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Assessment of Excess Mortality in Obesity

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Quantification of the excess mortality from all causes associated with obesity remains controversial. In this paper, 6,193 obese patients, those with a body mass index (weight (kg)/height (m)^2 (BMI)) range of 25–74 kg/m^2, recruited from 1961 to 1994 in Düsseldorf, Germany, were followed for a mean time of 14 (standard deviation, 8.2) years, yielding 87,179 observed patient-years. During the study period, 1,028 patients (16.6%) died. The entire cohort was grouped into approximate quartiles according to BMI: group 1, BMI from 25 to <32; group 2, BMI from 32 to <36; group 3, BMI from 36 to <40; group 4, BMI ≥ 40 kg/m^2. The following risk ratios were estimated by means of Cox proportional hazards models using the lowest BMI group as reference category: group 2 for men, 1.02 (95% confidence interval 0.76–1.37); for women, 1.23 (95% confidence interval 0.96–1.58); group 3 for men, 1.50 (95% confidence interval 1.09–2.06); for women, 1.33 (95% confidence interval 1.03–1.73); and group 4 for men, 2.10 (95% confidence interval 1.53–2.88); for women, 2.25 (95% confidence interval 1.78–2.84). The following standardized mortality ratios were calculated by using the respective geographic area (the Federal State of North Rhine Westphalia) as reference population: group 1 for men, 1.26 (95% confidence interval 0.98–1.61); for women, 1.00 (95% confidence interval 0.81–1.23); group 2 for men, 1.31 (95% confidence interval 1.09–1.57); for women, 1.20 (95% confidence interval 1.02–1.40); group 3 for men, 1.92 (95% confidence interval 1.53–2.38); for women, 1.27 (95% confidence interval 1.07–1.50); and group 4 for men, 3.05 (95% confidence interval 2.47–3.73); for women, 2.31 (95% confidence interval 2.04–2.60). In addition to age, sex, and BMI, Cox proportional hazards models revealed systolic blood pressure, glucose intolerance, diabetes, and smoking as significant independent mortality risk factors, whereas cholesterol was not significant. In this prospective study of a large cohort of obese persons, morbid obesity (BMI of ≥40 kg/m^2) was a strong predictor of premature death. Excess mortality risks associated with gross obesity (BMI from 32 to <40 kg/m^2) were considerably lower than hitherto assumed; moderate degrees of obesity (BMI from 25 to <32 kg/m^2) were not significantly associated with excess mortality. *Am J Epidemiol* 1998;147:42–8.

mortality; obesity; obesity in diabetes; obesity, morbid; proportional hazards models; survival analysis

Quantification of the mortality risk associated with obesity remains controversial. Studies show conflicting results as to whether increasing relative body weight is associated with excess mortality in both sexes, at all ages, from any genetic background, and at all degrees of obesity. Much of the current knowledge still relies upon the positive relation between relative body weight and mortality postulated on the basis of the data accumulated by the North American life insurance companies in the middle of this century (1). More recently, several prospective cohort studies aiming to describe the health hazards of being overweight have confirmed that obesity is associated with excess mortality (2–9). The conclusions of these studies were, however, controversial. Thus, recently published data from the Nurses' Health Study (7) suggested a statistically significant increase in mortality for all women (after controlling for smoking habits and other potential confounders) associated with a body mass index (weight (kg)/height (m)^2 (BMI)) above 27 kg/m^2, when compared with the leanest women (BMI below 19 kg/m^2). There was, however, no stratification of women with a BMI above 32 kg/m^2, and there was the problem of self-reported body weight data. Similarly, a 27-year follow-up of Harvard alumni indicated an increased mortality risk associated with an initial BMI above 26 kg/m^2 when compared with a reference group of men with a BMI below 22.5 kg/m^2 (6). In contrast, two large population studies suggested a sig-

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Abbreviations: BMI, body mass index; SD, standard deviation; SMR, standardized mortality ratio.

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nificant obesity-associated excess mortality above 30 kg/m^2 in men (without any obesity-associated mortality risk in women) (8) or above 31 kg/m^2 in men (4). These data were based upon follow-up studies of segments of general populations and of professional or employment groups, with only a minor fraction representing persons with gross obesity (BMI $\geq$ 32 kg/m^2). The mortality risk of obesity was assessed by comparing the death rates in persons with higher BMI classes with the mortality found in the segments of lean persons within the cohorts studied. In this study, we have compared the risk of premature death in a large cohort of obese patients, including a considerable number of morbidly obese (BMI $\geq$ 40 kg/m^2) individuals, with that of the general population.

MATERIALS AND METHODS

Study population and data

The Düsseldorf Obesity-Mortality-Study (DOMS) is a prospective cohort study of 6,193 obese patients (1,591 men, 4,602 women) that is based upon all consecutive patients who had been referred between 1961 and 1994 to the obesity clinic of this Department that serves as a tertiary care center for the metropolitan area of Düsseldorf and its surroundings. In general, the referral of overweight patients to the obesity clinic was made by their general practitioners in order to subject them to a 1,000-calorie dietary treatment, including elements of group therapy and behavior modification (10), often on the patients' own initiative; some patients were also referred by this Medical School's endocrine clinic or by surgical departments to achieve weight loss before elective surgery. All services of the obesity clinic are provided free of charge to the patient. The initial medical examination includes a case history, physical examination, and clinical chemistry analysis. Inclusion criteria for recruitment into the study were an age at entry between 18 and 75 years and a BMI of $\geq$25 kg/m^2. The following baseline information measured at the initial medical examination was extracted from the patients' records kept at the obesity clinic: date of examination, name, current address, sex, birthday, height, weight, blood pressure, glucose tolerance, and serum cholesterol level. Since 1977 additionally, information on smoking habits was collected systematically. Height and weight were measured with the patients in light clothes (shirts and trousers or skirts) without shoes. Blood pressure was measured by means of a mercury sphygmomanometer and serum cholesterol and blood glucose by routine clinical chemistry methodology, as described previously (11). Glucose tolerance was assessed by measuring the 2-hour capillary blood glucose level following a 100-g oral glucose load given after an overnight fast. Subsequently, the binary variables *diabetes yes/no* and *impaired glucose tolerance yes/no* were formed. A patient was classified as having diabetes if the diagnosis of diabetes was previously known, if fasting blood glucose was $\geq$ 6.7 mmol/liter (120 mg/dl), or if the glucose tolerance test yielded capillary blood glucose values of $\geq$11.1 mmol/liter (200 mg/dl). Glucose tolerance was classified as impaired if the glucose tolerance test yielded 2-hour capillary blood glucose values of $\geq$7.8 mmol/liter (140 mg/dl) and $<$11.1 mmol/liter (200 mg/dl). Thus, a patient was classified as having normal glucose tolerance if a glucose tolerance test resulted in 2-hour blood glucose values below 7.8 mmol/liter (140 mg/dl). After 1972, all patients had signed an agreement that their data could be used in the context of scientific studies.

Mortality follow-up

Vital status was ascertained three times from municipal residents' registries: at first up to 1977, secondly up to 1984, and finally up to 1994. In the final follow-up, the vital status of 5,775 patients (93.3 percent) could be determined. Additionally, the vital status of 278 patients up to any time point earlier than 1994 was obtained from the former follow-up investigations or from removal dates. Thus, we could calculate patient-year data based upon 6,053 patients (97.7 percent). Only 140 patients (2.3 percent) contributed no information regarding survival. All available survival data were used either as event or as censored observation. The patients' survival time was calculated on a daily basis.

Statistical analysis

In order to investigate the association between weight and mortality, we grouped the study population into approximate fourths according to BMI: group 1, from 25 to $<$32; group 2, from 32 to $<$36; group 3, from 36 to $<$40; and group 4, $\geq$40 kg/m^2. The exact quartile limits were not used because they were dependent on the cohort considered (entire cohort, men, women, subcohort with information about smoking, and so on). It seemed to be preferable to use the same BMI groups throughout to compare the results for all subcohorts analyzed. Overweight of BMI from 25 to $<$32 kg/m^2 (group 1) was referred to as *moderate obesity*, of BMI from 32 to $<$40 kg/m^2 (groups 2 and 3) as *gross obesity*, and of BMI $\geq$40 kg/m^2 (group 4) as *morbid obesity*.

The Cox proportional hazards model (12) was applied to estimate risk ratios for BMI groups 2–4 using BMI group 1 as reference category and to examine the

effects of blood pressure, glucose tolerance, cholesterol, and smoking on mortality. We applied different models having different sets of covariates separately and in common for men and women. The BMI was included in the models by means of three dummy variables for the last three BMI groups, using the least obese group as the reference category. Age, systolic blood pressure, and cholesterol were included as continuous covariates. Diabetes and impaired glucose tolerance were dichotomous variables, using normal glucose tolerance as the reference category. Smoking was a dichotomous variable, using never smoking as the reference category. The proportional hazards assumption was checked by plotting the *log* of the *negative log* of the estimated survival functions against *log* time. Interactions were investigated by testing the first-order cross-product terms.

We calculated standardized mortality ratios (SMRs) (13) stratified by sex and BMI group by using the male and female population of North Rhine Westphalia as reference populations, respectively. North Rhine Westphalia has a population of around 17 million people; in 1994, the mean life expectancy of the German population was 73.4 years for men and 79.7 years for women (14). Based upon data from the MONICA project, the median BMI of the German population aged 35–64 years can be estimated to be around 26 kg/m^2 for men and 25 kg/m^2 for women (15). The SMRs permit a comparison of the mortality of the obese study population with the mortality of a complete standard population living in the same geographic area. The mortality ratios were standardized in an indirect manner (13) according to age and calendar year using 1-year intervals. The population and mortality data of North Rhine Westphalia on a 1-year basis for age and calendar year were made available from the State Office for Data Processing and Statistics of North Rhine Westphalia, Düsseldorf. Significance tests and 95 percent confidence intervals for the SMRs were calculated by using Byar's approximation to the exact Poisson test and the exact Poisson limits (13). The percentage of attributable risk was calculated by means of the functional relation, attributable risk = (risk ratio − 1)/risk ratio (16).

For computations, the SAS procedures UNIVARIATE, MEANS, FREQ, LIFETEST, and PHREG were used (SAS Institute, Inc., Cary, North Carolina). SMRs and the corresponding p values and confidence intervals were calculated by means of our own programs written in matrix language (SAS/IML).

RESULTS

The patients were followed up for a maximum time of 33 years with a mean of 14 (standard deviation (SD), 8.2) years. The range for BMI was 25.0–74.4 kg/m^2 (mean, 36.6; SD, 6.1 kg/m^2) and for age was 18–74 years (mean, 40.4; SD, 12.9 years). Up to 1994, 1,028 (16.6 percent) patients died (365 men, 663 women). The total number of observed patient-years was 87,179 (men, 21,932; women, 65,247 patient-years). A descriptive analysis of the baseline data is shown in table 1.

At first, the conformity of the data with the assumptions of the Cox proportional hazards model was investigated. The plots of the *log* of the *negative log* of the estimated survival functions against *log* time showed no clear violations of the main model assumption, with the exception that BMI should not be treated as a continuous covariate. Hence, in all models, BMI was included by means of dummy variables. As all interactions were not significant ($p > 0.3$ for all interaction terms), the following models contained only main effects. Separate models for men and women were calculated, aiming at the estimation of relative risks for the BMI groups adjusted for period effect and age. As the period effect (date of examination) was not significant, it was dropped from the models. Hence, only age and the dummy variables for the BMI groups served as covariates. The results of these models for men and women are shown in table 2. As the reference category was the least obese group (BMI from 25 to <32 kg/m^2), the relative risk for this group is 1 by definition. Besides age, sex, and BMI, other covariates may have an independent effect on mortality. To assess the effect of other covariates, we used common proportional hazards models including both sexes, because separate models for men and women were based upon an insufficient number of events due to missing values in the covariates and were therefore unreliable. In a common proportional hazards model, systolic blood pressure, impaired glucose tolerance, and diabetes were identified as significant predictors of mortality (table 3). An analysis excluding early mortality (death within 2 years after the first visit) did not change the main results. The effect of serum cholesterol levels was not significant as either a continuous or a binary variable using different cutoff points. However, it should be noted that cholesterol data were missing for nearly 30 percent of the patients (table 1). Additionally, the effect of smoking was assessed, although information on smoking habits was not available for approximately 40 percent of the patients. The respective model containing sex, age, systolic blood pressure, glucose tolerance, diabetes, BMI groups, and smoking as covariates was of limited size (2,215 patients and 139 events), and the results must be interpreted carefully. However, it can be considered as an analysis of the subcohort recruited

TABLE 1. Descriptive analysis of baseline data within body mass index (BMI) groups, Düsseldorf, Germany, 1961-1994*

BMI group (kg/m²)	No.	BMI (kg/m²)		Weight (kg)		Height (cm)		Age (years)		Blood pressure (mmHg)				Cholesterol (mmol/liter)		Diabetes (no.)	IGT† (no.)	Ever smoking (no.)
										Systolic		Diastolic						
		Mean	SD†	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD			
Men																		
Total	1,591	36.6	5.9	112.6	20.3	175.3	7.1	39.7	13.2	161.4	26.9	100.8	17.3	6.21	1.51	304 (22)‡	217 (15)	341 (65)
25–<32	316	30.1	1.4	92.8	8.5	175.3	6.9	41.5	13.6	153.1	24.2	95.3	15.1	6.26	1.42	65 (24)	39 (14)	73 (65)
32–<36	548	34.0	1.1	104.8	9.4	175.4	7.4	40.4	13.8	158.9	25.2	99.1	16.7	7.11	1.79	105 (22)	66 (14)	105 (64)
36–<40	376	37.8	1.1	116.2	10.1	175.1	7.0	38.8	13.8	162.1	27.0	101.7	16.2	6.13	1.26	65 (20)	50 (15)	87 (67)
≥40	344	45.4	5.1	139.5	19.4	175.2	7.0	37.6	11.3	172.1	28.5	107.5	18.9	5.92	1.33	66 (21)	62 (20)	76 (65)
Women																		
Total	4,602	36.6	6.2	97.2	17.4	162.8	6.7	40.7	12.8	158.4	28.3	97.8	15.9	5.95	1.29	602 (15)	696 (17)	639 (37)
25–<32	1,122	29.7	1.6	80.2	7.4	164.2	6.5	39.0	12.6	146.7	23.6	92.1	13.4	5.89	1.29	95 (10)	129 (13)	164 (37)
32–<36	1,312	34.0	1.2	90.4	7.9	162.9	6.6	40.6	13.3	155.5	25.9	96.2	14.5	6.03	1.34	161 (14)	197 (17)	178 (35)
36–<40	972	37.9	1.2	100.2	8.7	162.5	6.7	41.4	12.9	159.8	27.5	98.8	14.9	5.96	1.31	129 (15)	147 (17)	128 (38)
≥40	1,191	45.0	4.6	117.9	14.9	161.8	4.6	41.5	12.2	169.8	31.5	104.2	17.9	5.90	1.21	217 (20)	223 (21)	169 (39)

* Values are missing for body mass index (n = 12), blood pressure (n = 260), glucose tolerance (n = 678), cholesterol (n = 1,724), and smoking (n = 3,937).
† IGT, impaired glucose tolerance; SD, standard deviation.
‡ Numbers in parentheses, percentage.

after 1976. In this model, smokers (smokers and ex-smokers at baseline) had a significantly higher mortality than did nonsmokers (table 4). The results for the other covariates were similar to those obtained before (table 3), with the exception of quite larger confidence intervals due to the smaller sample size.

The estimated SMRs for men and women are shown in table 5. The mortality of the male and female BMI group from 25 to <32 kg/m^2 was not significantly different from the expected mortality of these groups calculated from the male and female population of North Rhine Westphalia, respectively. Gross and morbid obesity (BMI of ≥32 kg/m^2) was associated with significant excess mortality in both men and women. A substantial excess mortality (SMR > 1.5), however, was documented only for men with BMIs above 36 kg/m^2 and for women above 40 kg/m^2. In terms of attributable risk, 67 percent of the deaths among morbidly obese men and 57 percent of the deaths among morbidly obese women can be attributed to their obesity.

DISCUSSION

This study represents by far the largest mortality follow-up of a cohort of obese patients. According to the study protocol, any bias of weight reporting depending on illness, due to expected financial consequences (as in life insurance data) or due to self-reporting, was ruled out.

We have compared the mortality rates of this cohort of obese patients with those of the general population of North Rhine Westphalia, the respective State of the Federal Republic of Germany, stratified for sex and standardized for age and calendar year. On quantitative comparison of the mortality rates of the study population and of the general population, SMRs for the morbidly obese patients (BMI ≥ 40 kg/m^2) were 2- to 3-fold elevated. This finding is compatible with earlier observations on morbidly obese patients (17–19) that, due to the small numbers of subjects, did not permit any reliable estimation (19). However, moderately obese persons (BMI from 25 to <32 kg/m^2) had no significantly increased mortality risk. While the corresponding confidence interval for moderately obese men was too wide to rule out an excess mortality, it can be concluded that the SMR for moderately obese women is lower than 1.2, with an estimate very close to 1. In grossly obese women (BMI from 32 to <40 kg/m^2), mortality was only moderately (i.e., SMR of 1.20 and 1.27, respectively) increased, whereas in men the excess mortality associated with BMI indices from 32 to <36 (SMR of 1.31) and from 36 to <40 kg/m^2 (SMR of 1.92) was markedly higher. Likewise, in the internal comparison, morbid obesity (BMI ≥ 40 kg/m^2) increased the risk of premature death substan-

TABLE 2. Results of separate proportional hazards models for men and women, Düsseldorf, Germany, 1994

Variable	Unit	Men (n = 1,584)			Women (n = 4,597)		
		RR*	95% CI*	p value	RR	95% CI	p value
Age	10 years	2.25	2.06–2.46	<0.0001	2.31	2.15–2.47	<0.0001
BMI*							
32–<36	vs. BMI <32	1.02	0.76–1.37	0.9056	1.23	0.96–1.58	0.1020
36–<40	vs. BMI <32	1.50	1.09–2.06	0.0119	1.33	1.03–1.73	0.0283
≥40	vs. BMI <32	2.10	1.53–2.88	<0.0001	2.25	1.78–2.84	<0.0001

* RR, risk ratio; CI, confidence interval; BMI, body mass index (weight (kg)/height (m)^2).

TABLE 3. Results of a common proportional hazards model including both sexes (n = 5,404), Düsseldorf, Germany, 1994

Variable	Unit	RR*	95% CI*	p value
Sex	Male vs. female	2.73	2.36–3.17	<0.0001
Age	10 years	2.10	1.97–2.25	<0.0001
Systolic blood pressure	10 mmHg	1.07	1.04–1.10	<0.0001
Impaired glucose tolerance	vs. normal glucose tolerance	1.25	1.03–1.51	0.0250
Diabetes	vs. normal glucose tolerance	1.85	1.57–2.18	<0.0001
BMI*				
32–<36	vs. BMI <32	1.10	0.87–1.38	0.4327
36–<40	vs. BMI <32	1.31	1.04–1.67	0.0245
≥40	vs. BMI <32	1.92	1.53–2.42	<0.0001

* RR, risk ratio; CI, confidence interval; BMI, body mass index (weight (kg)/height (m)^2).

tially, but obesity with a BMI from 32 to <36 kg/m^2 was not a significant predictor of mortality in either men or women, when compared with the BMI class from 25 to <32 kg/m^2.

It is debatable whether one should control for possible sequelae of obesity, such as hypertension and diabetes/glucose intolerance, in such a study, because those variables might represent possible intermediate steps (i.e., causal links) between obesity and its associated excess mortality risk. When comparing the obesity cohort of this study with the general population, such analyses were not possible. Thus, the mortality risk may have been disproportionally elevated in some subgroups of the cohort of this study, such as those patients with manifest diabetes or hypertension or other similarly well-established predictors of mortality. In multiple regression analyses, we were actually able to document that glucose intolerance, diabetes mellitus, blood pressure, and smoking were statistically significant independent predictors of mortality in this cohort of obese patients whereas, in this internal analysis, the relative risks of BMI categories were confirmed.

The following limitations of our study have to be considered. Systematically obtained information on possibly important confounders, such as alcohol con-

TABLE 4. Results of a proportional hazards model including smoking (n = 2,215), Düsseldorf, Germany, 1994

Variable	Unit	RR*	95% CI*	p value
Sex	Male vs. female	2.98	2.03–4.37	<0.0001
Age	10 years	2.19	1.84–2.81	<0.0001
Systolic blood pressure	10 mmHg	1.06	0.99–1.12	0.0917
Impaired glucose tolerance	vs. normal glucose tolerance	1.55	1.03–2.34	0.0345
Diabetes	vs. normal glucose tolerance	1.58	1.03–2.43	0.0348
Ever smoking	Yes vs. no	1.83	1.20–2.80	0.0052
BMI*				
32–<36	vs. BMI <32	1.06	0.64–1.76	0.8090
36–<40	vs. BMI <32	1.45	0.85–2.47	0.1711
≥40	vs. BMI <32	1.74	1.05–2.89	0.0327

* RR, risk ratio; CI, confidence interval; BMI, body mass index (weight (kg)/height (m)^2).

TABLE 5. Standardized mortality ratios (SMRs),* Düsseldorf, Germany, 1994

BMI† group (kg/m²)	Men			Women		
	SMR	95% CI†	p value	SMR	95% CI	p value
25–<32	1.26	0.98–1.61	0.0744	1.00	0.81–1.23	0.9844
32–<36	1.31	1.09–1.57	0.0053	1.20	1.02–1.40	0.0256
36–<40	1.92	1.53–2.38	<0.0001	1.27	1.07–1.50	0.0073
≥40	3.05	2.47–3.73	<0.0001	2.31	2.04–2.60	<0.0001
Total	1.67	1.51–1.85	<0.0001	1.45	1.34–1.57	<0.0001

* SMRs were calculated by using the male and female populations of North Rhine Westphalia as reference populations, respectively.
† BMI, body mass index; CI, confidence interval.

sumption, medication, body fat distribution, obesity-associated symptoms, and psychologic variables as well as the patients' social status and physical activity level, was insufficient to be included in the analysis of this large cohort recruited over a period of almost 35 years. No systematic information is available as to the course of obesity after recruitment of the patients; however, earlier analyses of subgroups of patients indicate that the overall long-term effect on weight reduction by our obesity clinic intervention was almost negligible. As in other comparable reports, around 50 percent of the patients did not attend the therapeutic program after the initial examination, and, for the remaining patients, a significant long-term weight reduction was demonstrable in less than 5 percent of the patients (10). Furthermore, we did not have the possibility to obtain reliable information on the causes of death. According to the prevailing law in Germany, after 1982 we were denied access to the death certificates of the patients in this study. Finally, the study cohort does not represent a random sample of the obese population, creating a potential for selection bias. (Self-)referral patterns may vary between subgroups of the study cohort, for example, men versus women, according to age group, concomitant diseases or symptoms, health beliefs of patients and their physicians, and according to the extent of obesity. Differential referral patterns may create spurious associations or mask existing ones. Because we were not able to recruit a representative sample of the obese population, there are no data to compare our cohort of obese people with the obese source population. More women than men participated. The relation between age and obesity was flat for women but inverse for men. It is fair to assume, however, that those obese patients who were referred to our obesity clinic not only tended to be more concerned about their health but also had more problems due to symptoms and diseases associated with obesity than did the remaining population of obese patients living in this geographic area. This interpretation is supported by the high mean values of blood pressure and the high proportion of people with diabetes and impaired glucose tolerance in our cohort. A part of the large excessive mortality risk in obese men compared with women could thus be due to a higher proportion of referrals after the onset of obesity-associated disorders or symptoms in men than in women. This is underlined by the facts that mean values of blood pressure are higher and that the proportion of diabetic individuals is greater in men than in women, whereas, in the general population, more women than men have diabetes. The inverse relation between age and BMI in men also indicates that many relatively young men were referred because of signs and symptoms related to overweight. This is consistent with the observation that men often seek medical advice only after the onset of cardiovascular signs and symptoms, whereas many women are interested in weight loss because of prevailing ideals of beauty.

Despite these limitations of the study, we conclude (consistent with some more recent population-based studies (4, 8)) that moderate obesity (BMI from 25 to <32 kg/m^2) is not associated with a substantial excess mortality and that the mortality risk of gross obesity (BMI from 32 to <40 kg/m^2) appears to be considerably lower than previously assumed (5, 20), especially in women. The excess mortality risk of morbid obesity (BMI $\geq$ 40 kg/m^2) was quantified as 3-fold in men and more than 2-fold in women.

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