

NHS to come under Race Relations Act

John Warden,
parliamentary correspondent, BMJ

Race relations laws in the United Kingdom are to be enforced more widely in the public sector, including the NHS, the UK government decided last week in the wake of a damning official report on "institutional racism" in the police service.

The Stephen Lawrence inquiry into the racist murder of a black teenager in South London in 1993 found that collective racism exists in both the Metropolitan Police—which failed to secure convictions—and other institutions countrywide. As a result, the home secretary, Jack Straw, announced an intention to extend the Race Relations Act to cover all public services, which are at present exempt.

The Race Relations Act 1976 makes racial discrimination unlawful in employment, training, and the provision of facilities and services. The Commission for Racial Equality conducts investigations and can make organisations change the way they operate. It issues codes of



Home secretary Jack Straw is extending the Race Relations Act to the NHS

practice and takes legal action over racially discriminatory job advertisements.

A new law to be introduced as soon as possible will allow the Commission for Racial Equality to investigate what is happening within the public services. The law would support those who were shown to have suffered racial discrimination.

The government plans a comprehensive bill to bring the public sector as a whole within the Race Relations Act. If the Lawrence report's recommendations to the police service are followed in the NHS, chief officers would be made liable for acts and omission of their subordinates under the race laws.

The report's definition of institutional racism, which Mr Straw accepts, is "the collective failure of an organisation to provide an appropriate and professional service to people because of their colour, culture, or ethnic origin." The definition covers also "unwitting prejudice" and racist stereotyping, which disadvantage people from ethnic minorities.

Accusations of bias against staff from ethnic minorities in the NHS are made regularly, most recently concerning the distribution of merit awards to consultants. It is one of the issues to be covered in a fundamental review of distinction awards due later this year (*BMJ* 1998; 317:429). (see p 616) □

Surgery increases risk of sporadic CJD

Zosia Kmietowicz, *London*

Surgery could be the leading risk factor for the development of the sporadic form of Creutzfeldt-Jakob disease (CJD), suggests an Australian study showing that people who have had more than three operations are twice as likely to contract the disease as those who have had no surgery.

The finding comes from a study carried out by the Australian National Creutzfeldt-Jakob Disease Registry at the University of Melbourne, designed to identify the risk factors for sporadic CJD (*Lancet* 1999; 353:693-7). The cause of sporadic CJD is unknown, whereas new variant CJD is linked to bovine spongiform encephalopathy (BSE) and

is specific to the United Kingdom. Although very rare, occurring at a rate of about one in a million throughout the world, sporadic CJD is the more common of the two types of disease and accounts for about 85-90% of all cases.

The Australian study compared the medical history and characteristics of 241 definite and probable cases of CJD that occurred between 1970 and 1997, with 784 volunteers matched as case controls. They found a significant association between surgical procedures and the development of sporadic CJD. This risk increased with the number of surgical treatments, doubling in people undergoing more than three

operations from one in a million to two in a million.

Another risk factor linked to increased risk of sporadic CJD was residence or employment on a farm or market garden for more than 10 years. Blood transfusions, organ transplantation, and major dental work were not linked to increased risk.

Professor Colin Masters, who heads the Australian registry, said that the group was still uncertain how surgical episodes may cause contamination leading to increased risk of sporadic CJD. He speculated that dental work carried negligible risk of transmitting infection because it involved minimal exposure to infectious tissue.

Commenting on the implications of the findings, Professor Masters concluded: "Our research indicates that continual vigilance in the hospital setting may be important in reducing the risk of sporadic CJD." □

Autoimmune reaction links chlamydia to heart disease

Abi Berger, *BMJ*

Researchers have found that the link between chlamydial infections and heart disease involves an autoimmune response in the infected host.

According to a study published in the journal *Science* (1999;283:1335-8), researchers from the University of Toronto have discovered that a surface protein on the chlamydia bacterium mimics a peptide found in mouse myosin. This "molecular mimicry" allows the bacteria to invade the mouse without necessarily triggering an immune response in the host. However, the researchers observed that sometimes the host's immune system does recognise the bacteria as "foreign" and, in addition to triggering an immune response against the bacteria, the host mouse begins to attack its own myosin. This results in inflammatory heart disease.

Earlier research had suggested that inflammatory heart disease arises when autoantibodies are produced against the host's own myosin. In order to confirm this, purified myosin peptides were injected into mice. This was found to result in severe heart disease.

In the latest set of experiments, Dr Kurt Bachmaier and his colleagues identified the myosin peptide region responsible for inducing the autoimmune response as a 14 amino acid sequence. They searched databases of known viral and bacterial peptide sequences to see if they could find a match for the mouse myosin peptide. "To our amazement," said Dr Bachmaier, "we found similarity in the outer membrane of chlamydia bacteria."

To test their discovery, the researchers injected the chlamydia peptides into mice and confirmed that the same immune cells (CD4 T cells) were activated as when the myosin peptide had been injected. Up to 88% of the mice subsequently developed inflammatory heart disease. □