## June 5, 1965 No. 4988

GSSG via the pentose phosphate pathway is inadequate, the ability to synthesize glutathione impaired, or the sulphydryl stability of haemoglobin decreased; GSH assumes a vital role when either normal or abnormal erythrocytes are exposed to aromatic catalysts, by helping to absorb the vastly increased oxidative potential, and thus with the aid of the process of methaemoglobin formation to buffer against irreversible damage.

It is recognized that this unified concept may fail to account for possible contributory factors. The ideas are put forward in order to direct attention to the selectivity of different forms of oxidant haemolysin, and to emphasize the variable significance of the protective role of GSH in different types of human erythrocyte placed under varying degrees and types of oxidative stress.

J. D. HARLEY

- Children's Medical Research Foundation, Royal Alexandra Hospital for Children, Camperdown, Sydney.
- <sup>1</sup> Jandl, J. H., Engle, L. K., and Allen, D. W., J. Clin. Invest., **39**, 1818 (1980).
- <sup>2</sup> Beutler, E., Nature, 196, 1095 (1962).
- <sup>8</sup> Beutler, E., *Blood*, **14**, 103 (1959). <sup>4</sup> Allen, D. W., and Jandl, J. H., *J. Clin. Invest.*, **40**, 454 (1961).
- <sup>6</sup> Harley, J. D., and Mauer, A. M., *Blood*, **17**, 418 (1961). <sup>6</sup> Harley, J. D., and Robin, H., *Nature*, **193**, 478 (1962).
- <sup>7</sup> Harley, J. D., and Robin, H., Austral. J. Exp. Biol. Med. Sci., **41**, 281 (1963).
- <sup>8</sup> Jacob, H. S., and Jandl, J. H., J. Clin. Invest., 41, 779 (1962).
- <sup>9</sup> Jacob, H. S., and Jandl, J. H., J. Clin. Invest., 41, 1514 (1962).
- <sup>16</sup> Harley, J. D., M.D. thesis, University of Sydney (1964).
- <sup>11</sup> Waller, H. D., Sc 37, 898 (1959). , Schlegel, B., Müller, A. A., and Löhr, G. W., Klin. Wschr.,
- <sup>12</sup> Brewer G. J., Tarlov, A. R., Kellermeyer, R. W., and Alving, A. S., J. Lab. Clin. Med., **59**, 905 (1962).
  <sup>13</sup> Prankerd, T. A. J., J. Physiol., **143**, 325 (1958).
- <sup>14</sup> Bonsignore, A., Fornaini, G., Fantoni, A., Leoncini, G., and Segni, P., J. Clin. Invest., 43, 834 (1964).
- <sup>15</sup> Marks, P. A., Johnson, A. B., and Hirschberg, E., Proc. U.S. Nat. Acad. Sci., 44, 529 (1958).
- <sup>16</sup> Iohr, G. W., Waller, H. D., Karges, O., Schlegel, B., and Müller, A. A., Klin. Wschr., 36, 1008 (1958).
  <sup>17</sup> Rigas, D. A., and Koler, R. D., J. Lab. Clin. Med., 58, 417 (1961).
- <sup>18</sup> Brewer, G. J., Tarlov, A. R., and Kellermeyer, R. W., J. Lab. Clin. Med., 58, 217 (1961).
- <sup>19</sup> Rigas, D. A., and Koler, R. D., Blood, 18, 1 (1961).
- 20 Oort, M., Loos, J. A., and Prins, H. K., Vox Sang., 6, 370 (1961).
- <sup>21</sup> Allison, A. C., Nature, 188, 37 (1960).
- <sup>21</sup> Brewer, G. J., and Powell, R. D., Nature, 199, 704 (1963).
  <sup>22</sup> Grimes, A. J., Nature, 198, 1312 (1963).
- 24 Anderson, H. M., and Turner, J. C., J. Clin. Invest., 39, 1 (1960).

## PATHOLOGY

## Agar Electrophoresis of Antibiotics produced by Serratia marcescens

INDIVIDUAL strains of Serratia marcescens may liberate two different bacteriocins<sup>1</sup>. The distinction is made on grounds of temperature of inactivation and susceptibility to various other destructive agents coupled with distinctive spectra of activity. During an investigation of antibiotics produced by Serratia the foregoing phenomenon was also encountered and electrophoretic separation of the components was attempted. Strains, media and electrophoretic techniques were those previously used<sup>2,3</sup>. In addition, twenty strains isolated locally and numbered Sm 16-19 and Sm 37-52 were also investigated. All 51 Serratia strains could be differentiated on grounds of lysogenicity and phage susceptibility Antibiotic activity was detected by the method of Fredericq<sup>4</sup> or by spotting the clear supernatants of overnight growths of cultures induced by ultra-violet light on plates freshly spread with indicator organisms.

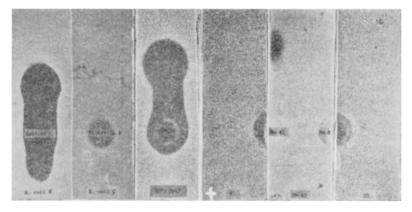


Fig. 1. Antibiotics of Serratia strains were spotted at intervals across the middle of an electrophoresis plate and a potential of 50 V applied for 18 h. The plate was then divided into 6 compartments with glass strips, and after sterilization with chloroform vapour the compartments were flooded with appropriate indicator organisms and incubated overnight. The fourth and fifth glass strips from the left bisected the original area of spotting. The indicator strains are identified at the foot of the comparisms MCTC 2847 and Sm 42 are Serratia and H1 is a strain of Hafnia

On electrophoresis, 17 of the strains produced a pattern (Fig. 1, compartments 4 and 5 from left) which consisted of a fraction A which migrated to the cathode and was bactericidal for various Serratia strains, and a stationary component B which was inactive on Serratia but was detected by its action on Escherichia coli  $\varphi$ , Hafnia or Aerobacter strains. Despite the fact that the various A fractions could be divided into eight sub-groups by their action on Serratia mutants resistant to other A agents, only the A component of strain Sm 41 possessed a slightly different mobility. The antibiotic of strain Sm 52 produced a different electrophoretic pattern (Fig. 1, compartment 3). Like the  $\hat{A}$  fractions, it only killed other Serratia. Nineteen strains only produced electrophoretically non-mobile bactericidal substances. These stationary substances could be divided into two groups according to their spectra of activity. Group (i) substances were produced by 14 strains and had a range of activity identical with that of the B components mentioned here (fifth and sixth compartments, Fig. 1), while group (ii) antibiotics (produced by five strains) were active on a wide range of organisms including some Gram-positive species. The group (i) substances and the B agents appear to comprise a single bacteriocin in so far as mutants of indicator strains resistant to any one of them were cross-resistant to all other agents of these groups. Judged on the same grounds, the antibiotics of group (ii) also constituted a separate single entity. Fourteen of the 51 Serratia strains did not produce detectable antibiotics.

A bacteriocin produced by S. marcescens which has a spectrum of activity which corresponds to fraction Bdescribed here has been classified as a sub-species of colicin k (ref. 5). The reason is that mutants of an E. coli strain resistant to the *Serratia* bacteriocin are simultaneously resistant to colicin k. We also observed that mutants of E. coli  $\varphi$  resistant to B components were resistant to colicin k. As illustrated in Fig. 1, a nice distinction, based on electrophoretic mobility, was possible between colicin k and the B type Serratia bacteriocins.

This work was aided by grants from the South African Council for Scientific and Industrial Research.

> Hèléne E. Prinsloo I. J. MARÉ

J. N. COETZEE

Department of Microbiology, University of Pretoria.

- <sup>1</sup> Hamon, Y., and Peron, Y., Ann. Inst. Pasteur, 100, 818 (1961).
- <sup>2</sup> Prinsloo, H. E., and Coetzee, J. N., Nature, 203, 211 (1964).
- <sup>8</sup> Maré, I. J., Coetzee, J. N., and de Klerk, H. C., Nature, 202, 213 (1964).
- <sup>4</sup> Fredericq, P., Annu. Rev. Microbiol., 11, 7 (1957).
- <sup>5</sup> Mandel, M., and Mohn, F. A., Abstr. Eighth Intern. Congr. Microbiol., 26 (Montreal, 1962).