carrier rate is 1 in 25 and if 80 per cent of CF genes are ΔF_{508}, the risk to the offspring of a known ΔF_{508} carrier and a partner testing negatively falls to 1 in 500, with at least a proportion destined to have a milder form of the disease.

To compare this risk with that in the unscreened population, as Ten Kate suggests, creates needless anxiety in those who opt for screening. In genetic counselling, we often have to quote risks much greater than for the population as a whole, but which those counselled regard as encouraging. Risks comparable with 1 in 500, for example, obtain in the case of the relatives of those with CF; so far, the great majority have undertaken or continued with pregnancies without any form of prenatal diagnosis.

In the general population, the great majority would test negatively for ΔF_{508}, and the risk factor for couples of such people would fall from 1 in 2,500 to 1 in 62,500. All screening causes extra anxiety for some of those involved, but can it be wise to deny reassurance to the great majority of couples related to known cases of CF?

Were testing to be offered tomorrow to women in early pregnancy, it would perform rather better than the criteria now applied in the avoidance of Down's syndrome, where indicators such as maternal age, low serum alphafetoprotein and high human chorionic gonadotropin define those with risks greater than 1 in 250 and which, followed by amniocentesis, detect some 60 per cent of Down's syndrome cases.

For Down's syndrome, assuming a population incidence of 1 in 650, in 100,000 pregnancies there would be 5,000 amniocenteses and 92 of 154 cases would be detected (Wald, N., Cuckle, H., and Royston, P., *Lancet* **ii**, 1362; 1988). With CF, 100,000 pregnancies would require 52 amniocenteses which would detect 26 of 40 cases (arithmetic available on request).

A further benefit of an early start on ΔF_{508} screening is that it would assist with public education, encouraging people to opt for carrier testing before pregnancy and widening their options for the future. Naturally, we acknowledge that, as with other screening for autosomal recessive disease, the need for accurate information on paternity is a consideration of which those to whom testing is offered should be aware, Furthermore we do not underestimate the need that ΔF_{508} screening should be undertaken only when there is a back-up infrastructure of genetic counselling and support.

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Endothelin in myocardial infarction

SIR—There has been a surge of publications on the newly identified vasoconstrictive peptide endothelin since the first report by Yanagisawa *et al.*[1]. Although the mechanisms of action and the effects of exogenous endothelin have been widely studied, the pathophysiological roles of endogenous endothelin are still unclear. Recently, we established a monoclonal antibody against endothelin[2] and an appropriate sandwich enzyme immunoassay system. Using these probes, we have succeeded in demonstrating that endogenous endothelin contributes to the extension of myocardial infarction.

The essence of our findings is as follows. First, treatment with the antibody for endothelin reduces the infarction size in the rat heart. Left-coronary artery ligation (1 h) followed by reperfusion (24 h) induces myocardial infarction in 35 per cent (n=8) of the left ventricle in rats. In this infarction model, administration of the antibody significantly reduces the size in a dose dependent manner; 4.5 mg per kg i.v. reduces it to 20 per cent (a 43 per cent reduction in size). Second, plasma and cardiac tissue concentrations of endogenous endothelin are increased to roughly 4–7 times the control value after the coronary ligation-reperfusion procedure: 0.93 to 4.0 pg per ml for the plasma and 5.0 to 33.1 pg per g tissue for the heart. These results indicate that endogenous endothelin exists in the bloodstream as well as in cardiac tissue under normal conditions, and ischaemic conditions cause an increase in these concentrations, which in turn establishes myocardial infarction.

Several factors have been reported to be involved in establishing myocardial infarction, including free radicals, catecholamines, thromboxanes and various catabolites[3]. Although there is still controversy whether the elimination of these factors reduces the infarction size, we have demonstrated that neutralizing endogenous endothelin with the antibody results in a smaller size. Therefore, we believe that endothelin is an important member of the family of factors necessary to establish the infarction.

Our findings are also clinically significant. It is beneficial to limit the myocardial infarction, because a larger infarction area may cause severe arrhythmias and haemodynamic depression. Thus, reducing the endogenous endothelin level is a promising method in the treatment of patients following a heart attack.

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El Chichòn dust a persistent problem

SIR—Recent discussions on the use of satellite data to derive increases of global sea surface temperatures[1-3] have highlighted the importance of understanding all parameters, and their interactions, contributing to the global heat budget. Solid volcanic particles in the stratosphere[3] should be included, but the first analyses of satellite data assume that particulates from the El Chichòn volcanic eruptions of 29 March and 3–4 April 1982 interfered only with satellite data collected in the two-year period after the eruptions[1,2].

The residence time of aspherical particulates in the stratosphere is probably severely underestimated when calculated on settling rates. A study[4] of morphology and size distributions of ash particles in the El Chichòn stratospheric cloud, in combination with settling rate calculations for aspherical particles, concluded that the eruptive cloud ceiling reached an altitude of 34–36 km, consistent with lidar and optical polarization observations[5], or that turbulent mixing of the volcanic cloud significantly enhances the residence time of ash particles in the stratosphere.

In May 1985, a balloon flight from Palestine (Texas) carried a dust collector to sample the stratosphere between 34 and 36 km altitude[6]. Electron microscope analyses of samples (by J. R. Arnold, University of California) show that an important fraction of particulates larger than about 0.5 μm are platey angular shards of silica, plagioclase feldspar and Fe, Mg-silicate grains, and clusters of these grains up to 5 × 4 μm in size. Typically, various amounts of smaller particulates, including sulphuric acid droplets, adhere to these grains.

Settling rate calculations for aspherical particulates show that these large shards and clusters should have settled from 35 km to the tropopause in about one year[4,7]. The samples also include rare particulates of barite ($BaSO_4$) and PbO_2 and unidentified submicrometre-sized grains containing iron, nickel, zinc and lead, and occasionally sulphur. Of the plinian volcanic eruptions ejecting par-