British Journal of Psychiatry (1995), **166**, 462–471

Anorexia Nervosa
No Evidence of an Increase

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Background. This paper examines the evidence that rates of anorexia nervosa have increased over time.

Method. Epidemiological studies that have provided time trends in incidence rates in specific countries or psychiatric registers and prevalence surveys were reviewed to assess whether or not prevalence estimates are higher in the most recent studies.

Results. Estimated trends in incidence rates showed an increase but no study ruled out plausible alternative explanations. Studies showing no upward trend were as numerous and were generally based on larger samples. Analysis of 29 cross-sectional surveys conducted over the last 25 years indicated that the median prevalence rate was 1.3 per 1000 females. There was no indication that more recent surveys yielded higher prevalence rates.

Conclusion. Anorexia nervosa remains a rare disorder and there is no evidence of a secular increase in its incidence.

Among eating disorders, anorexia nervosa (AN) represents the less common, but the most severe form of eating disorder. The mean age of recognised onset for AN is about 17 years (Hsu, 1990), and its incidence is about ten times higher among girls compared with boys. Higher rates of comorbid disorders have been consistently reported in samples of anorectic patients. Follow-up studies of anorectic patients have shown a high persistence of eating symptoms and of interpersonal difficulties, poor psychosocial adaptation in one-fifth of them and increased mortality rates (Steinhausen *et al*, 1991). Family studies (Strober *et al*, 1990) have shown an increased incidence of eating disorders among the first- and second-degree relatives of patients diagnosed with AN. That genetic factors might be implicated in the causation of AN has been supported by twin studies showing higher concordance rates for monozygotic than for dizygotic twins (Treasure & Holland, 1991). However, heritability estimates suggest also that non-genetic factors play a crucial role in the development of AN. In light of the high individual and societal costs associated with this disorder, reports of an increased incidence of AN in recent decades need to be carefully examined, as such evidence might help to identify psychosocial risk mechanisms explaining secular changes in rates of AN. The aim of this paper is primarily to establish whether or not current epidemiological data support the notion of an increased incidence of anorexia nervosa.

Methods

Problems of definition and measurement

In comparing studies across countries and over time, one should bear in mind that various definitions have been used in published studies. For example, the diagnostic criteria for AN changed from DSM–III (American Psychiatric Association, 1980) to DSM–III–R (American Psychiatric Association, 1987). Former criteria required a weight loss of 25%, whereas this was reduced to 15% in DSM–III–R. By contrast, a new diagnostic criterion, i.e. the absence of three consecutive menstrual cycles, was added in the latter definition. Obviously, these changes in diagnostic definitions could lead in some instances to spurious variations in rates of disorders across studies. Furthermore, studies also vary in their adherence to strict diagnostic criteria and in several studies 'partial' syndromes of AN have been included in the case definition.

Another source of variation is introduced by studies that relied on self-report measures to assess the prevalence of anorectic types of behaviour. Questionnaire measures have low positive predictive value (Fairburn & Beglin, 1990) and they often lack demonstrated cross-cultural validity (King & Bhugra, 1989). We therefore excluded from this review studies that relied solely on questionnaire-based definitions and only studies relying on a psychiatric diagnostic approach to case definition were included.

https://doi.org/10.1192/bjp.166.4.462 Published online by Cambridge University Press

Data sources

Psychiatric and hospital records

Two main types of studies were used to examine time trends in eating disorders. The first group of studies are those based on psychiatric case registers or on hospital admission records. The advantage of using these studies is that in some areas long runs of data are available, going back to the 1930s in a few instances. Their drawbacks are that records cover only cases that came to the attention of the relevant health services, that diagnostic criteria are not always precisely defined, vary between sites or between clinicians at the same sites, and may change over time. The fact that only treated cases are used in these studies may certainly lead to an underestimation of rates of AN. However, these findings may be used to study trends over time assuming that the proportion of cases missed at each historical interval remained constant. However, it is difficult to establish whether that assumption is tenable. Indeed, as shown below, the proportion of cases that came to the attention of specialists might have changed with increasing accessibility of services, with change in health-seeking behaviour and with increasing recognition of the disorder by health professionals and the general population. The second limitation can be somewhat circumvented by limiting variation in diagnostic criteria. Records can be re-examined using fixed diagnostic procedures over time. However, the quality of the records may be uneven and information may be missing on core symptoms especially those that have become important diagnostic criteria since the time when the records were compiled.

Community surveys

The second group of studies are community surveys carried out among both adolescent and adult populations. Community surveys vary enormously in their sampling methodologies, design, interviewing techniques and participation rates. One possible way to use their results to assess time trends would be to compare lifetime rates of AN across successive birth cohorts using survival analysis methods. Then, decreased lifetime rates with age could be taken as evidence for an increased incidence of the disorder among recent birth cohorts. However, this method could not be used since absolute rates of AN were too low for declines of lifetime rates with age to be detected. Furthermore, had it been possible, differential mortality or decreased participation in surveys with age could not have been ruled out as possible artefacts. The other way to use cross-sectional surveys is to compare the average prevalence rates obtained from surveys conducted at successive time periods.

Results

Trends in incidence rates for AN

A total of 16 published studies were available where incidence rates for AN were derived (Theander, 1970; Kendell *et al*, 1973; Crisp *et al*, 1976; Jones *et al*, 1980; Willi & Grossman, 1983; Hoek & Brook, 1985; Szmukler *et al*, 1986; Cullberg & Engström-Lindberg, 1988; Lucas *et al*, 1988, 1991; Ben-Tovim & Morton, 1990; Nielsen, 1990; Willi *et al*, 1990; Hoek, 1991; Joergensen, 1992; Moller-Madsen & Nystrup, 1992). It is worth noting that incidence rates were not reported in the same format and that not all authors computed age- and gender-specific rates. Where rates for young women were available, there was a huge variation in reported rates, which ranged from a low of 0.117/100 000/year for 12–18-year-old Australian girls (Ben-Tovim & Morton, 1990) to a high of 69.4/100 000/year among 15–19-year-old American girls (Lucas *et al*, 1991). Confidence intervals were inconsistently reported and the precision of estimates is necessarily uneven, as shown by the wide variation in the number of female cases identified in these studies. Thus, the total number of female cases varied from 1 (Ben-Tovim & Morton, 1990) to 844 (Moller-Madsen & Nystrup, 1992); the median number of cases was 53.5 for 14 investigations. When available, incidence rates were compared among females and males and this showed a consistent tenfold increase in incidence for women. Incidence rates for AN consistently peaked for the 15–19-year-olds, and decreased somewhat for the 20–24-year-olds. No evidence was found of a bimodal age of onset for AN as was once reported in one isolated clinic sample (Halmi *et al*, 1979).

Assessment over time

A subset of these studies was selected because it allowed for an assessment of changes over time in rates of AN in defined areas or populations (Table 1). In general, no firm conclusion can be drawn from studies that were conducted in the same country but at different locations or by different authors. Differences in diagnostic criteria and in case-finding methodologies are the rule and jeopardise attempts to contrast results across studies. Thus, the incidence obtained recently in Sweden (Cullberg & Engström-Lindberg, 1988; see Table 1) cannot meaningfully be interpreted against

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Table 1
Anorexia nervosa: incidence rates from studies on medical records for women (unless otherwise indicated)

Place	Reference	Period	Diagnostic criteria	Age group	Annual rate/ 100 000/ year	Total no. of female cases
Denmark: nationwide register	Nielsen (1990)[1]	1973–87	ICD-8	All	1.90	744
Denmark (Fyn County)	Joergensen (1992)[1]	1977–86	DSM-III-R	All	3.25	87
Denmark (first psychiatric admissions, national register)	Moller-Madsen & Nystrup (1992)	1970–89	ICD-8	All ages[2]		844
				1970	0.42[3]	
				1988	1.36[3]	
				1989	1.17[3]	
				15–24 (women)		536
				1970	3.37	
				1987	11.96	
				1989	8.97	
Netherlands: local psychiatric case register	Hoek & Brook (1985)	1974–82	ICD-9	All[3]	5.00[3]	20
Netherlands (58 general practices)	Hoek (1991)	1985–86	DSM-III	All[3]	6.30[3]	19
Sweden (southern Sweden)	Theander (1970)	1931–40	Clinical	All	0.08	94
		1941–50			0.19	
		1951–60			0.45	
Sweden (south-east Stockholm)	Cullberg & Engström Lindberg (1988)	1984–85	DSM-III modified	All	2.60	16
Switzerland: Zurich	Willi & Grossmann (1983)	1956–58	Clinical	All	0.38	10
		1963–65			0.55	17
	Willi *et al* (190)	1973–75			1.12	38
		1983–85			1.43	48
		1956–58		12–25	3.98	
		1963–65			6.79	
		1973–75			16.76	
		1983–85			?	
UK: Scotland (psychiatric register)	Kendell *et al* (1973)	1966–69	Clinical	All	1.60[3]	28
	Szmukler *et al* (1986)	1978–82	Russell	All	4.68[3]	120 (approx.)
USA: Monroe county, New York[4]	Jones *et al* (1980)[5]	1960–69	Clinical	All[3]	0.35[3]	
				All women	0.49	16
				15–24	0.55	
		1970–76		All[3]	0.64[3]	
				All women	1.16	29
				15–24	3.26	
USA: Rochester, Minnesota	Lucas *et al* (1988)	1935–49	DSM-III-R/ Russell	10–19	52.40	128
		1950–64			21.60	
		1965–79			48.60	
	Lucas *et al* (1991)	1935–84	DSM-III-R/ Russell	Age adjusted rate for all women	14.60	166
		1950–54			7.00	
		1980–84			26.30	

1. These two studies do not provide separate incidence rates across the study period but graphical displays of yearly incidence rates of number of cases identified were produced by the authors and showed no evidence of an increase. Nielsen (1990) obtained a non-significant result for a statistical test for time trends.
2. For the diagnosis of anorexia nervosa and other eating disorders.
3. Incidence rate for total population (both sexes combined).
4. Rates are for the white population only of Monroe county (the estimated average annual incidence rate for the non-white female population was 0.42 for the period 1970–1976).
5. Slightly different rates and numbers have been published by Kendell *et al* (1973) but the pattern of results is broadly similar.

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former rates obtained by Theander (1970). Similarly, the two Dutch studies (Hoek & Brook, 1985; Hoek, 1991) were too dissimilar in their methodologies to lead to useful inferences in gauging time trends in rates; for the same reasons, the Danish study that yielded the highest incidence rate (Joergensen, 1992) cannot be compared to the other two Danish studies, which, by contrast, shared similar sampling and diagnostic features (Nielsen, 1990; Moller-Madsen & Nystrup, 1992). However, it is worth noting that the methodology appears to play a substantial role in explaining variations in rates across these three Danish studies, which encompassed roughly the same time interval. Due to these limitations, time trends in rates are easier to examine from repeated investigations conducted at a single site.

An increase over time has been detected for various periods between 1930 and 1990 at five different sites: Monroe county (Jones *et al*, 1980; Kendell *et al*, 1973), north-east Scotland (Kendell *et al*, 1973; Szmukler *et al*, 1986), Sweden (Theander, 1970), Switzerland (Willi & Grossman, 1983; Willi *et al*, 1990) and Rochester, Minnesota, USA (Lucas *et al*, 1988, 1991). An upward trend was shown in every study based on case registers in which incidence could be estimated at more than one point in time. However, four limitations in the interpretation of these data need to be borne in mind.

Diagnostic methods

Firstly, most of these register data relied on hospitalised cases and, with few exceptions (Szmukler *et al*, 1986; Lucas *et al*, 1988, 1991), no attempts were made to assess the validity and reliability of diagnoses or to ensure that consistent diagnostic criteria had been used throughout the study periods. Accordingly, trends in rates may reflect only changes in diagnostic practice. For example, anorectic patients were recorded as 'hysterical neuroses' prior to 1966 in the north-east Scottish register, precluding the derivation of any incidence estimates for AN in the first years of operation of the register (Kendell *et al*, 1973).

Availability of services

Secondly, trends could be caused by an increased use of services. For example, there is some evidence that the increased frequency of AN reported in Monroe county (Jones *et al*, 1980) could have reflected, at least in part, an improved availability and use of local mental health services by children and adolescents for a range of psychiatric disorders (Roghmann *et al*, 1982). Similarly, evidence has been produced that the proportion of cases of AN identified as formal psychiatric cases had increased over time, thereby suggesting that upward trends in rates might partially reflect better case identification methods. Thus, in the Aberdeen region, 63% of the total number of cases were known to the psychiatric case register in 1970–74, a proportion that rose to 87% in 1978–82 (Szmukler *et al*, 1986).

Incidence rates

Thirdly, incidence rates have often been reported as crude, unstandardised estimates. Crude rates may be misleading because they relate the number of cases to the total population, whereas most sections of the population have virtually zero risk of eating disorders. Yet incidence rates for the section of the population mainly at risk (girls and young women) have not consistently been reported and variation in crude rates could merely reflect changes in the demographic structure of the underlying populations. Fourthly, formal tests of the statistical significance of the recorded increases have scarcely been used. This is a striking weakness of several psychiatric case register studies, particularly in light of the limited number of cases often identified in these studies (see Table 1, right column).

The Lucas case-study

The study by Lucas *et al* (1988, 1991) deserves more attention because it avoided many of these pitfalls. Cases were identified through medical records, regardless of whether the subject had been hospitalised or in contact with a psychiatrist. Conducted in a circumscribed geographical area, the study spans a longer period than any other, has a relatively large ($n = 166$) sample of cases and supports fine-tuned analyses of short-term trends over five decades. Cases were diagnosed by applying consistent diagnostic criteria to all records screened, rates were adjusted and standardised, and appropriate analyses of trends were provided. For girls aged 10–19 there was a quadratic trend with high rates of AN in 1935–39, a regular decline over the next 20 years until the 1950s, then an increase up to 1980–84, the last quinquennium for which there are published data (Lucas *et al*, 1991). Rates of AN showed a statistically significant linear increase from the 1950s to the 1980s (Lucas *et al*, 1991). Nevertheless, there are four limitations in the analysis and interpretations of the data as presented in the published papers.

Firstly, the linear increase in rate of AN since the 1950s is strongly dependent on the last data point (accounting for 23% of cases) and also on the

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inclusion of the 1950–54 data in this analysis, whereas prior tests for a linear trend in the most recent period (Lucas *et al*, 1988) had started with the 1955–59 quinquennium only. Furthermore, in their most recent analysis (Lucas *et al*, 1991), the authors reported that, for the differently selected age group of 15–24 year olds, there has been a significant linear increase in AN starting in the 1930s. However, it is not clear why these overlapping age groups (females aged 10–19 and 15–24) should show an opposite pattern of change over time (a fall between the 1930s and 1950s for those aged 10–19, and a linear increase for those aged 15–24). If confirmed, these findings would imply that in the early quinquennial periods, rates went in opposite directions for different age groups, a surprising result needing careful interpretation.

Secondly, Lucas and colleagues recognised the possibility that a spurious increase in the rate of AN could occur because of differential migration. To control for this possibility, they excluded cases where the patient had moved into the Rochester area during the 12 months prior to the consultation. Whether this is an adequate control is uncertain as the first symptoms of AN generally appear several years before the first medical contact (Garfinkel & Garner, 1982; Hsu, 1990; Willi *et al*, 1990). In their first report (Lucas *et al*, 1988) stated that 11% of the original body of cases had been rejected because the patients had recently migrated into the area; unfortunately, no corresponding figures were quoted in the later report for the more recent period. Also, the possibility remains that people already resident in the area will be less likely to move out if a member of their family has anorexia than otherwise. Thirdly, Lucas *et al* (1988, 1991) noted over the time period studied a shift in the local economy from farming and light industry to employment based on the Mayo Clinic and computer industry. This may well have led to an upward trend in social class that could account partially for the trend, since AN appears to be more prevalent among high social classes. Fourthly, the reliability of the diagnoses was not assessed for the whole series of cases nor compared between periods.

Demographic factors and readmission rates

By contrast, three investigations have suggested that the apparent increase in the incidence of AN could be explained by demographic changes in the general population (Williams & King, 1987; Willi *et al*, 1990) and by higher readmission rates (Williams & King, 1987; Nielsen, 1990). The Zurich study found an increase from the mid-1950s to the 1970s, but no difference between the 1970s and the 1980s (Willi *et al*, 1990). More specifically, the difference in crude incidence rates for the 1970s and the 1980s was not statistically significant and disappeared entirely after standardisation for age and sex (Willi *et al*, 1990). Similarly, Williams & King's (1987) analysis of psychiatric first admissions and readmissions with the diagnosis of AN concluded that the increase shown by English hospital statistics for the period 1972–81 was accounted for by changes in the age structure of the general population in the case of first admissions, and by changes in patterns of treatment in the case of readmissions. Similarly, analysing the 807 first psychiatric admissions for AN identified in a nationwide study of psychiatric registers conducted in Denmark for the period 1973–87, Nielsen (1990) found that rates of first admissions remained stable during this period. A reduction by over one-third in the number of psychiatric beds, combined with increasing readmission rates for AN could have contributed to the false impression of an epidemic of AN. Another Danish register study (Moller-Madsen & Nystrup, 1992) found a significant increase in rates between 1970 and 1988 but the authors used a broader case definition (including anorexia nervosa and other eating disorders) and a different case identification method. Since most of the reported increase was found after 1982, it is clear that these results might have been confounded by the newly recognised bulimic disorders. In addition, rates were not standardised and the authors provided only weak evidence that their results were not a reflection of a differential pattern of health services' use for eating disorders, with more eating disordered subjects being admitted in psychiatric, rather than somatic, hospitals in the more recent period.

Prevalence surveys

The results of 29 studies are summarised in Table 2. One recent study (Childress *et al*, 1993) of 3175 9–16-year-old children was excluded as, due to a lack of information on the second-stage sample, it was not possible to derive a meaningful prevalence estimate. In Table 2, the prevalence of AN ranges from nil among schoolgirls in Japan (Suzuki *et al*, 1990) to 1.3% among German-speaking girls living in a small rural town of northern Italy (Rathner & Messner, 1993). It is difficult to perform comparisons between studies because of differences in the methods used. In general, studies conducted on large sample sizes ($n > 5000$) and among young adults tended to yield lower prevalence rates for AN. Studies conducted among school-age adolescent girls and having a specific focus on eating disorders yielded

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Table 2
Prevalence studies of anorexia nervosa

Reference	Period	Place	Age group	Sample size	Diagnostic criteria	Rate/100
Crisp *et al* (1976)	1967–74	UK (7 girls schools)	All ages	? (private schools)	Clinical	F 0.215[1]
			16+	2786 (state schools)	Clinical	F 0.036[2]
Nielsen (1990)	1973–87	Denmark	All	300 000	ICD-8	F 0.0067
			15–19			F 0.0347
			10–24			F 0.0237
Lucas *et al* (1988)	1980	USA: Rochester, Minnesota	All ages	F 30 628	DSM-III-R	F 0.204[3]
			15–19	F 2654		F 0.330
Whitehouse & Button (1988)	1980	UK (Chichester)	16–19	F 446	Feighner/ Russel DSM-III	F 0.220 F 0.450
Robins *et al* (1984)	1981–82	USA: New Heaven	18–65	3058	DSM-III	0.030
		USA: Baltimore	18–65	3481	DSM-III	0.100
		USA: St Louis	18–65	3004	DSM-III	0.100
Mann *et al* (1983)	1982	UK (2 London schools)	15	F 262	Clinical	F 0.000
Szmukler (1983)	1982	UK (6 private girls schools)	14–19	F 1331	Clinical	F 0.830
			16–18		Clinical	F 1.110
		(state schools)	16+		Clinical	F 0.140
Joergensen (1992)	1977–86	Denmark (Fyn County)	All	450 000	DSM-III-R	F 0.026
			10–14			F 0.070
			15–19			F 0.120
			20–24			F 0.060
Cullberg & Engström-Lindberg (1988)	1984–85	Sweden (south-east Stockholm)	16–24	F 4651	DSM-III modified	F 0.258
Whitaker *et al* (1990)	1984	USA: New Jersey	14–17	F 2544	DSM-III	F 0.200
Lee *et al* (1987)	1984	Korea (Seoul and rural area)	18–65	5100	DSM-III	F 0.038[2]
Hwu *et al* (1989)	1982–86	Taiwan (nationwide)	18+	11 004	DSM-III	0.000
Rastam *et al* (1989)	1985	Sweden (Göteborg)	15	F 2136	DSM-III and	F 0.70[4]
					DSM-III-R modified	F 0.84[4,5]
Rastam & Gillberg (1992)	1987	Sweden (Göteborg)	17		DSM-III-R	F 0.94[4]
						F 1.08[4,5]
Bland *et al* (1988)	1983–86	Canada (Edmonton)	18+	3258	DSM-III	0.100
Lucas *et al* (1991)	1985	USA: Rochester, Minnesota	All	F 32 353	DSM-III-R	F 0.270[3]
			15–19			F 0.480
Hoek (1991)	1985–86	Netherlands (58 general practices)	All	151 781	DSM-III	0.0184
Suzuki *et al* (1990)	1986–87	Japan (5 junior high schools Gunma prefecture)	12–15	1992	Clinical	F 0.000
Wells *et al* (1989)	1986	New Zealand (Christchurch)	18–64	1498	DSM-III	0.100[6]
			25–44			F 0.300[6]
Ben Tovim & Morton (1990)	1987	Australia (South Australia)	12–18	F 5705	DSM-III	F 0.105
Johnson-Sabine *et al* (1988)		UK (8 London schools)	14–16	F 1010	Clinical	F 0.000
Patton *et al* (1990)		UK (8 London schools)	15–17	F 176	Russell	F 0.2[2,4]
						F 0.000
Lewinsohn *et al* (1993)	1987–89	USA (Oregon)	15–18	1710	DSM-III-R	0.000[7]
				F 891		F 0.450[6]
Scheinberg *et al* (1992)	1989	Israel (female army recruits)	18	F 1112	DSM-III-R	F 0.180
Beglin & Fairburn (1992)		UK (2 family practices)	16–35	F 285	DSM-III-R	F 0.23[4,7]
						F 1.40[4,6]
Whitehouse *et al* (1992)	1987	UK (3 general practices)	16–35	F 540	DSM-III-R	F 0.18[2,4,7]
						F 0.37[2,4,6]
de Azevedo & Ferreira (1992)	1987	Portugal (secondary school in Azores)	12–20	F 654	DSM-III	F 0.00
						F 0.76[5]
Rathner & Messner (1993)	1988 (?)	Italy (4 German-speaking schools in Southern Tyrol)	11–20	F 517	DSM-III-R	F 0.58
			11–14	F 286	DSM-III-R	F 0.00
			15–20	F 231	DSM-III-R	F 1.30

(continued)

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Table 2 *(continued)*

Reference	Period	Place	Age group	Sample size	Diagnostic criteria	Rate/100
Götestam & Agras (1995)	1992	Norway	18–59	F 1849	DSM-III-R	F 0.27[7] F 0.43[6]
			18–29	F 556	DSM-III-R	F 0.36[7] F 0.90[6]

1. Much higher rates (1%) are often reported for this study. These estimates were, however, wrongly calculated by the authors since they were confounded with the period of observation (3–6 year rates) and by the duration of the disease (the contribution of each case to the numerator was weighed by the number of years of the disease). The rate reported in this table was compiled by the author from the original data (Table II, p. 552) and corresponds to the rates found in Table I (p. 551) of the original paper under the heading "presenting cases per 1000 girl-years".
2. Rates computed by the author.
3. Age-adjusted rate to the 1970 US female white population.
4. Rates computed after taking into account information on non-respondents.
5. This rate takes into account 'partial' syndromes (see text).
6. Lifetime prevalence.
7. Current prevalence.

F: indicates that numbers and rates are for females only; otherwise the figures are for both sexes combined.

higher prevalence rates. In each study where age-specific rates were derived among girls, prevalence rates consistently peaked in the 15–19-year-old age band, with the exception of one Swedish study where the prevalence reached a peak at the age of 14 (Rastam & Gillberg, 1992). These findings are consistent with those from incidence studies (see Table 1), and with findings derived from clinical samples.

Non-response bias

Non-response bias could be assessed by some authors and, whenever it was possible to take into account information about non-respondents, we computed a rate controlling for non-response. Thus, in three studies, the prevalence of AN among survey respondents was nil, although some non-respondents were clear-cut cases of AN (Johnson-Sabine *et al*, 1988; Beglin & Fairburn, 1992; Whitehouse *et al*, 1992). However, other surveys have not found a similar association between participation and morbidity estimated by the body mass index (Whitaker *et al*, 1990).

The use of confidence intervals

Most surveys identified only a handful of cases and, as a consequence, there is a relative lack of precision in the estimation of rates. Unfortunately, rates have been rarely reported with their 95% confidence intervals. For example, the two studies that gave rise to prevalence rates over 1% for relatively representative female samples were based on 3 cases (Rathner & Messner, 1993) and 23 cases (Rastam & Gillberg, 1992). The corresponding 95% confidence intervals (computed by us, using exact limits for the binomial distribution; Armitage & Berry, 1987) are 0.27–3.75% and 0.69–1.48% respectively. In the latter study, the matter is even more complicated as the authors used three different case definitions. The rate of 1.08% corresponds to the inclusion of *partial* syndromes of AN, i.e. cases who did not meet all diagnostic criteria (in this instance, there was no weight loss of more than 15%). If full syndromes only ($n = 20$) are considered, the rate could be estimated at 0.94% (95% CI: 0.57–1.34%). Finally, if cases are defined by the most stringent DSM-III diagnostic criteria ($n = 15$), the rate of AN among 17-year-old girls is 0.70% (95% CI: 0.39–1.10%). This illustrates how rates vary by a factor of 1.5 within the same investigation, depending upon the particular definition that is applied. Reliance on a 'partial' definition for AN has inflated rates in other investigations as well (de Azevedo & Ferreira, 1992).

The data from the 29 studies included in Table 2 were summarised by computing the median prevalence rate across studies. For each study, the rate specific to the 15–20-year-old age group (or the closest approximation of that age band) was selected, when these age-specific rates were available. The study by Wells *et al* (1989) was excluded as only lifetime rates were available. Studies that were only follow-up studies of prior samples (Patton *et al*, 1990), or that led to several published rates (Rastam *et al*, 1989), were excluded for obvious reasons. In those studies where separate rates had been produced for high-risk samples (Crisp *et al*, 1976; Szmukler, 1983), only the rates corresponding to the more representative samples were included. Although part of the same nationwide survey of psychiatric disorders, the study by Robins *et al* (1984) was treated as three different

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investigations as it was conducted in three different sites, each with a large sample size. For the 28 studies analysed, the median prevalence estimate of AN for 15–20-year-old women was 1.3/1000. Half of these studies yielded rates that ranged between 0.35/1000 and 2.39/1000. The three studies conducted in Asian countries (Lee *et al*, 1987; Hwu *et al*, 1989; Suzuki *et al*, 1990) yielded rates that were virtually nil, supporting the view that cross-cultural differences exist in the prevalence of AN. When these three studies were excluded, the median prevalence rate for AN was 1.8/1000 for the remaining 25 studies.

In order to gauge time trends, we computed the median rate for the 12 studies conducted before 1985 and for the 13 studies conducted from 1985 onwards (again excluding the studies by Lee *et al*, 1987; Hwu *et al*, 1989; Wells *et al*, 1989 and Suzuki *et al*, 1990 for the aforementioned reasons). For the first period, the median prevalence rate was found to be 1.3/1000, whereas it was 1.8/1000 in the most recent decade. Thus, bearing in mind that in the group of 12 studies conducted earlier several rates had been obtained from surveys of mostly adult respondents (Robins *et al*, 1984) or from psychiatric case registers (Nielsen, 1990; Joergensen, 1992), both of which tended to yield lower prevalence rates, there was therefore no evidence of an increase in prevalence rates of AN over the last 20 years. It should also be noted that in the Epidemiological Catchment Area study, designed to assess with precision rates of psychiatric disorders among American adults (Robins & Regier, 1991), only 11 cases of AN were detected in a sample of nearly 20 000 persons; accordingly, prevalence rates could barely be estimated on three sites (see Table 2; Robins *et al*, 1984). Because this was a very large general population survey that is judged to have provided good data on most psychiatric disorders, its finding of a very low rate of prevalence of AN must be given considerable weight.

Discussion

AN is a rare condition, so that secular changes in its incidence or prevalence are difficult to detect. Since most surveys yield prevalence rates around 1–2/1000 women, sample sizes are too small to support comparisons between rates for different birth cohorts that might otherwise provide evidence on time trends. Comparisons between surveys carried out at different times, although weakened by differences in the methods used, suggest that prevalence rates probably remained stable over the 1970s and 1980s. However, it should be acknowledged that the time span encompassed by community surveys is relatively narrow, thereby reducing the opportunity to detect secular changes in rates of disorder.

Incidentally, studies of referrals to treatment centres have provided additional support to the conclusion derived from our review of cross-sectional epidemiological surveys. Thus, referral rates for AN in an urban area of New Zealand remained fairly stable over a 10-year period (Hall & Hay, 1991) as they were in two Canadian specialised treatment centres for eating disorders from 1972 to 1986 (Garner *et al*, 1985; Garner & Fairburn, 1988).

Studies based on records kept by health services are a richer source of information on time trends than surveys. Incidence rates derived from the same registers at different periods have shown an apparent increase over time. However, over the period covered by these studies, knowledge and recognition of the disorder have considerably improved, and diagnostic definitions, availability of services and patterns of care have also changed substantially. Therefore, it is not possible to assess how far changes in rates derived from health services records reflect changes in the response to the condition as opposed to changes in its incidence. Several studies have shown that the effect of such artefacts may indeed have been great enough to create a false impression of a secular increase in the incidence of AN.

The most robust data are those from Rochester (Lucas *et al*, 1991), which were based on a range of health service records. The findings show a linear increase in rates of AN from 1950 to 1984 among 10–19-year-old women, although the apparent increase is very small in magnitude. However, because of some unresolved analytical problems, these findings must be interpreted with caution. Furthermore, this whole time-series consisted of only about 100 cases. If the results from 'positive' studies were to be weighted by the number of cases recorded, the evidence from studies showing no increase over time would be overwhelming.

The finding from a single site is not sufficient to support the conclusion that there was a true increase in the incidence of AN in the modern era, particularly in view of the methodological limitations discussed above. The apparent increase in Rochester (Minnesota) would have to be confirmed by data for more recent periods in the same area and the study would have to be replicated in other areas before it could be concluded with confidence that an actual increase in the incidence of AN had occurred. In any case, current data suggest that any increase over time could not have been large.

https://doi.org/10.1192/bjp.166.4.462 Published online by Cambridge University Press

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(First received 3 May 1994, final revision 20 July 1994, accepted 12 September 1994)

https://doi.org/10.1192/bjp.166.4.462 Published online by Cambridge University Press