References

- ¹ Schmid-Schonbein H, Wells RE, Goldstone J. Fluid drop-like behaviour of erythrocytes—disturbance in pathology and its quantification. *Biorheology* 1971;**7**:227-34.
- ² Goldsmith HL. Microrheology of red blood cell suspension. *J Gen Physiol* 1968;**52**:55.
- ³ Chien S, Usami S, Bertler JF. Abnormal rheology of oxygenated blood in sickle cell anemia. *J Clin Invest* 1970;**49**:623-34.
- ⁴ Murphy JR. The influence of pH and temperature on some physical properties of normal erythrocytes and erythrocytes from patients with hereditary spherocytosis. *J Lab Clin Med* 1967;**69**:756-75.
- ⁵ Dintenfass L. *Rheology of blood in diagnostic and preventive medicine*. London: Butterworths, 1976:137-42.
- ⁶ Kannel WB, Gordon T, Wolf PA, McNamara P. Haemoglobin and the risk of cerebral infarction: the Framingham study. *Stroke* 1972;**3**:409-20.
- ⁷ Pearson HA, Johnston D, Smith KA, Touloukian RJ. The born-again spleen. *N Engl J Med* 1976;**25**:1389-592.
- ⁸ Lux SE, John KM. Isolation and partial characteristics of a high molecular weight red cell membrane protein complex normally removed by the spleen. *Blood* 1977;**50**:625-41.
- ⁹ Ellis BC, Stransky A. A quick and accurate method for the detection of fibrinogen in plasma. *J Lab Clin Med* 1961;**58**:477-88.
- ¹⁰ Buchan PC. Evaluation and modification of whole blood filtration in the measurement of erythrocyte deformability in pregnancy and the newborn. *Br J Haematol* 1980;**45**:97-105.
- ¹¹ Likhite VV. Immunological impairment and susceptibility to infection after splenectomy. *JAMA* 1976;**236**:1376-7.
- ¹² Robinette CD, Fraumeni JF. Splenectomy and subsequent mortality in veterans of the 1939-45 war. *Lancet* 1977;ii:127-9.
- ¹³ Mayer GA. Blood viscosity in healthy subjects and patients with coronary artery disease. *Can Med Assoc J* 1964;**91**:951-4.
- ¹⁴ Anonymous. Red-cell deformability. *Lancet* 1978;iii:1348-9.
- ¹⁵ Crosby WH. Splenic remodelling of red cell surfaces. *Blood* 1977;**50**:643-5.
- ¹⁶ Nathan DG. Rubbish in the red cell. *N Engl J Med* 1969;**281**:558-9.
- ¹⁷ Lipson RL, Bayrd ED, Watkins CH. The post-splenectomy blood picture. *Am J Clin Pathol* 1959;**32**:526-32.
- ¹⁸ Stuart S, Kenny MW. Blood rheology. *J Clin Pathol* 1980;**33**:417-29.

(Accepted 22 June 1981)

Increase in drug resistance among *Shigella dysenteriae*, *Sh flexneri*, and *Sh boydii*

R J GROSS, B ROWE, T CHEASTY, LINDA V THOMAS

Abstract

Two thousand three hundred and seventy strains of *Shigella dysenteriae*, *Sh flexneri*, and *Sh boydii* isolated in England and Wales from 1974 to 1978 were tested for resistance to 12 antimicrobial drugs. Eighty per cent of strains were resistant to one or more drugs, with sulphonamide resistance occurring most frequently. Resistance to streptomycin, tetracycline, ampicillin, and chloramphenicol increased during the period, as did the incidence of multiple resistance.

Most infections due to *Sh dysenteriae*, *Sh flexneri*, and *Sh boydii* are acquired abroad, and the increasing incidence of drug resistance among these organisms contrasts with the decreasing incidence of resistance among the indigenous *Sh sonnei*. These findings may indicate the need for better control of antibiotic use, particularly in developing countries.

Introduction

The number of *Shigella dysenteriae*, *Sh flexneri*, and *Sh boydii* isolations in England and Wales is small when compared with that of *Sh sonnei*. Nevertheless, the proportion of infections due to these subgroups increased from 2.4% of all shigella infections in 1965¹ to 16% in 1978.²

There is little published information on the incidence of drug resistance in *Shigella* in the United Kingdom, although the antibiotic resistance of *Sh sonnei* strains isolated in London has

been documented.³ We describe here the occurrence of resistance in *Sh dysenteriae*, *Sh flexneri*, and *Sh boydii* isolated in England and Wales from 1974 to 1978.

Methods

Bacterial strains—Two thousand three hundred and seventy strains of *Sh dysenteriae*, *Sh flexneri*, and *Sh boydii* isolated in England and Wales from human faeces during the period 1974-8 were examined in the Division of Enteric Pathogens, Central Public Health Laboratory, London. *Sh sonnei* strains are not referred to this laboratory. All the strains were identified as members of the genus *Shigella* by the methods of Edwards and Ewing⁴ and were serotyped according to the internationally accepted scheme.⁵

Drug resistance tests—The shigella strains were tested by an agar plate dilution method⁶ for resistance to the following concentrations of antibacterial drugs: ampicillin (10 mg/l), cephaloridine (30 mg/l), chloramphenicol (30 mg/l), gentamicin (30 mg/l), nalidixic acid (30 mg/l), neomycin (30 mg/l), furazolidone (30 mg/l), polymixin B (100 U/ml), streptomycin (30 mg/l), sulphonamides (250 mg/l), tetracycline (10 mg/l), trimethoprim (1.25 mg/l).

Results

Of the 2370 shigella strains examined, 194 were *Sh dysenteriae*, 1867 *Sh flexneri*, and 309 *Sh boydii*. One thousand eight hundred and ninety-five strains (80%) were resistant to one or more of the antimicrobial drugs, while 782 strains (33%) were resistant to three or more drugs.

The prevalence of resistance to each drug is summarised in table I. Resistance to sulphonamides was most common (75.7%), followed by resistance to streptomycin (57.6%), tetracyclines (36%), chloramphenicol (12.5%), and ampicillin (12%). The proportion of strains resistant to these five drugs increased during 1974-8, as shown in table II. Resistance to cephaloridine, trimethoprim, gentamicin, and nalidixic acid was rare (<1%). All strains were sensitive to polymixin B.

The most common resistance patterns were SSu (25% of all strains), SSuT (17%), and Su (16%).

Division of Enteric Pathogens, Central Public Health Laboratory, London NW9 5HT

R J GROSS, MA, MSC, principal microbiologist

B ROWE, MB, MRCPATH, director

T CHEASTY, BSC, senior microbiologist

LINDA V THOMAS, BSC, microbiologist

TABLE I—Drug resistance in *Shigella* strains isolated in England and Wales 1974-8

	% Of strains resistant			Total
	<i>Sh dysenteriae</i>	<i>Sh flexneri</i>	<i>Sh boydii</i>	
Ampicillin	7.7	13.8	5.2	12.0
Cephaloridine	0.5	0.9	0.3	0.8
Chloramphenicol	17.0	13.7	2.9	12.5
Gentamicin	0.5	0.05	0	0.1
Nalidixic acid	0	0.05	0	0.04
Neomycin	1.5	2.3	2.3	2.2
Furazolidone	1.0	1.2	1.6	1.3
Polymixin B	0	0	0	0
Streptomycin	71.6	54.3	68.6	57.6
Sulphonamides	77.8	76.7	68.6	75.7
Tetracycline	33.5	37.6	28.2	36.0
Trimethoprim	0	0.3	0.3	0.3
No of strains tested	194	1867	309	2370

TABLE II—Increase in drug resistance in *Sh dysenteriae*, *Sh flexneri* and *Sh boydii* isolated in England and Wales 1974-8

	% Of strains resistant				
	1974	1975	1976	1977	1978
Sulphonamides (250 mg/l)	68	75	75	79	78
Streptomycin (30 mg/l)	43	49	56	64	66
Tetracycline (10 mg/l)	15	26	35	42	51
Ampicillin (10 mg/l)	2	3	10.5	15.5	23.7
Chloramphenicol (30 mg/l)	2.6	5.4	10.2	15.2	18.5

Discussion

This study shows that over 80% of *Sh dysenteriae*, *Sh flexneri*, and *Sh boydii* were antibiotic resistant. Ampicillin resistance increased from 2% in 1974 to 23.7% in 1978. The incidence of resistance to chloramphenicol, tetracycline, and streptomycin also increased during this period, while resistance to sulphonamides remained high throughout. In most strains these resistances have been shown to be plasmid-determined (report in preparation).

Although *Sh dysenteriae*, *Sh flexneri*, and *Sh boydii* account for only 16% of all shigella infections in England and Wales²

such infections may be severe, possibly requiring admission to hospital and antibiotic treatment.⁷ Multiple drug resistance—that is, resistance to three or more antimicrobial drugs—has, however, increased in these organisms from 13% in 1974 to 49% in 1978. In contrast, Davies has found that the proportion of *Sh sonnei* strains with multiple transferable drug resistance decreased from 38% in 1972 to 8% in 1977 (J Davies, personal communication).

In a previous study⁸ we showed that at least 52% of infections due to *Sh dysenteriae*, *Sh flexneri*, and *Sh boydii* in England and Wales were acquired abroad. Visitors to the Indian subcontinent accounted for 44% of infections with these organisms, and this may reflect the extent of travel to this area by members of the immigrant community. A further 17.6% were acquired in the Mediterranean countries of North Africa and were usually infections among tourists visiting holiday resorts. The increasing incidence of drug resistance in *Sh dysenteriae*, *Sh flexneri*, and *Sh boydii* strains originating abroad is in sharp contrast to the decreasing incidence of resistance among the indigenous *Sh sonnei*. These findings may indicate a need for greater control of antibiotic use in certain areas of the world, particularly in the developing countries.

References

- 1 Chief Medical Officer of the Ministry of Health. *Annual report for the year 1965*. London: HMSO, 1966.
- 2 Office of Population Censuses and Surveys. *Statistics of infectious diseases, December quarter 1978*. London: HMSO, 1979.
- 3 Davies JR, Farrant WN, Uttley AHC. Antibiotic resistance of *Shigella sonnei*. *Lancet* 1970;ii:1157-9.
- 4 Edwards PR, Ewing WH. *Identification of enterobacteriaceae*. Minneapolis: Burgess Publishing Company, 1972.
- 5 Subcommittee on Taxonomy of the Enterobacteriaceae. Report. *International Journal of Systemic Bacteriology* 1958;8:95.
- 6 Haltalin KC, Markley AH, Woodman E. Agar plate dilution method for routine antibiotic susceptibility testing in a hospital laboratory. *Am J Clin Pathol* 1973;60:384-94.
- 7 Rowe B, Gross RJ, Allen HA. *Shigella dysenteriae* and *Shigella boydii* in England and Wales during 1972 and 1973. *Br Med J* 1974;iv:641-2.
- 8 Gross RJ, Thomas LV, Rowe B. *Shigella dysenteriae*, *Sh flexneri*, and *Sh boydii* infections in England and Wales: the importance of foreign travel. *Br Med J* 1979;ii:744.

(Accepted 2 July 1981)

THE LESSER CELANDINE, USUALLY KNOWN BY THE NAME OF PILEWORT AND FOGWORT. I wonder what ailed the ancients to give this the name Celandine, which resembles it neither in nature nor form; it acquired the name of Pilewort from its virtues, and it being no great matter where I set it down, so I set it down at all, I humoured Dr Tradition so much, as to set him down here.

This Celandine or Pilewort (which you please) doth spread many round pale green leaves, set on weak and trailing branches which lie upon the ground, and are flat, smooth, and somewhat shining, and in some places (though seldom) marked with black spots, each standing on a long foot-stalk, among which rise small yellow flowers, consisting of nine or ten small narrow leaves, upon slender foot-stalks, very like unto Crowsfoot, whereunto the seed also is not unlike being many small kernels like a grain of corn sometimes twice as long as others, of a whitish colour, with fibres at the end of them.

It grows for the most part in moist corners of fields and places that are near water sides, yet will abide in drier ground if they be a little shady. It flowers betimes, about March or April, is quite gone by May; so it cannot be found till it spring again.

It is under the dominion of Mars, and behold here another verification of the learning of the ancients, viz that the virtue of an herb may be known by its signature, as plainly appears in this; for if you dig up the root of it, you shall perceive the perfect image of the disease which they commonly call the piles. It is certain by good experience, that the decoction of the leaves and roots wonderfully helps piles and

haemorrhoids, also kernels by the ears and throat, called the king's evil, or any other hard wens or tumours.

Here's another secret for my countrymen and women, a couple of them together; Pilewort made into an oil, ointment, or plaister, readily cures both the piles, or haemorrhoids, and the king's evil: The very herb borne about one's body next the skin helps in such diseases, though it never touch the place grieved; let poor people make much of it for those uses; with this I cured my own daughter of the king's evil, broke the sore, drew out a quarter of a pint of corruption, cured without any scar at all in one week's time. (Nicholas Culpeper (1616-54) *The Complete Herbal*, 1850.)

Correction

Evidence for defect of complement-mediated phagocytosis by monocytes from patients with rheumatoid arthritis and cutaneous vasculitis

We regret that three errors occurred in the paper by Dr N P Hurst and Professor G Nuki (27 June, p 2081). The first sentence of the abstract and the seventh and eighth sentences of the third paragraph of "Patients and methods" (starting at the last line of p 2081) should refer to human serum *in* (not *from*) patients, etc (see letter by the authors in this week's issue).