The preparations were cultured for lactobacilli on MRS agar (Unipath, Basingstoke, Hampshire), for enterococci on m-enterococcus agar (Difco, East Molesey, Surrey), and for anaerobes on Columbia agar (Unipath) plus 5% whole horse blood. Either one whole tablet was crushed in a sterile mortar or the content of a capsule (opened under aseptic conditions) or a weighed amount of powder (approximately 500 mg) was suspended in 10 ml of MRS broth, blended with a Vortex mixer for 1 minute, and allowed to stand for 30 minutes; viable counts were made from the supernatant fractions. Plates were incubated for 48 hours at 37°C. As many colony types as possible on each medium were counted and identified with 50CH, API Strep 20, and API rapid ID 32A kits (bio-Mérieux, Basingstoke, Hampshire), as indicated.

Only two products (B and H) matched their labelled microbiological specifications qualitatively and quantitatively (table). The 11 other brands did not contain *L. acidophilus*, contained extra species, or lacked a listed species, or numbers were less than a tenth of those stated. Four brands (C, E, G, and K) contained bacterial species not stated on the label (*Enterococcus faecium* in two, *Pediococcus pentosaceus* in two). Five brands (A, F, J, K, and L) did not contain the listed *L. bifidus* (which was reclassified as a *Bifidobacterium* sp (an obligate anaerobe) more than 20 years ago.³ Oral probiotics may be beneficial in certain specific conditions—for example, *Saccharomyces boulardii* or *B. bifidum* plus *Streptococcus thermophilus* in diarrhoea.^{4,5} But claims made on the labels of some of the products tested here—"maintains a healthy

digestion," "improves digestibility and assimilation of food," "keeping the intestinal contents sweet," "assist . . . general well-being," "protects skin . . . against harmful microbes," "builds immunity"—are not supported. The *British National Formulary* states that "lactobacillus preparations are valueless" in acute gastrointestinal infections.

In conclusion, the public and health professionals should be aware that the labelling of some probiotics may be misleading, in terms of both the microbiological content and possible beneficial effects.

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Microbiological characterisation of 13 probiotics

Brand	Formulation	Composition as stated on label*	Composition when analysed
A	Capsule	$>5 \times 10^9$ { <i>Lactobacillus bifidus</i> † <i>L. acidophilus</i> <i>L. rhamnosus</i> <i>Streptococcus faecium</i> ‡	Not detected Not detected 2 strains of <i>L. rhamnosus</i> <i>E. faecium</i>
B	Capsule	4×10^9 <i>L. acidophilus</i>	4.7×10^9 <i>L. acidophilus</i>
C	Capsule	$>1 \times 10^9$ <i>L. acidophilus</i>	5.1×10^9 <i>L. delbreuckii</i> 5×10^9 <i>Enterococcus faecium</i>
D	Capsule	2×10^7 <i>L. acidophilus</i>	1×10^4 <i>L. acidophilus</i>
E	Powder	$>1 \times 10^{10}$ <i>L. acidophilus</i>	2.2×10^9 <i>L. rhamnosus</i> 1.4×10^9 <i>E. faecium</i>
F	Tablet (enteric coated)	$>5 \times 10^7$ <i>L. acidophilus</i> $>5 \times 10^7$ <i>L. bifidus</i>	1.5×10^9 <i>L. actobacillus</i> spp Not detected
G	Tablet	500 mg§ <i>L. acidophilus</i>	7.8×10^7 <i>L. acidophilus</i> 4.8×10^7 <i>L. fermentum</i> 1×10^7 <i>Pediococcus pentosaceus</i>
H	Capsule	6.4×10^9 <i>L. acidophilus</i>	1.2×10^7 <i>L. acidophilus</i>
I	Capsule	$>2 \times 10^9$ <i>L. acidophilus</i> # R	5.4×10^7 <i>L. acidophilus</i> 8.4×10^7 <i>Lactobacillus</i> spp
J	Capsule	2×10^9 { <i>L. acidophilus</i> <i>L. rhamnosus</i> <i>L. bifidus</i> <i>S. faecium</i>	Not detected 2 strains of <i>L. rhamnosus</i> Not detected <i>E. faecium</i>
K	Capsule	2.5×10^9 { <i>L. acidophilus</i> <i>Bifidobacterium bifidum</i>	3×10^7 <i>L. acidophilus</i> Not detected 9×10^7 <i>L. plantarum</i> 4.9×10^4 <i>Pediococcus pentosaceus</i>
L	Capsule	2×10^9 { <i>L. acidophilus</i> <i>L. rhamnosus</i> <i>S. faecium</i> <i>L. bifidus</i>	5×10^9 <i>L. acidophilus</i> 2.9×10^9 2 strains of <i>L. rhamnosus</i> 6×10^9 <i>E. faecium</i> Not detected
M	Capsule	5×10^7 { <i>B. breve</i> <i>B. longum</i> <i>S. thermophilus</i> <i>L. rhamnosus</i> <i>L. acidophilus</i> <i>L. casei</i> subsp <i>casei</i> <i>L. bulgaricus</i>	8.6×10^6 { 2 strains of <i>Bifidobacterium</i> sp <i>S. thermophilus</i> <i>L. rhamnosus</i> <i>L. acidophilus</i> <i>L. paracasei</i> Not detected

*Bacterial count per capsule, tablet, or gram, as appropriate.

†Reclassified as *Bifidobacterium* sp (see text).

‡This species has been renamed *Enterococcus faecium*.

§Not helpful: 500 mg of lactobacilli = 10^{12} bacteria.

Deaths in police custody

EDITOR,—During the past 12 months 15 deaths have occurred in police custody in Britain, according to the national police authorities. Forensic investigation has comprised at least a postmortem examination in all cases, and in only one case is prosecution of the police officer concerned pending. It therefore seems that in some circumstances a pathological entity exists that predisposes subjects in police custody to certain forms of natural death. Underlying cardiomyopathies, cardiac valve lesions, and cardiac conduction defects have all been mooted as causes of death. Among potential hypotheses to explain this phenomenon are supranormal secretion of catecholamines and supranormal surges in blood pressure due to the psychological and physical stress of police custody.

I believe that a prospective confidential inquiry into the cause of deaths in police custody should be set up. It is imperative that the BMA is not perceived by the public to be in complicity with the police authorities on this issue. The effect of such a perception on the South African Medical Association in the 1980s was all too evident. We need to ascertain and document the cause of death in all cases. Such an inquiry could serve to identify a hitherto unknown clinical syndrome and allow preventive measures to be taken against deaths in police custody.

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Patients taking stable doses of morphine may drive

EDITOR,—A guide for medical practitioners published by the Medical Commission on Accident Prevention, *Medical Aspects of Fitness to Drive*, has been widely distributed within the medical profession.¹ In a section on drugs acting on the central nervous system it states that "the more powerful narcotic analgesics such as morphine produced marked sedation and patients requiring them should not drive." This has important implications for the quality of life of patients receiving palliative care who require regular doses of morphine to control pain.

Experience in palliative care indicates that the symptoms that occur at the start of treatment with morphine generally resolve within a few days of the dose being stabilised. It is commonly believed, therefore, that patients taking a stable dose may drive without hazard to themselves or other road users. There are, however, few objective data confirming this belief.

Vainio *et al* have recently reported the results of psychological and neurological tests designed for professional drivers of motor vehicles that were conducted in two groups of patients with cancer. Twenty four of the patients were taking slow release morphine orally at regular intervals to control cancer pain, and 25 were free of pain without taking regular analgesics. The mean daily dose in the treated patients was 209 mg (range 60-1100 mg) and had been stable for at least two weeks. The results showed that long term, stable, treatment with morphine at these doses had only a slight and selective effect on functions related to driving and that this effect would not be hazardous in traffic. Hanks, commenting on these findings, suggested reasons why long term morphine for pain should not result in the same degree of sedation that results from single doses in people who do not take opioids regularly.

These findings support the impression of most palliative care physicians that driving need not be