

groups. I was also wondering about the validity of making a personality disorder diagnosis in such young individuals. Overall, if the diagnosis of anorexia could become bulimia, EDNOS or no eating disorder, the authors failed to consider the reverse being true (with the relative exception of bulimia to anorexia) at the important initial stage of this study.

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Authors' reply: Dr Sekar has concerns that the individuals in our study had other eating disorders before the onset of anorexia in adolescence. The aim of the original study, that took place in 1985, was to investigate the prevalence of adolescent-onset anorexia (and to examine background factors in this sample), not the prevalence of bulimia nervosa or eating disorder not otherwise specified. The mothers of the individuals, who were diagnosed with anorexia at the time of the original study, were interviewed thoroughly regarding premorbid eating disturbances.¹ Furthermore, the individuals themselves were interviewed regarding the same topic. No individual in the anorexia group (or the comparison group) had another eating disorder before the onset of anorexia. The school nurses at the schools in Göteborg continued to follow all pupils born in 1970 regarding weight and height until leaving school, usually after age 18 years. In the process, individuals with a later adolescent-onset of anorexia were also found. We believe that we have missed no cases of anorexia born in 1970 with anorexia onset before age 18 years. Since the original study focused on adolescent-onset anorexia we have not continued the screening of individuals born in 1970 after leaving school. Mean age of anorexia onset in our sample was 14.3 years. Bulimia typically presents during or after late adolescence and it is rare for onset to occur before the age of 14 years.^{2,3}

The study has a prospective and not a cross-sectional design, i.e. we have examined all individuals at four occasions, but we have interviewed them both regarding current eating disorders (and other psychiatric disorders) as well as eating disorders during the follow-up period.^{4,5} Data regarding eating disorders during the last follow-up period, between Study III and Study IV, are available from the first author.

Dr Sekar is also worried about the two subgroups being too diverse; the birth cohort with individuals born in 1970 was pooled together with a group of individuals with adolescent-onset anorexia born in adjacent years (in most cases 1971–1973). In the original study, the two groups were compared using several hundred background parameters and found to be similar in virtually all key respects.¹

The use of personality disorder diagnoses with teenagers is arguable, but we considered (and still consider) it justifiable in cases persistently (over a period of several years) showing the essential characteristics of a personality disorder described in the DSM–III–R (the diagnostic manual used at the time of the original study). This is explicitly suggested by DSM–III–R

guidelines. In the original study, apart from the age criterion, all DSM–III–R criteria had to be fulfilled for a diagnosis of personality disorder to be made. All individuals receiving a diagnosis of personality disorder showed significant impairment in social functioning and/or subjective distress.¹

To conclude, since the aim of the original study was to investigate prevalence of adolescent-onset anorexia, we did not screen for other eating disorders. Nevertheless, from the time of entering our study, all participants (anorexia group and comparison group) were examined in great detail regarding eating disorders (past, present, and longitudinally at several follow-up occasions). We believe that we can safely say that there were no individuals who had crossed-over from another eating disorder to anorexia before the onset of anorexia in adolescence.

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Fallacies in standardised mortality ratios in anorexia nervosa

The article by Papadopoulos *et al*¹ adds to the evidence of high mortality rates in anorexia nervosa. An impressively large cohort was obtained through the Swedish Cause-of-Death Register which includes all Swedish persons who died since 1952. The crude mortality rate for 6009 females with at least one hospital admission for anorexia nervosa was 4.41% over a mean follow-up of 13.4 years (averaging 0.33% per annum). This rate compares favourably with other studies (0.5–2.2% per annum),² yet the authors, after much manipulation of their data, conclude that the mortality rate in Swedish women was 'astonishingly' high.

We contest this finding based on misleading calculations of standardised mortality ratios (SMRs). Standardised mortality ratios are a means of comparing mortality in a specified patient population with a standard population. The SMR value will exceed 1 in proportion to the risk of death from the disease under study.

The authors have two different usages of SMR. The first is the customary one when the calculation is applied to a cohort of persons who have been given a specific diagnosis at the outset. In Table 3 this SMR is given as 6.2 for the 6009 patients with anorexia nervosa, among whom there occurred 265 deaths whereas the expected deaths were 42.6. So far, so good.

Their second approach was to count the number of deaths according to each specific cause of death, yielding a different kind

of SMR. For example, there were 84 suicides yielding an SMR of 13.6, signifying that suicide was 13.6 times more common among the cohort of patients with anorexia nervosa than generally expected. Similarly, the SMR for deaths due to respiratory disease was 11.5. But the SMR for anorexia nervosa as a cause of death was said to be 650.0 and it is this figure which leads the authors to conclude the death rate in their sample was astonishingly high.

So it would be if it had clinical and statistical validity. The authors' errors arise from estimating the SMR for a subgroup ($n=39$) of the original cohort using the fraction:

$$\frac{\text{observed number of deaths}}{\text{number of expected deaths}}$$

The numerator is given as 39 patients in whom anorexia nervosa was the main cause of death on the death certificate. It is the denominator which is elusive in its estimated value. It is given as 0.1 but the authors' own data suggest this is an approximation for 0.06, a very low figure which results in an inflated value for the SMR (650) in this ambiguous subgroup of anorexia nervosa. We suggest that when an underlying cause of death (e.g. suicide, respiratory infection) was not identified, the certifier of the death entered anorexia nervosa on recognising a cachectic state, especially as malnutrition does not feature in the list of 'underlying' causes.

These objections do not apply to the first calculation of the SMR in the full cohort of patients with anorexia nervosa whose value was found to be 6.2, by no means an astonishing death rate.

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Authors' reply: Professor Russell and Dr Ward raise the issue of the suspected erroneously inflated value for SMR (650) for the subgroup of women in whom anorexia nervosa was stated as the underlying cause of death on the death certificate in our paper.¹ The expected number of deaths for this subgroup was indeed 0.06 (denominator) as they point out and it was presented with its one decimal approximation (0.1). Russell & Ward further suggest that the certifiers of the death would be prone to enter anorexia nervosa on the death certificate when a specific underlying cause of death could not be identified but a cachectic state was evident. We agree that this could be true, but we do not believe that such 'misclassification' would be problematic if those women had an active anorexia nervosa at the time of death. On the contrary, it would be worrisome if women with other diagnoses that lead to cachectic states (other than anorexia nervosa) were misclassified as anorexia nervosa on death certificates, but our inclusion criteria were specifically selected in order to reduce this possibility. In addition, we believe that the estimation of the SMR value for this specific subgroup of patients does not confer more information than what common sense dictates, namely that those with a lifetime diagnosis of anorexia have a much higher risk of dying from it.

Overall, women with anorexia nervosa in our cohort had a sixfold increased mortality compared with the general population.

This excess mortality in anorexia nervosa is two to three times higher when compared with the excess mortality observed in mental disorders in general² and more specifically in schizophrenia,³ bipolar and unipolar disorder.⁴ Moreover, we would like to point out that we were most astonished by the persistence of this unfavourable outcome throughout the lifetime, with high SMRs for both natural and unnatural causes of death even 20 years or more after the first admission for anorexia nervosa.

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Suicide rates in people of South Asian origin in England and Wales

A notable finding in McKenzie *et al*'s study¹ of suicide rates in people of South Asian origin is that the high relative rates in younger Asian women reported in previous research studies are found in the 1993–98 data-set but not that for 1999–2003, which shows high relative rates for Asian women over 65. In discussing their results, the investigators acknowledge potential problems with the study's methodology, including the numerator (how well the SANGRA name recognition algorithm ascertains individuals of South Asian origin in more recent samples) and denominator (the validity of a linear interpolation of numbers over their period). However, perhaps cautions are required with respect to the overall robustness of the SANGRA algorithm and the issue of numerator/denominator compatibility: the numerator uses an operational definition of ethnicity (derived from name information) and the denominator is based on self-assignment by individuals to census categories.

These matters are brought into focus in the derivation of denominators. The investigators use the counts for the 1991 categories 'Bangladeshi', 'Indian', 'Pakistani' and 2001 categories 'Asian or Asian British: Bangladeshi, Indian, Pakistani'. They also include the 2001 category 'White and Asian' (numbering around 190 000 in the census) on the grounds that people in it '... could be identified by SANGRA if any of their names were of South Asian origin'. We have no systematic data on how offspring of these inter-ethnic unions are named, although qualitative research has revealed the complexity of the process.² Inclusion of the 'White and Asian' category also introduces heterogeneity into the South Asian collectivity. Evidence from the Office of National Statistics (ONS) Longitudinal Study for members having a 1991 and 2001 ethnic group showed that half (49.0%) of the 993 'White and Asian' persons identified as 'White' in 1991 and just 9.5% identified as one of the three South Asian groups.³ Similarly, in recent research half in the 'White and Asian' group prioritised 'White' when asked to name just one ethnic group that contributes most strongly to their identity. Our collective