MIXED BAG

Race, ethnicity and medical research

A recent paper in the *British Medical Journal* once again uses 'race' as a scientific and not a social construct. Sarah McDowell and her colleagues, from Birmingham in the UK, carried out a systematic review and meta-analysis of ethnic differences in the risks of adverse reactions to drugs used in cardiovascular medicine. Their conclusion: patients from different ethnic groups have different risks for important adverse drug reactions to cardiovascular drugs and that these differences in the responses of different ethnic groups should be taken into consideration when a drug is licensed. They identified papers for their analysis by mention of ethnicity, ethnic groups or racial groups. At this stage in our understanding of the human genome and of genomics, one must ask two questions: why did the authors carry out this study at all and why did the editor of the *BMJ* publish it?

When I was a second-year medical student in 1985, Alan Morris, senior lecturer in anatomy, told us unequivocally that the human population is so heterogeneous that we all share an enormous amount of our genes, across and within populations – making the concept of race scientifically meaningless. The same applies to the so-called racial differences in susceptibility to different chronic diseases and reactions to drugs, which has resulted in the promotion of a race-specific 'niche market' by the ever-hungry multinational pharmaceutical companies.

In 2003 geneticists were questioning the notion of ethnic differences in disease susceptibility and reactions to drugs. At the end of the 1990s randomised trials were interpreted to show that a combination of vasodilators is more effective in treating heart failure in black people than in white people and that angiotensin-converting enzyme (ACE) inhibitors have little efficacy in blacks. However, when examined more closely it became apparent that the results of the vasodilator trials were inconsistent and never achieved statistical significance for an interaction between treatment and race. And another analysis of the data from the ACE-inhibitor trial showed that the original result, showing a racial difference, was unique to the end point chosen – in the portion of the trial focusing on prevention, the drug was equally effective in blacks and whites in reducing the incidence of the combined end point of death or development of new-onset heart failure. And yet, in the USA there is a drug called BiDil – a combination of hydralazine and isosorbide dinitrate – that is marketed as being more effective in certain ethnic groups.

The promotion of a drug for a race-specific niche is potentially dangerous, because, as Richard Cooper and colleagues point out, in a paper in the *New England Journal of Medicine* in 2003, this could distract people from therapies which we know work in certain conditions. The idea behind race-specific therapy is a presumption that the frequencies of genetic variants that influence the efficacy of the drug are substantially different among races. But this has not been unequivocally demonstrated for any class of drugs if the way in which ethnicity is chosen is carefully examined. Race may help to target screening for a disease-associated mutation (such as sickling trait) that is present in high frequency in one population, but almost absent in another. But – and this is very important – 'it is impossible for race as we recognise it clinically to provide both perfect sensitivity and specificity for the presence of a DNA-sequence variant'. So race is never an adequate proxy for choosing a drug – only genotype testing will provide this information. And the cluster of genes that can place people very accurately into populations coming from particular geographical locations are unlikely to be functional – in the words of Cooper *et al.* the clusters are similar to a last name – they simply give the geographical location of a person's origins.

If we are seriously trying to make medicine more applicable for individuals, and that is one of the goods that is potentially going to come from our understanding of the human genome (in the developed world at least), then we have to start understanding what genetics and genomics (the study of all the nucleotide sequences in a chromosome) are all about and use this, and not preconceived ideas about what race and ethnicity mean. Cooper *et al.* point out that people of 'African ancestry' have as wide a variation in rates of hypertension and diabetes as any other large continental population. But as long as we try to look only at 'race' we are in danger of forgetting about the other determinants of disease – social circumstances, diet, levels of education and all the other variables that determine sickness and health in populations.

Cooper S, *et al. NEJM* 2003; **348:** 1166-1170.
McDowell SE, *et al. BMJ* 2006; **332:** 1177-1181.

Family meals and old age

According to some, getting old is hard. But others seem to manage to continue to enjoy life as they age and a characteristic of these happy souls seems to be the ability to maintain their independence for as long as possible. The burgeoning numbers of retirement villages in South Africa attest to people's need to continue to live, as far as they possibly can, in their own home until death. But this is generally an expensive option and there are those who simply cannot afford that luxury. So, making the more traditional old-age home as friendly as possible is important. A study in the Netherlands looked at the effect of family-style mealtimes on quality of life, physical performance and body weight of nursing home residents. As the authors point out, those living in old-age homes lose their privacy, their independence, often their spouse and a familiar environment. These are all factors that can lead to loneliness and depression and a poor quality of life. Mealtimes provide one opportunity

to integrate and implement physical care measures that improve quality of life. Eating is a social event and residents can interact with others in the home, and both residents and staff have a choice over what they want to eat and when they want to relax.

Most Dutch nursing homes apparently offer two types of care: psychogeriatric care for those with dementia or chronic care for patients with conditions such as stroke or Parkinson's disease. These two groups of residents live in separate wards, with about 30 patients per ward. Traditional wards have 3 or 4 single rooms, 4 - 6 double rooms, and 4 dormitories for 4 people each. However, many of these facilities are undergoing major reorganisation to offer residents their own room and better care generally. Family style mealtimes are part of this reorganisation.

The authors of the paper randomised 178 residents of 5 Dutch nursing homes into 95 elderly people who received family-style meals and 83 who received the usual pre-plated meals individually. None of the patients were demented. The family-style meal intervention included a nicely set table, with proper glassware and cutlery, a choice of two types of meat and vegetables served at the table and no pre-prepared sandwiches. Staff had to sit at the table and chat with residents and drugs were handed out at the start of the meal. About 6 residents were seated per table and they served themselves, unless they needed help from the staff. There were no other activities taking place, such as doctors' visits, cleaning, laundry delivery and visits during the meal, which did happen during mealtimes for residents served with pre-plated meals.

Perhaps, not surprisingly, they found that those who were able to sit around a table with other people had significant improvement in their overall quality of life, fine motor function and body weight. Such a simple intervention to provide a far better environment for people at the end of their lives.

Nijs K, *et al*. *BMJ* 2006; **332:** 1180-1184.

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