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

	% Of strains resistant			
	Sh dysenteriae	*Sh flexneri*	*Sh boydii*	Total
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 Wales[2] 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 study[8] 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.

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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 neather 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).