medium (Anderson, Gillespie, and Richmond, 1973). Reasons for the discrepancy between results obtained *in vivo* and *in vitro* were therefore investigated.

R factor transfer between donor and recipient strains of *Escherichia coli* was found to be completely inhibited in nutrient broth by dense suspensions of *Bacteroides fragilis*. Comparable amounts of inert bacterial matter (formolized suspensions of *E. coli* or *B. fragilis*), populations of *Streptococcus faecalis*, or bile salts were only moderately inhibitory. Strict anaerobiosis had no effect upon R factor transfer. Population densities of organisms used in these studies were similar to those found in faeces.

The presence of *Bacteroides fragilis* thus provides a satisfactory explanation for the almost total inhibition of conjugation in the human gastrointestinal tract in the absence of antibiotics. Other factors inhibiting conjugation to a lesser degree may reinforce the effect of *B. fragilis*.

#### Reference

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#### Serum Amylase and Related Enzymes in Diabetic Ketoacidosis

D. M. GOLDBERG, R. J. SPOONER, AND A. H. KNIGHT (*Royal Hospital, Sheffield*) In previous studies we have confirmed the high incidence of hyperamylasaemia in diabetic ketoacidosis and have shown that this is not related to acute pancreatitis, renal failure, macroamylasaemia, or hepato-biliary disease (Knight, Williams, Ellis, and Goldberg, 1973; Knight, Williams, Spooner, and Goldberg, 1973) nor does it appear to influence the prognosis in individual cases.

It has recently been proposed that the source of the amylase in such subjects is the hepatocyte, and that amylase is released from its endoplasmic reticulum as a consequence of attack by lysosomal enzymes (Belfiore, Napoli, and Lo Vecchio, 1972; Belfiore and Napoli, 1973). This proposal rested on the demonstration that raised levels of lysosomal enzymes were found in the serum of subjects with diabetic ketoacidosis and hyperamylasaemia and followed a similar time-course to the latter.

The following lines of evidence exclude the hepatocyte as the source of hyperamylasaemia in diabetic ketoacidosis and cast doubt on the role of lysosomes in its release from other tissues.

1 Sequential determinations of serum enzyme activities in 10 consecutive patients revealed that whereas beta-glucuronidase was elevated at some time in all patients, amylase was raised in only eight and acid phosphatase in only four.

2 A low correlation was found between amylase and beta-glucuronidase, and between amylase and acid phosphatase in the above, whether peak activities or activities of all samples were considered.

3 Measurement of the same enzymes in 24 cases of acute viral hepatitis showed that whereas raised beta-glucuronidase activities were found in 20, amylase was raised in only three, and acid phosphatase in but a single case. Again, correlation between amylase and beta-glucuronidase was poor.

4 Analysis of six samples of normal postmortem human liver revealed that, in contrast to acid phosphatase and beta-glucuronidase, its amylase content was negligible, especially when care was taken to remove all pooled blood. In fact the concentration of amylase in

human liver is far below that of human serum, even when steps are taken to ensure solubilization of lysosomal and microsomal enzymes whereas the hepatic content of acid phosphatase and beta-glucuronidase are respectively approximately 30-fold and 6000-fold, the upper normal limit for serum.

#### References

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#### Aspects of EB Virus Infection

R. N. P. SUTTON (*King's College Hospital Medical School, London*, introduced by H. A. SISSONS) The association of the EB virus with Burkitt's lymphoma, infectious mononucleosis, nasopharyngeal carcinoma, and possibly with some other conditions (notably Hodgkin's disease) is now well recognized. Asymptomatic infection with this virus is also frequent and most of the population have acquired antibodies by early adult life.

Although the isolation of EB virus from nasopharyngeal secretions is possible in acute infectious mononucleosis, this procedure is not practicable at the moment as a routine measure and evidence of infection depends upon the demonstration of rising antibody titres. A variety of such antibodies may be demonstrated, including antibodies to virus capsid antigen, membrane, and complement-fixing antibodies. We have observed that antibodies to EB virus capsid antigen develop more rapidly than those to EB soluble complement-fixing antigen and this discrepancy could form the basis for a relatively simple diagnostic test for the presence of recent infection.

Infections with EB virus also result in the development of autoimmune antibodies and in the impairment of cell-mediated immunity. In our report, we describe some of these phenomena in active infectious mononucleosis and also in asymptomatic infections.

#### SYMPOSIUM IN POLYCYTHAEMIA

#### The Assay of Erythropoietin

J. S. MALPAS (*Department of Medical Oncology, St Bartholomew's Hospital, London*) Investigation of erythro-