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### Effect of Estonian law on prospects for public health research

Sir—On Feb 12, 2003, with only 26 of its 101 members present, the Estonian Parliament (the Riigikogu) unanimously adopted a new law on data protection. The law that came into force on Oct 1, will inflict profound damage on public health research in a country where life expectancy at birth is almost 8 years less than in the European Union (EU), which it is due to join in May, 2004.

Unlike similar laws in other countries, which include exemptions for epidemiological surveillance and research, subject to appropriate safeguards, the Estonian legislation precludes any use of personally identifiable health data unless the individual has given explicit consent for how it will be used. The expression “public interest” is mentioned only once in the legislation, and there is no mention of either “scientific” or “research”. The regulation is much more restrictive than the European Directive 95/46/EC that the Estonian Government is meant to be implementing.

The consequence will be to prohibit virtually all registry-based epidemiological research where record-linkage has been based on a personal identification number. Although Estonia boasts of its progress in “e-government” and has declared its intention to become an “e-state”, the country’s few epidemiologists and public health researchers can only dream of undertaking studies such as those in neighbouring Nordic countries which have done so much to advance

understanding of the determinants and mechanisms of disease.

The new law places in jeopardy existing national registries, including the Estonian Cancer Registry, which has accumulated 150 000 cases since 1968, and which is highly respected internationally. Bureaucratic obstacles have already prevented the registry from linking to data from the national mortality database, so that about 5% of incident cases, for which the only primary information is from death certificates, are now lost. This gap clearly obscures real trends in incidence and thus gives a false impression of the health of the Estonian population. The use of personal identifiers in the national mortality database, maintained by the Statistical Office of Estonia, has been declared as violating privacy rules and their removal is now under discussion.

Those drawing up the EU directive on data protection were persuaded of the need for exemptions to the general right to privacy on grounds of public health when the dangers of failing to do so were brought to their attention,<sup>1</sup> and although the implementation of the legislation has given rise to substantial confusion by over-zealous authorities in some countries,<sup>2</sup> such uncertainty is now being resolved in most. Yet as the Estonian decision shows, there is a need for continuing vigilance by the European public health community, who must continue to speak out in support of our colleagues who are struggling to understand the health of their populations.

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### A bad dose of the 'flu

Sir—Cases of acute necrotising encephalopathy (ANE) have been reported mainly from Japan.<sup>1</sup> D L Jardine and colleagues (Oct 11, p 1198)<sup>2</sup> describe one case, and suggest that subtle antigenic changes to influenza virus might be the cause. However, if so, why in this era of large-scale global travel is ANE so rare among Western people?

First, ANE is seen not only in patients with influenza, but also in febrile

patients with upper respiratory infections caused by other viruses. Mizuguchi and colleagues<sup>3</sup> reported ANE cases with exanthema subitum, Cocksackievirus A9 and B4 infection, herpes simplex, and measles, as well as influenza A and B.

Second, many doctors in Japan prescribe stronger antipyretics than aspirin (eg, diclofenac and mefenamic acid) to febrile children. In the national survey of 1998–99, correlation was noted between the use of antipyretics and death due to influenza-associated encephalopathy, including ANE. ANE seems to be caused by an exaggerated cytokine response resulting in vascular damage and breakdown of the blood-brain barrier.<sup>1</sup> However, the causative agent is not necessarily the virus itself. Cytokine responses to drugs in the febrile state could have a major role. In Japan, the Ministry of Health and Welfare banned prescription of diclofenac in 2000, and mefenamic acid in 2001, for influenza. Whether cases of influenza-associated encephalopathy will decrease, remains to be seen.

Third, many Japanese doctors prescribe several drugs at once to febrile children with upper respiratory infections. In one report, a 3-year-old girl treated with cefdinir (an antimicrobial), procaterol (a  $\beta_2$ -adrenergic agent), ambroxol (an expectorant), alimemadine (an antihistamine), and acetaminophen succumbed to ANE; in addition, a 1-year-old boy prescribed erythromycin, tulobuterol ( $\beta_2$ -adrenergic agent), ambroxol, carbocysteine (expectorant), bromhexine (expectorant), and ephedrine also died of ANE.<sup>4</sup> Although, in these two cases, too many drugs were used to clarify the cause-effect relation with ANE, the possibility that drugs induce influenza-associated encephalopathy, including ANE, should be kept in mind.

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