

Redefining overweight and obesity in rheumatoid arthritis patients

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Objectives: To assess whether body mass index (BMI) and body fat (BF) differ between rheumatoid arthritis (RA) patients, patients with non-inflammatory arthritis (osteoarthritis, OA) and healthy individuals, and whether disease specific measures of adiposity are required to accurately reflect BF in these groups.

Methods: 641 individuals were assessed for BMI (kg/m^2) and BF (bioelectrical impedance). Of them, 299 (174 RA, 43 OA and 82 healthy controls (HC)) formed the observation group and 342 (all RA) the validation group. RA disease characteristics were collected.

Results: ANOVA revealed significant differences between disease groups for BMI ($p < 0.05$) and BF ($p < 0.001$). ANCOVA showed that age accounted for the differences in BMI ($F_{1,294} = 5.10$, $p < 0.05$); age ($F_{1,293} = 22.43$, $p < 0.001$), sex ($F_{1,293} = 380.90$, $p < 0.001$) and disease ($F_{2,293} = 18.7$, $p < 0.001$) accounted for the differences in BF. For a given BF, patients with RA exhibited BMI levels reduced by $1.83 \text{ kg}/\text{m}^2$ ($p < 0.001$) compared to HC; there were no significant differences between OA and HC. A predictive model for BF was developed ($R^2 = 0.769$, $p < 0.001$) and validated using limits of agreement Analysis against measured BF in the validation group ($95\% \text{ LIM}_{\text{AG}} = 6.17$; $\text{CV} = 8.94$).

Conclusions: In individuals with RA, BMI cut-off points should be reduced by $2 \text{ kg}/\text{m}^2$ (that is, to $23 \text{ kg}/\text{m}^2$ for overweight and $28 \text{ kg}/\text{m}^2$ for obesity). The equation developed can be used to accurately predict BF from BMI in RA patients. These findings may be important in the context of the cardiovascular comorbidity of RA.

Excess body fat (BF) is a prominent health hazard¹ significantly contributing to the development of cardiovascular disease (CVD).² About two-thirds of patients who have had a myocardial infarction (MI) exhibit increased body weight.³ Obesity increases the risk of coronary heart disease (CHD) through a number of different pathophysiological pathways, including insulin resistance, type 2 diabetes, hypertension and dyslipidaemia.^{4–5}

Assessments for overweight or obesity include the calculation of body mass index⁶ (BMI, in kg/m^2) or more accurate estimations of relative adiposity (BF percentage) through a number of techniques (for example, skinfold thickness, hydrostatic weighing and bioelectrical impedance).⁷ BF estimations require sophisticated equipment and trained personnel, whereas BMI is easy to obtain and is widely used in the routine clinical setting.

In the general population, BMI of $< 25 \text{ kg}/\text{m}^2$, $25\text{--}30 \text{ kg}/\text{m}^2$ and $> 30 \text{ kg}/\text{m}^2$ indicate healthy, overweight, and obese individuals and associate with low, medium and high CVD risk, respectively.^{8–9} However, BMI is only a proxy of body fat,⁶ and over recent years its validity has been questioned.^{3–7, 10–13} Overweight as defined by BMI of $> 25 \text{ kg}/\text{m}^2$, has poor specificity in detecting excess body fat in healthy men and women of all ages⁶ as well as in patients with coronary heart disease.³ In specific subpopulations, such as people of Indian-Asian race,¹⁰ women^{11–13} and large size athletes,⁷ new BMI cut-off points have been suggested that optimally reflect BF and may better predict CVD risk.

The weakness of BMI is that it does not distinguish between lean body mass and fat mass. Consequently people of similar stature and weight, but different muscle content, will have the same BMI but different BF levels. This tends to be more evident in individuals with low BMI levels.⁶ Such limitations of the BMI may explain the better cardiovascular outcomes observed in overweight and mildly obese patients with established CHD

compared to their normal weight counterparts, who may have proportionately more BF.³ Therefore, although it is well established that CHD risk increases with advancing BMI levels,⁹ global cut-off points may be misleading for several populations.

Central obesity poses a great risk for CVD.^{13–15} Regional fat distribution, as measured by waist to hip ratio, has been proposed as a more accurate predictor of CHD risk than BMI.^{14–15} Indeed, it has been suggested that obesity should be redefined based on waist to hip ratio instead of BMI, since waist to hip ratio is significantly associated with MI risk in most ethnic groups.¹⁶ However, its predictive strength can be negatively affected by sex and overall body weight,¹⁷ in a way that pear-shaped or obese individuals might have optimal waist to hip ratio but increased overall body weight. More research is necessary to identify the optimal definition of obesity as a predictor for CHD in the general population and specific subgroups.¹⁸

Patients with rheumatoid arthritis (RA) have an increased risk for CHD events.¹⁹ RA is a chronic inflammatory disease which affects predominantly synovial joints, causing pain, swelling, stiffness and eventually irreversible damage and deformity, all of which may lead to significant reduction in physical activity. RA associates with increased mortality particularly from CHD,¹⁹ most probably because of accelerated atherogenesis secondary to the metabolic and vascular effects of systemic inflammation.²⁰ Nearly two-thirds of all individuals with RA experience involuntary loss of fat-free mass and progressively increased fat mass in the presence of stable or even slightly decreased weight, a condition referred to as

Abbreviations: ANCOVA, analysis of co-variance; ANOVA, analysis of variance; BF, body fat; BMI, body mass index; CHD, coronary heart disease; CVD, cardiovascular disease; DAS, disease activity score; HC, healthy controls; MI, myocardial infarction; LIM_{AG} , limits of agreement; OA, osteoarthritis; RA, rheumatoid arthritis

Table 1 Demographic and disease characteristics of all volunteers (mean (SD))

	Observation group						Validation group	
	Male (n = 110)			Female (n = 189)			Male	Female
	RA	OA	HC	RA	OA	HC	RA	RA
Number	56	15	39	118	28	43	99	243
Age	60.6 (11.8)**	56.7 (13.3)*	45.1 (13.3)	59.6 (12.2)** †	52.8 (12.5)*	46.8 (11.5)	62.1 (11.6)	61.7 (11.9)
Height	173.6 (7)*	171.3 (6.7)*	177.3 (6.7)	159.1 (6.5)**	161 (5)	163.6 (6.9)	174 (6.8)	160.4 (6.7)
Weight	83.6 (13.3)	78.4 (14.8)	80.9 (11.4)	68.6 (15)	70.8 (16.5)	68.1 (16.3)	82.7 (15.8)	70.2 (14.4)
BMI	27.7 (4.3)*	26.8 (4.7)	25.7 (3)	26.9 (5.7)	27.2 (5.7)	25.4 (5.5)	27.3 (4.4)	27.3 (5.3)
BF	28.7 (7.7)**	24.8 (7.9)*	19.2 (5.2)	38.3 (7.3)** †	35.2 (8.5)	32.1 (8.2)	27 (6.4)	38.3 (7.1)
Trunkal fat	30.5 (8)**	26.6 (8.9)*	21.4 (6)	35.7 (8.6)** †	31.6 (9.6)	29.1 (8.7)	27.4 (7.7) §	35.4 (8.1)
DAS28	4.2 (1.2)			4.3 (1.4)			4.1 (1.4)	4.3 (1.4)
ESR (mm in 1st hour)	23.2 (18.5)			26 (22.1)			25.3 (21.5)	30 (26.3)
CRP	15.6 (15)			15.8 (14.9)			16.8 (18.6)	17.6 (23.6)
Disease duration	11.4 (10.2)			11.3 (9.9)			12.5 (11)	13.2 (11)

RA, rheumatoid arthritis; OA, osteoarthritis; HC, healthy controls; BMI, body mass index; BF, body fat; DAS28, disease activity score-28; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein.
 One way ANOVA: *Significant difference compared to HC (p<0.05).
 **Significant difference compared to HC (p<0.001).
 †Significant difference compared to OA (p<0.05).
 §Significant difference compared to experimental RA group (p<0.001).

rheumatoid cachexia.²¹ The exact mechanisms causing rheumatoid cachexia remain undetermined, but muscle loss due to systemic inflammation and reduced physical activity may both contribute.²²

We hypothesised that for a given BMI, RA patients exhibit significantly higher proportions of fat mass than healthy individuals, or even than patients with movement restriction due to a non-inflammatory arthritis, such as osteoarthritis (OA). The possible consequences of this, in the context of the increased CVD mortality in RA, are obvious. In the present study we aimed to investigate whether BMI and BF differ according to arthritic disease (OA vs RA) and within RA according to disease state (for example, active vs inactive, early vs established disease). We also developed and validated RA specific BMI cut-off levels and algorithms to calculate BF from BMI.

METHODS

Participants

Consecutive patients attending routine rheumatology or orthopaedic outpatient clinics at the Dudley Group of Hospitals NHS

Trust, UK, and healthy controls (hospital and university staff) were invited to participate. The study had local research ethics committee approval by the Dudley ethics committee, and all volunteers provided informed consent. The observation group (n = 299) included 174 volunteers with RA (1987 revised American Rheumatism Association criteria²³), 43 with OA of the hip²⁵ or knee,²⁶ and 82 healthy controls (individuals who by self report did not have any known clinical conditions and were taking no medication). The validation group (n = 342) consisted of RA patients only. Demographic and disease characteristics from all subjects appear in table 1.

Assessments

All volunteers were subjected to the same data collection procedures overseen by the same trained investigators. Specifically, standing height was measured to the nearest 0.5 cm on a Seca 214 Road Rod portable stadiometer. Body composition was assessed by bioelectrical impedance, using a Tanita BC-418 MA Segmental Body Composition Analyzer, which incorporates eight tactile electrodes (Tanita Corporation,

Table 2 BMI and BF of RA patients (observation group) according to categorisation based on their disease characteristics

Disease characteristics	Categories	BMI		BF	
		Male	Female	Male	Female
DAS28 (DAS28 score)	Remission (<2.6)	27.2 (3.46)	27.2 (5.6)	26.5 (7.6)	39.5 (6.7)
	Mild (2.7–3.2)	28 (4.3)	27.3 (4.6)	28 (6)	39.3 (6.6)
	Moderate (3.3–5.1)	27.8 (4.5)	27 (5.3)	27.4 (6.8)	37.3 (7.7)
	High (>5.1)	25.3 (5.5)	27.3 (5.5)	26.1 (5.6)	37.7 (7.2)
ESR (mm in 1st hour)	Normal*	27.9 (4.4)	26.9 (4.8)	27.1 (7.2)	38.3 (6.3)
	High	26.4 (4.6)	27.6 (6.1)	26.7 (5.9)	37.6 (8.9)
CRP (mg/l)	Low (<3)	26.5 (2.4)	28.3 (6.2)	25.9 (5.4)	38.5 (8.7)
	Normal (3–8)	27.8 (4.7)	26.5 (4.7)	26.7 (8)	37.6 (6.6)
	High (>8)	26.9 (4.6)	27.6 (5.7)	27.3 (5.8)	38.3 (7.9)
Disease duration (years)	Early (<3)	26.4 (5)	26.1 (5)	26.4 (7.9)	37.9 (8.3)
	Established (3–10)	28.8 (4.1)	27.8 (5.7)	27.8 (6.3)	38.2 (7.5)
	Longstanding (>10)	26.8 (4.4)	27.1 (5.1)	27.7 (5.7)	38.8 (6.7)
Rheumatoid factor	Positive	26.6 (3.6)	27.2 (5.7)	25.1 (6.7)	38.3 (7.3)
	Negative	27.5 (5)	27.1 (5.1)	27.7 (6.4)	37.9 (7.3)
Corticosteroid administration	Yes	27.1 (4.4)	27.3 (5.3)	26.2 (6)	38.1 (7.4)
	No	24.5 (4.9)	26.7 (5.3)	27.8 (7.3)	37.7 (7)

DAS28, disease activity score-28; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein.
 For all differences between groups: p>0.05.
 *Normal ESR: <50 years: male <15, female <20.
 >50 years: male <20, female <30.

Tokyo, Japan). This apparatus measures total body mass and assesses body composition in terms of percentage body fat, fat mass, fat free mass and total body water, as well as fat distribution in different body segments (abdominal and peripheral fat) and has a standard error of <3.²⁶ After initial manual entry of their demographic details, participants stood bare footed on the analyser and held the handgrips provided until the apparatus printed the results. BMI (kg/m^2) was calculated on the basis of measured height and weight. In RA patients, contemporary serological inflammation and clinical disease activity were assessed by the erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) (using routine laboratory procedures) and the disease activity score-28 (DAS28).²⁷ Disease duration was recorded from review of the patients' hospital notes.

Data management and analysis

Data were inserted in a purpose designed spreadsheet (Microsoft Excel 2003) and audited for accuracy weekly. They were exported for analysis to the Statistical Package for Social Sciences version 11.0 (SPSS Inc, Chicago, IL, USA). Preliminary evaluation of the variables using a Kolmogorov-Smirnov test of normality revealed that none of them required logarithmic transformation to reach normality. Means (SD) were calculated for all variables.

The method of analysis was to define either BMI or BF as the dependent variable and then to incorporate all other known parameters thought to influence these measures of adiposity as either factors in an ANOVA or factors with covariates in an ANCOVA. Factors included sex and disease status (RA, OA and HC) while age, disease activity and duration, and serological inflammation were entered as continuous covariates. The initial ANCOVA analysis incorporated all these factors and covariates, but only those found to be significant were subsequently retained and reported in the prediction equation model below.

Within the RA population of the observation group, correlations of disease activity (DAS28, ESR, CRP) and disease duration with BMI and BF were obtained for each sex. RA patients were also subgrouped according to their clinical disease activity (DAS remission <2.6, mild 2.7–3.2, moderate 3.3–5.1, high >5.1²⁷), serological inflammation (ESR²⁸ and CRP²⁹), disease duration (early <3 years, established 3–10 years, longstanding >10 years), rheumatoid factor positivity

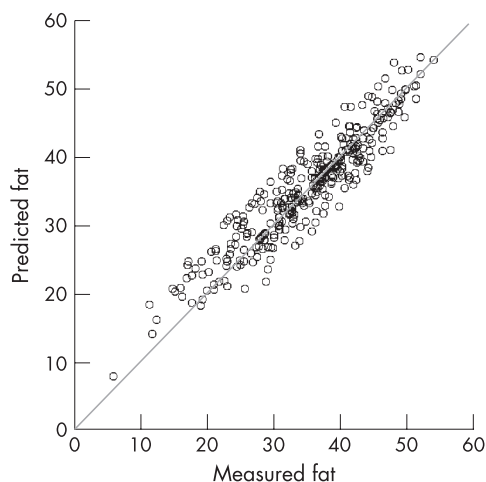


Figure 1 Agreement between predicted and measured fat in patients with RA. Body fat was measured by bioelectrical impedance using a Tanita BC-418 MA Segmental Body Composition Analyzer. Predicted fat was assessed using the formula: $\text{BF} = 4.273 + \text{sex} - 0.719 + 0.108 \times \text{age} + 1.059 \times \text{BMI}$. 95% limits of agreement were 6.17 with a coefficient of variation of 8.9.

(ever), or corticosteroid administration (yes/no ever): differences between these subgroups in relation to BMI and BF were assessed using ANCOVA (table 2). The level of significance was set at $p < 0.05$.

The external validity of the predictive model was tested with the limits of agreement (LIM_{AG}) method³⁰ against BF of the validation group. The limits of agreement were obtained as follows:

- (1) We calculated the mean (d) and the standard deviation (s) of the differences that indicate the level of bias and the random variation between the two measures of BF (that is, the predicted BF and measured BF of the validation group, respectively).
- (2) Provided the differences are normally distributed, the 95% limits of agreement are given by: $d \pm (1.96 \times s)$.

Bland and Altman³⁰ argue that, provided that differences within these limits are not clinically important, the two measurement methods can be used interchangeably.

RESULTS

Observation group

Within the RA population of the observation group, no significant correlations were found between DAS28, ESR, CRP, disease duration and BMI or BF. Similarly, when RA patients were grouped according to these variables as well as rheumatoid factor positivity and corticosteroid use, no significant differences for BMI and BF were observed ($p > 0.05$ in all cases, see table 2).

Between the different disease groups, one way ANOVA revealed significant differences in BMI ($p < 0.05$) and BF ($p < 0.001$; table 1): RA males had higher BMI and BF (including trunkal fat) than HC males, and RA females had higher BF than HC females, even though their BMI did not differ significantly. ANCOVA revealed that BMI differences between the groups were mainly the result of the significant effect of the covariate age ($F_{1,294} = 5.10$, $p < 0.05$) and not because of disease ($F_{2,294} = 1.00$, $p > 0.05$), sex ($F_{1,294} = 0.59$, $p > 0.05$) or their interactions.

ANCOVA also revealed that RA and OA patients exhibited lower BMI levels than their HC for a given BF. However, differences were only significant for the RA patients (RA: $-1.826 \text{ kg}/\text{m}^2$ ($p < 0.001$); OA: $-0.352 \text{ kg}/\text{m}^2$ ($p > 0.05$)). BMI was significantly ($p < 0.001$) predicted by age, disease, sex and BF ($R^2 = 0.58$).

When BF was adopted as the dependent variable, ANCOVA identified significant differences between disease groups ($F_{2,293} = 18.70$, $p < 0.001$) and sex ($F_{1,293} = 380.90$, $p < 0.001$) together with a significant covariate, age ($F_{1,293} = 22.43$, $p < 0.001$). The contribution of BMI as a covariate in this analysis was also significant ($F_{1,293} = 370.74$, $p < 0.001$). For a given BMI, RA patients exhibited significantly increased levels of BF (4.273, $p < 0.001$) compared to healthy controls. The difference for OA patients was non-significant (1.648, $p > 0.05$). The variation of BF was predicted by age, gender, BMI, and disease type ($R^2 = 0.769$, $p < 0.001$). This was only very slightly improved (for RA) by the addition of RA disease duration ($F_{1,293} = 0.340$, $p > 0.05$) in the equation (from 76.9% to 77.1%), so we did not include this variable in the final model. The predictive model obtained from this analysis is:

- $\text{BF} = \text{disease status} + \text{sex} - 0.719 + 0.108 \times \text{age} + 1.059 \times \text{BMI}$
- Disease status: RA = 4.273, OA = 1.648, HC = 0.
- Sex: male = -11.294 , female = 0.

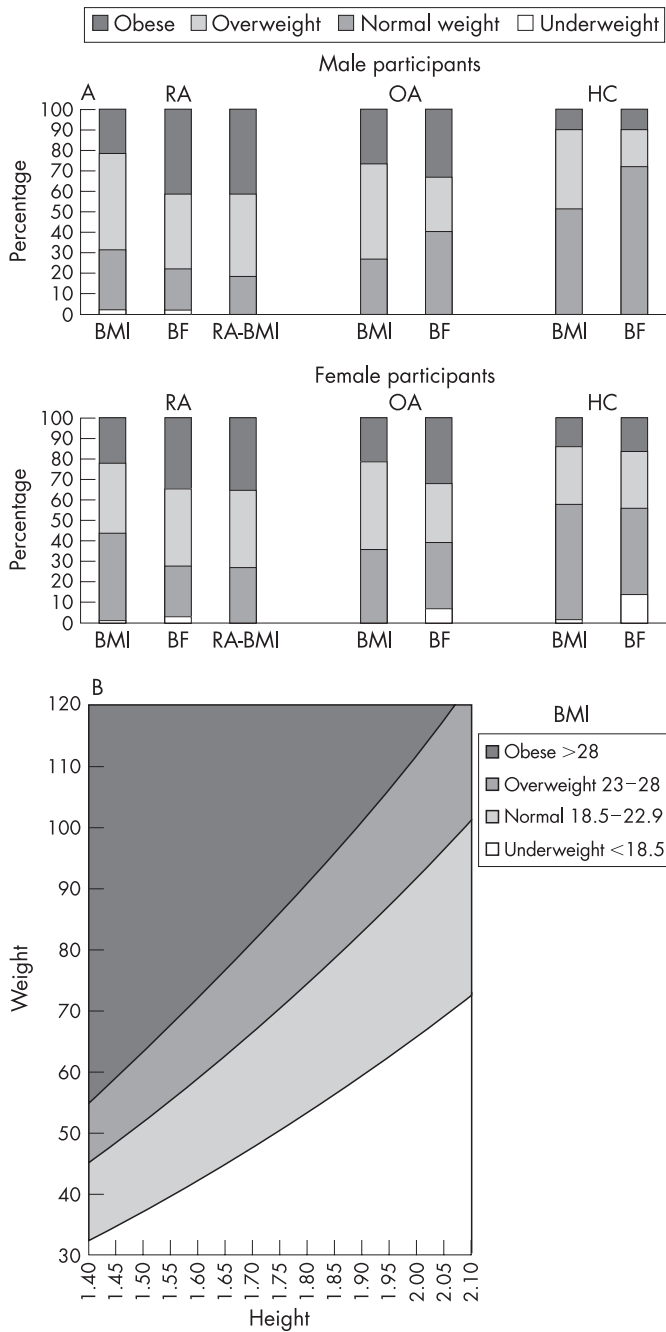


Figure 2 (A) Classification of male (top) and female (bottom) participants into obese, overweight, normal and underweight groups according to currently accepted BMI cut-off points (BMI), body fat content (BF) and RA specific BMI cut-off points (RA-BMI). Accepting BF as the most accurate assessment of body fatness, currently accepted BMI cut-off points misclassify a significant proportion of both males and females with RA (notice the difference in the respective bars). This misclassification is corrected when the proposed RA specific BMI cut-off points are applied. RA, patients with rheumatoid arthritis; OA, patients with osteoarthritis; HC, healthy controls; BMI, classification according to existing body mass index (BMI) cut-off points of 25 kg/m² for overweight and 30 kg/m² for obesity; BF, classification according to age and sex specific cut-off points for body fat percentage; RA-BMI, classification according to the proposed RA specific BMI cut-off points of 23 kg/m² for overweight and 28 kg/m² for obesity. (B) BMI chart developed specifically for patients with RA. Values were calculated using the formula: BMI = weight (in kg)/height² (in metres) for the rheumatoid arthritis specific BMI levels identified in the present study (23 kg/m² for overweight, 28 kg/m² for obesity). The generally accepted lower threshold for normal BMI (18.5 kg/m²) was not altered.

Validation group

To establish external validity of our predictive model, we assessed its agreement with the measured BF in 342 patients with RA. Preliminary analyses for LIM_{AG} revealed no heteroscedasticity, thus the LIM_{AG} can be reported as absolute measurements.³⁰ Our analyses suggested that the bias of our prediction is 0.4 (that is, our model overpredicts BF by 0.4) with a standard error of 3.2 (95% LIM_{AG} = 6.17, coefficient of variation = 8.9; fig 1). The difference is statistically significant (*t* = 2.3, *p* < 0.05), but the coefficient variation (CV = 8.9) is within acceptable limits.

RA specific BMI cut-off levels

The fact that patients with RA exhibited increased BF values for a given BMI compared to HC suggested that BMI cut-off points in the RA population would be more appropriate if they were reduced by approximately 2 kg/m² (to 23 kg/m² and 28 kg/m² for overweight and obesity, respectively). We therefore compared the proportions of subjects in each group that would be correctly classified as overweight or obese using the widely accepted BMI cut-offs of 25 kg/m² and 30 kg/m² vs the proposed (for RA) 23 kg/m² and 28 kg/m² vs the age and sex specific cut-off points of measured BF. This analysis showed that 9% of male and 15% of female RA patients would be misclassified as of normal weight based on traditional BMI cut-offs. Such misclassification was not a problem either for OA or HC, where if anything, BMI overestimated BF. Application of the proposed RA specific BMI cut-offs of 23 kg/m² and 28 kg/m² corrected this misclassification (fig 2A). A modified, RA specific BMI chart for the classification of patients with RA into underweight, normal, overweight and obese categories was developed and is provided in figure 2B.

DISCUSSION

The validity of BMI as an acceptable measure of overweight or obesity, and as an accurate reflection of body fat (BF) content, has been repeatedly questioned and the need for population specific BMI cut-off points has been highlighted.^{7 10-13} Ideally, individualised assessment of BF should be pursued in the clinical setting, as BF percentage is a more reliable measure of fatness than BMI, at least in the general population.³¹ Indeed, our data indicate that only 58% of the variance in BMI can be predicted, as opposed to 77% in BF. BF in vivo can be determined via a number of methods such as underwater weighing, dual energy x ray absorptiometry, total body water, total body nitrogen, ⁴⁰K whole body counting and urinary creatinine excretion.³²⁻³⁴ BF can also be estimated from the thickness of partial subcutaneous fat, near infrared rays and ultrasound.³⁵ However, none of these methods can be practically used in the routine clinical setting as they require sophisticated apparatus and specialised personnel.³³

In recent years, a bioelectrical impedance method for the estimation of BF in different populations has become popular and widely recommended, as it is reliable, objective, practical, relatively inexpensive and does not require highly trained personnel.^{32 33} The validity of this method has been confirmed in various studies.^{32 36-39} Devices with eight tactile electrodes using single frequency electrical current, similar to the one used in this study, generate highly reproducible measurements of total BF and segmental fat distribution.⁴⁰ Their correlation with the “gold standards” of dual energy x ray absorptiometry and hydrostatic weighing is 0.90 and 0.80, respectively, with a standard error of around 3.0, producing a coefficient of variation of <10%.³³ This suggests that bioelectrical impedance measurements (especially when using eight electrodes) are valid and suitable for body composition studies.^{32 39 40} Patients

are usually happy to undergo such a measurement because of its simplicity and similarity to normal weighing.

In the absence of the necessary equipment or expertise, the predictive model presented here can be used to easily calculate BF of RA patients from BMI. The cross validation of this predictive model in patients with RA is reassuring. Even though there was a statistically significant difference between the measured and the predicted BF, closer examination of the means indicates that this difference is at a level of less than 0.5% of BF with a coefficient of variation of <10%. The statistical significance of such a small difference can be attributed to the very large number of the validation group and is clinically not significant. However, the parts of the equation referring to OA patients and healthy individuals need further prospective validation in sufficiently large samples of the relevant populations.

BMI remains the most commonly used indicator of body fatness in the clinical setting, and the cut-off points of 25 kg/m² and 30 kg/m² (for overweight and obesity, respectively) used for the general population are also routinely applied in RA patients. This study shows that application of these BMI cut-off points misclassified 9% of male and 15% of female RA patients in terms of actual body fatness. For a given BMI, RA patients exhibited an average 4.3% increase in BF compared to healthy controls. In contrast, for the same level of BF, RA patients had BMI values almost 2 kg/m² lower than those of healthy controls. We propose that BMI cut-off points in the RA population should be lowered to 23 kg/m² (from 25 kg/m²) for overweight, and 28 kg/m² (from 30 kg/m²) for obesity. The lowest limit for normal BMI (that is, 18.5 kg/m²) should remain unaltered, as low BMI levels have been related to increased cardiovascular risk in patients with RA.^{41–42} We also provide a chart for the classification of RA patients in normal, overweight and obese categories according to these BMI cut-offs, for use in the routine clinical setting (fig 2B).

The most likely explanation for the BMI and BF differences observed in RA is rheumatoid cachexia associated with the chronic inflammatory response, given that such differences were not as prominent in OA. RA patients experience accelerated involuntary loss of fat-free mass, predominantly in the skeletal muscle, in excess of what is normally expected as a result of the ageing process.⁴³ Although the underlying mechanisms for rheumatoid cachexia remain unknown, possible contributing factors include the overproduction of inflammatory cytokines such as tumour necrosis factor α and interleukin 1 β .^{43–44} Our subanalyses within the RA population revealed that neither BMI nor BF were associated with current clinical or serological disease activity, seropositivity for rheumatoid factor (which tends to associate with more severe disease) or corticosteroid administration. This is not totally surprising as disease activity may vary within small periods of time, depending on medication and the disease itself, whereas changes in body composition are longer term processes. On the other hand, disease duration appeared to be of some importance. It is possible that most alterations in body composition of RA patients occur in the first few years of the disease, as it has previously been reported,²¹ irrespective of disease characteristics or medical treatment.

The results of the present study are reminiscent of the observations made for Asian populations, which have significantly higher CVD risk than white people: BF in Asians has been found to be 3–5% higher than that of white people with similar BMI, whereas BMI was 3–4 kg/m² lower than that of white people with similar BF.³² Differences in body build (trunk to leg length ratio and slenderness) and in muscularity have been suggested as possible explanations for these discrepancies. As a result, new cut-off points for Asian populations have been

set at 23 kg/m² and 27 kg/m² for overweight and obesity, respectively,¹⁰ and have been shown to be more sensitive in identifying Asians at increased risk for CVD.⁴⁵

In our participants, lowered BMI cut-off points would reflect an average reduction of 5–6 kg, or 8%, in the ideal weight (the weight one should have in order to be below the BMI cut-off for overweight). Such reductions in body weight are likely to lead to physiological benefits in the cardiovascular system: in the general population, even a 5% reduction of body weight is known to favourably affect most classic CVD risk factors.^{46–47}

The reduced BMI cut-off points for RA suggested here may be of significance both for the management of individual patients and for further research into the cardiovascular morbidity and mortality of RA. In the clinical arena, the reduction of these thresholds would identify an additional 10–15% of people with RA as overweight or obese, and may trigger closer scrutiny for other CVD risk factors and appropriate intervention, if necessary. Moreover, obesity, defined by the BMI, is one of the WHO criteria for the metabolic syndrome.⁴⁶ Aggressive identification and reduction of classic CVD risk factors in patients with RA is an obvious strategy for reducing the increased cardiovascular mortality of this disease.¹⁹ From the research perspective, the new thresholds may trigger re-analysis of previously published cohorts or further analysis of prospective cohorts as to the importance of body fat as a predictor of CVD in RA and its association with other individual risk factors.

We conclude that, in the clinical setting, body fatness of RA patients should be evaluated based on the BMI cut-off points of 23 kg/m² for overweight and 28 kg/m² for obesity. In the absence of specialised equipment, if necessary, BF of patients with RA can be estimated from BMI using the equation provided.

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Competing interests: none.

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