Obesity and low back pain
A review of the literature

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As part of the continuing development of the Greater Glasgow Back Pain Service (GGBPS) a literature review was undertaken to investigate an area of low back pain (LBP) and its implications for clinical practice. The focus of this review is the relationship between obesity and LBP.

**Introduction**

**Obesity**

Before consideration of obesity – we must first define it. Obesity can be defined as the storage of excess calories as fat. This definition separates weight, which is a measure of total mass, from obesity, which is a measure of fat mass. Body Mass Index (BMI) is the most widely used measure and involves a simple weight for height calculation which classifies adult weight as follows:

<table>
<thead>
<tr>
<th>Classification</th>
<th>BMI (kg/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight</td>
<td>&lt; 20</td>
</tr>
<tr>
<td>Healthy weight</td>
<td>20–24.9</td>
</tr>
<tr>
<td>Overweight</td>
<td>25–29.9</td>
</tr>
<tr>
<td>Obese</td>
<td>30–39.9</td>
</tr>
<tr>
<td>Morbidly Obese</td>
<td>40 or more</td>
</tr>
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</table>

A critical flaw in this measure however, is that it fails to differentiate lean muscle mass from fat mass (NICE guideline Dec 2006).

Obesity is a problem of epidemic proportion (Mirtz et al 2005). In Scotland more than 20% of the population are obese and, if this trend continues, then by 2020 this figure will be 33% (Rennie & Jebb 2005). The issues of overweight and obesity are important public health problems because they are associated with increased risks for hypertension, coronary heart disease,
Type 2 diabetes, stroke, gall bladder disease, certain cancers, osteoarthritis, and sleep apnoea (Rennie & Jebb 2005). Indirect costs of these co-morbidities are difficult to quantify but conservative estimates are in the order of hundreds of millions of pounds per year. These associations are clear; the link with Low Back Pain (LBP) is less so.

**Low Back Pain**

Low Back Pain (LBP) is a considerable public health problem and a common diagnosis resulting in absenteeism and the need for disabilities pensions. For these reasons, it has been hailed as a “20th century medical disaster” (Waddell, 2005). It is frequently cited as a significant economic burden and the cause of much personal hardship. Commonly quoted figures indicate direct healthcare costs to the NHS and community care services at more than £1 billion each year including: £141m each year for GP consultations, £150.6m for NHS physiotherapy and £512m for hospital care (Mandiakis and Gray, 2000). When other costs, such as the estimated 4.9 million working days lost to LBP annually in the United Kingdom (UK), are taken into consideration, the total cost of LBP to the UK is closer to £5 billion per annum (Health and Safety Executive, 2005). A more recent study (Wynne-Jone et al 2008) suggests that the economic impact of LBP may in fact be higher than previously estimated and gives an estimate of £9090 million lost in the UK in 1998 alone, through loss of work time. These authors suggest that previous economic analysis has only considered loss of work time through absence, with no consideration of the impact of those who, although at work, are performing restricted duties and consequently less productive.

Low Back Pain is a common yet elusive disorder. Possibly because of its heterogeneous, multidimensional nature, little is understood about its aetiology (Kohlmann et al 1997, Waddell et al 1992). LBP is accepted as having a multifactorial aetiology. The main predictors of LBP include physical stress e.g. prolonged lifting, driving; forceful or repetitive movements
involving the back, psychosocial stress e.g. high perceived workload and time
pressure, low control and lack of social support at work and personal
characteristics such as psychological status and tobacco use (Power et al
2001). The physical characteristics of body weight and obesity are less clear
(Leboeuf-Yde 2000).

**Obesity & Low Back Pain**

Obesity is one of several lifestyle factors that has been suspected of not
merely relating to, but in fact causing LBP. There are several hypotheses
relating to a link between obesity and LBP. It has been postulated that
excessive body weight could have mechanical ill effects on the back caused
by excessive weight bearing (Aro & Leino 1985, Bostman 1993, Deyo & Bass

These increased mechanical demands from obesity have been suspected of
causing LBP through excessive wear and tear (Kostova et al 2001, Felson
1996). It has also been suggested that metabolic disorders may be
detrimental; Buckwalter et al 1993 postulated that obesity, combined with its
co-morbidities of diabetes and hypertension may alter the pathophysiology of
diseases of the tendons and ligaments during the process of aging thus
potentially leading to LBP.

The conventional wisdom behind this assumed link is that - as overweight
persons are at risk of osteoarthritis in weight bearing joints such as the
knees, hips and feet - this trend has been alleged to generalise and
extrapolate to the spinal joints also (Felson D.T. 1996). This conventional
wisdom has led to weight loss being recommended as a treatment for chronic
LBP yet little evidence exists to support this recommendation. The justification
for weight loss as a general health improvement tool is clear. However it begs
the question “does obesity in LBP blind practitioners to other possible causes
of the symptoms?” (Garzillo & Garzillo 1994).

So we see that it is widely assumed that being overweight is related to having
LBP. Scientific evidence to support this however, is scanty and conflicting
A positive association between high body weight and LBP has been noted in some studies (Deyo & Bass 1989, Liira et al 1996) but not others (Ryden et al 1989, Manninen et al 1995). With such societal costs, it is clear therefore - that from a public health perspective - it would be important to know whether lifestyle factors such as body weight and obesity play an important part in LBP genesis. The literature reveals some confusion.

Even a superficial review of the literature makes it clear that some consider obesity a possible but not particularly strong contributory factor of LBP (Mortimer et al 2001) whereas others do not think it is a risk factor of LBP at all (Bener et al 2003). For this reason and against this background of poor clarity we decided to review the literature more thoroughly in search of any evidence based recommendations or conclusions that could be made on this elusive relationship.

**Method**

The search targeted articles in English from the databases Medline, Cinahl and Amed from 1994 - 2006. The year 1994 was chosen as a start point as this was when the CSAG report was published. Key words used in the search were Low Back Pain, Back Pain, Obesity, Body Mass Index, and Overweight. The main selection criterion was that the paper appeared to focus on the relationship between obesity and low back pain, regardless of methodology. This search yielded 68 papers. The titles and abstracts of these 68 papers were studied by the authors of the current review and by consensus 16 were selected as being relevant for further study. Inclusion and selection into this final 16 was contingent upon the relationship between obesity and LBP proving to be the focus of the research and not merely an incidental consideration. The SIGN search tools for cohort and case control studies and literature reviews were used in reviewing the selected papers. A number of factors made it difficult to synthesise the literature, including ambiguity relating to definitions of obesity and low back pain. A summary is shown in Table 1.
<table>
<thead>
<tr>
<th>Authors Year</th>
<th>Study design</th>
<th>Subjects</th>
<th>Results/ Conclusion</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>LeBoeuf-Yde et al. 1999</td>
<td>Cross Sectional Postal survey</td>
<td>29,424 Danish twin subjects, aged 12-41</td>
<td>Obesity is modestly positively associated with LBP, in particular with chronic or recurrent LBP</td>
<td>- 3 Subgroups re duration of LBP &lt; 7 days, 8-30 days, &gt; 30 days. “Chronic LBP” more usually Pain. 12 or 24 weeks before described as chronic defined as &gt; 30 days even though episodes of NSLBP may last 6-8 weeks - In view of impact of recurrent &amp; Chronic LBP even modest association may be important</td>
</tr>
<tr>
<td>LeBoeuf-Yde 2000</td>
<td>Systematic review of epidemiological literature</td>
<td>56 Reports on 65 Studies (1965-1997) of general population with &gt; 3000 subjects</td>
<td>32% of the studies show a statistically significant positive but weak association between body weight and LBP. “Due to lack of data” Body weight a possible weak indicator but insufficient data to assess if it is a true cause of LBP “due to lack of evidence”</td>
<td>- Search method “most articles were obtained from reference lists of research reports and reviews” - Review process</td>
</tr>
<tr>
<td>Mirtz &amp; Green 2005</td>
<td>Literature Review, articles 1990-2004</td>
<td>(Self reported LBP)</td>
<td>There is a positive association but no evidence of causality between LBP &amp; obesity</td>
<td></td>
</tr>
<tr>
<td>Smith et al 2006</td>
<td>Cross sectional analysis of Self report (from a larger longitudinal survey)</td>
<td>38,050 women participating in Australian Longitudinal Study of Women’s Health</td>
<td>Obesity not strongly related to LBP. BMI not strongly associated with the incidence of LBP. Mid aged and older obese women had higher odds of experiencing LBP</td>
<td>- Some Ambiguity eg ? justification for some of the conclusions drawn of moderate to high risk of LBP associated with BMI &gt; 40</td>
</tr>
<tr>
<td>Faneule et al., 2002</td>
<td>Survey</td>
<td>15,974 patients with spinal disease</td>
<td>Obese patients with LBP more likely than non obese patients to have radicular pain and neurological signs General &amp; disease specific functional status significantly worse for obese patients</td>
<td>- Considers subclasses of LBP and impact of LBP ie disability - rather than only consider incidence of LBP -Subjects attending “spine centres” speciality clinics- ? representative of general LBP population</td>
</tr>
<tr>
<td>Study</td>
<td>Design</td>
<td>Sample Size</td>
<td>Description</td>
<td>Key Finding</td>
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</table>
| Han et al, 1997 | Cross sectional survey       | 5887 men, 7018 women aged 20-60 from a larger Dutch study | Women who are overweight & have a predominantly abdominal fat mass have a greatly increased likelihood of LBP | -Author does not offer any theories as to why this may be the case  
- Pain radiating to the knees or feet “taken to indicate disc herniation” Questionable |
| Webb et al, 2003 | Multiphase cross sectional survey of musculoskeletal pain | Sample of 5752 patients from 3 general practice registers in the UK | BMI is an important independent predictor of LBP and its severity | UK population |
| Mortimer et al, 2001 | Population based case referent study? | 2401 subjects, 791 with LBP, 1610 without LBP, taken from a larger study in Sweden | High body weight is associated with an increased risk of LBP in men but not women | -Self reported weight and height  
- Women may under report weight  No other theory offered to explain the finding |
| Bener et al., 2003 | Cross sectional survey       | 802 people who attended a primary health care clinic in the United Arab Emirates | Obesity is moderately associated with LBP | Many details unclear e.g. 802 out of 1,103 subjects selected for statistical analysis, criteria for selection unclear |
| Baker & Giles, 1999 | Prospective correlational study | 71 women, 81 men with chronic LBP in a spinal pain unit | No support for the concept that patients with chronic LBP are more overweight that the general population with the possible exception of morbidly obese patients | Small number of subjects |
| Tsuritami et al, 2002 | Survey                      | 709 Japanese women aged >40 | No significant association between BMI and frequency of LBP | |
| Toda et al, 2000 | Survey                      | 330 Japanese men and women aged 45-69 with LBP >3 months or recurrent LBP | In women, central obesity may be a higher risk factor for chronic LBP without positive straight leg raise. | |
| Baker & Giles, 1999 | Prospective correlational study | 71 women, 81 men | No support for the concept that people with chronic pain are more overweight that general population, with possible exception of morbidly obese | Small number of subjects |
| Slojje, 2004 | Cross sectional study        | 88 urban Norwegian adolescents | LBP associated with a higher than normal BMI | |
| Jones et al, 2005 | A matched case control study | Adolescents 28 with LBP, 28 without LBP | BMI was not identified as a significant risk indicator for recurrent non specific LBP | Small numbers |
OBESITY & LOW BACK PAIN

Discussion (Local Context)

Prior to discussion of any relationship between obesity and LBP, it is useful to consider in more detail the local context in which they lie.

Obesity

In Scotland, as in the rest of the UK, rates of obesity are increasing. In 1998, the Scottish Health Survey reported that 62% of men and 54% of women were either overweight or obese. Within the three years between 1995 and 1998 there has been an increase in the prevalence of obesity of 3% in both men and women. Worryingly, the prevalence of obesity in Scotland is greater than England and is a recognised factor in the health inequalities between Scotland and England (Rennie & Jebb, 2005). There is also evidence that the children of Scotland are more obese than elsewhere in the UK, with the prevalence increasing from 8.7% in 5–6 year olds to 15.8% in 14–15 year olds (Reilly & Armstrong, 2003).

National survey data on the prevalence of obesity in Great Britain have been recently reviewed by Rennie & Jebb, 2005. Some of their findings are outlined below.

In 2002, 23% of men and 25% of women were classed as obese.

The prevalence of obesity has trebled since 1980, when 6% of men and 8% of women were classed as obese.

In Scotland over a fifth of men and women are obese.

In Scotland over half of men (62%) and women (54%) are either overweight or obese. If this trend continues then by 2020 1 in 3 Scottish adults will be obese.
Low Back Pain Prevalence

- 1 Year Prevalence = 38 % of population will have low back pain
- 32% Intermittent Symptoms
- 6% Chronic Symptoms
- Of 62% Painfree 1/3 will develop new symptoms in any 1 year
  (Waddell 2005)

Given the relatively high prevalence of these 2 conditions it is not surprising that Health Care Professionals who assess and treat spinal pain commonly encounter patients who are in addition clinically obese. *It is important to identify, if possible with current evidence, whether there is a causal relationship or indeed any association between low back pain and obesity as this would have implications for prevention and best management of this costly condition.* If one exists, then what is the strength of any such relationship or association?

Discussion

Causal Relationship

A causal relationship exists between two variables in which changes in the value of one cause the value of the other to alter. The papers reviewed herein found conflicting results.

Pathogenesis of Low Back Pain

It was speculated by Leboeuf-Yde (2000) that the increased mechanical loading caused by obesity might be responsible for low back pain through “excessive wear and tear”.

Baker & Giles (1999) measured the Body Mass Index (BMI) of 152 persons with spinal pain. It was found that those with low back pain are not more overweight than the general population since their average BMI (26.3) was identical. Consequently, it was concluded that with the exception of the
morbidly obese, excess weight is *not* a significant factor in the pathogenesis of low back pain.

*Conversely, obesity in isolation could demonstrate no influence but rather that certain types of obesity may present in people with a generally poor lifestyle and that LBP occurs as a result of “the combination of several slovenly habits” in such a population* (Leboeuf-Yde et al 1999).

**Predictors of Low Back Pain**

A cross sectional study via a screening questionnaire of 5752 adults identified 4515 subjects with spinal pain (Webb et al 2003). These were followed up via the Oswestry Disability Questionnaire. *The authors found obesity to be a predictor of back pain and disability.* The causal association was thought to be equivocal. *It is of note however, that the underweight BMI (<20) group demonstrated a higher prevalence of all the categories of back pain than subjects with a normal BMI. In this study the highest prevalence of LBP was found in obese (>30) persons.* The small number of participants involved limited this study.

Han et al (1997) reported that using BMI as a measure fails to differentiate lean muscle mass from fat. The authors cross sectional study of 12,905 Dutch adults reported that a high waist/hip ratio, indicating a central obesity pattern, was significantly associated with chronic low back pain in women but not in men. *The authors do not postulate as to why this may be the case.* The distribution of lean body mass and body fat was shown to be more closely associated with risk of chronic low back pain than Body Mass Index.
**Relationship Between Obesity and Low Back Pain**

In studying the relationship between obesity and LBP, the possible modifying effects of age, gender, type of work and smoking were controlled for by Leboeuf-Yde et al (1999). A cross sectional postal survey of 29,494 Danish twin subjects was employed. This identified a moderate, positive association between BMI and low back pain that increased with the duration of low back pain, particularly when chronic; this association also increased in direct proportion with recurrence of low back pain. The association was weak due to the lack of a positive monotonic dose response. It was felt that the association was not causal since the effect disappeared in the control group of 3,751 monozygotic twins with differing body size.

Fanuele et al (2002) conducted a cross sectional study of 15,974 patients with spinal pain. In addition to assessing BMI, functional status was measured via the SF-36 Physical Component Summary score, with the Oswestry Disability Index serving as a disease-specific measure. Physical disability correlated with obesity (p<0.05). Compared with non-obese patients, the higher the BMI the greater the functional disability. Morbidly obese patients also had more radicular pain (p<0.001) (33.6% non obese vs. 47.2% morbidly obese). There were also greater neurological signs (p<0.01) (22.4% non obese vs. 32.7% morbidly obese). Obese patients tended to have more chronic pain (>3 months) and were statistically more likely to present with co-morbidities such as cardiovascular/pulmonary disease, cancer, degenerative and inflammatory arthritis, diabetes, stroke and endocrine disease. Although LBP may not be directly correlated with obesity, it was found that each might be related to anxiety, depression and psychological distress. Fanuele established a high prevalence of depression amongst obese patients. The authors concluded, *"without treatment of the obesity, increased BMI will likely impair the patient’s functioning even after a successful spine treatment."*
A review of the literature by Leboeuf-Yde (2000) identified a weak association between obesity and low back pain. But there was not enough evidence to establish a causal relationship.

In 2005 Mirtz & Greene conducted a further literature review, which found no clear evidence of a dose–response relationship between BMI and low back pain. The quality of the literature was varied with few samples of large enough size to be externally valid. However it was thought that individuals with a BMI <30 are at minimal risk of developing low back pain. A moderate risk is found at BMI > 30, whilst >40 Body Mass Index indicates a high risk for future spinal pain.

So we see a general lack of clarity and conflicting results which have led to the suggestion that obesity may be a marker (Heliövaara et al 1987) or confounder (Aro & Leino 1985, Deyo & Basss 1989) for some other factor that is a cause of LBP (Leboeuf 2000). It has been postulated therefore that obesity - in itself - has no influence on LBP, but that certain types of obesity may present in people with a generally poor lifestyle, and that it is “the combination of several slovenly habits that cause LBP” (Leboeuf 1999).

According to the biopsychosocial model there is a relationship between biology, pathology, the individual and the environment which results in the final expression of a disease or disorder in terms of pain or disability. This interplay may account for the lack of definitive evidence for or against a causal link between LBP and obesity (Leboeuf-Yde 2003). Also, inconsistency in the definition of what constitutes LBP and obesity has clouded the issue further.
Conclusion

Although some of the studies identified a weak association between obesity and low back pain, there was insufficient evidence to establish a direct causal relationship. This may in part be due to the quality of the studies, sampling, variation in data collection and measurement. The following quotes are reflective of the research reviewed herein.

“Not enough evidence to determine whether there is or not….“(a direct link between two) Le Boeuf-Yde et al 1999

“Patients with chronic spinal pain are no different to the general population in terms of BMI” Baker et al 1999

“Obesity in itself has no influence on LBP” Le Boeuf 2000

“May not be directly related to one another but each may result from depression, anxiety and/or distress.” Faneule et al 2002

It is clear that both obesity and low back pain are major determinants of ill health and disability. A link could reasonably be expected between these two factors since these conditions share many covariates; including low occupational status, sedentary lifestyle and psychological distress. Leboeuf et al have demonstrated a mild positive relationship between weight and recurrent or chronic low back pain; although not a direct causal link. This hints that perhaps obesity contributes to some important factor concealed within the heterogeneous nature of low back pain. Perhaps in the future, research will see low back pain differentiated into etiologically distinct groups to allow us to tease out this relationship. As it stands, further research is required to confirm or refute any significant relationship between these two common health problems.
**Clinical Implications: Advice for Patients and Health Care Providers**

Based on this conclusion - what advice can be formulated that would be useful for patients with excessive weight gain and low back pain?

The Cochrane Collaboration Review “Exercise for Overweight or Obesity” postulates that weight gain is due to a reduction in exercise and increased consumption of energy-dense, high carbohydrate/fat, low nutrient foodstuffs. Being overweight increases the risk of serious chronic diseases including Type II diabetes, cardiovascular disease, hypertension, stroke and some cancers (Shaw, K et al, 2007).

The current evidence would not support advising patients that reducing weight will produce a direct reduction in the intensity of their low back pain, (Baker and Giles, 1999). This does not eliminate the benefit of weight loss on the co-morbidities listed above. The authors suggest that clinicians who imply that weight loss may reduce back pain may cause distress to patients and reduce their adherence to any exercise regimen.

Obese individuals often have restricted exercise capacity and so attempting to lose weight by exercising alone may prove more difficult and stressful, further disadvantaging the patient.

The current advice form the Cochrane Report is that patients should try to maintain and gradually improve fitness levels to avoid further weight gain. This is best facilitated by means of a sensible eating pattern and suitable programme of low impact regular exercise. In a perfect world, this would be moderate or vigorous exercise for thirty minutes, five times per week. It is known that few patients will achieve this level of participation (Baker and Giles 1999; Shaw et al 2007).
Acknowledgements

The authors acknowledge the contribution of Colin White and Linda Shand (Both previous members of the GGBPS team) in the early stages of this review and Lorna Breslin (current GGBPS team) in the final stage of collating this review.
Reference List


NICE guideline: Obesity guidance on the prevention, identification, assessment and management of overweight and obesity in adults and children (Dec 2006).


Rennie K.L, Jebb S.A. 2005 Prevalence of obesity in Great Britain Obesity Reviews Volume 6, Issue 1, Date: February 2005, Pages: 11-12


Smith MD, Russell A, Hodges PW. Disorders of breathing and continence have a stronger association with back pain than obesity and physical activity. Australian J of Physiotherapy 2006; 52: 11-16.


Appendix / Glossary / Additional information

Disability

“Any restriction or lack (resulting from an impairment) of ability to perform an activity in the manner or within the range considered normal for a human being” *WHO (1980) International Classification of Impairments, Disabilities and Handicaps. World Health Organisation, Geneva.*


“An alteration of an individual’s capacity to meet personal, social or occupational demands because of an impairment.” *AMA (2000) Guides to the evaluation of permanent impairment, 5th Edition, American Medical Association, Chicago.*

At the core of all the definitions is that Disability is restricted activity.

Obesity

Obesity is the storage of excess calories as fat or adipose tissue. This definition separates weight which is a measure of total mass from obesity which is a measure of fat mass.

Body Mass Index (BMI)

The classification of weight that has fewest limitations, is most practical for adults, and is most widely accepted is that published by the World Health Organization, which is based on the body mass index (BMI)
The Body Mass Index (BMI) is a weight for height measure and is calculated by dividing the body weight in kilograms by the square of the height in metres.

**Underweight:** is defined as BMI less than 20 kg/m²

**Normal weight:** is defined as BMI between 20 kg/m² - 25 kg/m²

**Overweight or pre-obese:** is defined as BMI between 25 kg/m² - 29.9 kg/m².

**Obesity:** is defined as BMI at least 30 kg/m²

**Morbid Obesity:** is defined as BMI over 40 kg/m²

**BMI** has limitations when used to define overweight and obesity, and these should be taken into account when basing clinical decisions on it. It does not take into account body frame or the proportion of lean mass or the proportion of fluid. BMI therefore should not be used to assess overweight and obesity in:

- **Pregnancy**
- **Athletes with well-developed muscles**
- **People who are oedematous or who have gross ascites**

Although BMI criteria are the same for men and women, a man and a woman with a similar BMI may not have the same degree of fatness (NICE guideline Dec 2006).
The BMI also fails to take into account the distribution of body fat. This is relevant clinically because excess intra-abdominal fat accounts for most of the extra cardiovascular risk associated with obesity.

Other measures used to differentiate the ratio of lean tissue Vs fat body fat quoted in the literature include: Waist to Hip Ratio; Caliper Skin Fold; Segmental Skin Impedance (Bio-impedance) for % body fat; and Waist Circumference.

These measures have been recently reviewed in the NICE guideline: Obesity guidance on the prevention, identification, assessment and management of overweight and obesity in adults and children (Dec 2006). The recommendations made within this document for use of such measures are outlined below.

**Appropriate Measures of Overweight or Obesity**

**Adults**
Body mass index (BMI) should be used as a measure of overweight in adults, but needs to be interpreted with caution because it is not a direct measure of adiposity.

Waist circumference may be used, in addition to BMI, in people with a BMI less than 35 kg/m².

**Children**
BMI (adjusted for age and gender) is recommended as a practical estimate of overweight in children and young people, but needs to be interpreted with caution because it is not a direct measure of adiposity.
Waist circumference is not recommended as a routine measure but may be used to give additional information on the risk of developing other long-term health problems.

**Adults** and **children**
Bioimpedance is not recommended as a substitute for BMI as a measure of general adiposity.

**Classification of overweight or obesity**

**Adults**
The degree of overweight or obesity in adults should be defined as follows.

<table>
<thead>
<tr>
<th>Classification</th>
<th>BMI (kg/m²)</th>
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</thead>
<tbody>
<tr>
<td>Healthy weight</td>
<td>18.5–24.9</td>
</tr>
<tr>
<td>Overweight</td>
<td>25–29.9</td>
</tr>
<tr>
<td>Obesity I</td>
<td>30–34.9</td>
</tr>
<tr>
<td>Obesity II</td>
<td>35–39.9</td>
</tr>
<tr>
<td>Obesity III</td>
<td>40 or more</td>
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</tbody>
</table>

BMI may be a less accurate measure of adiposity in adults who are highly muscular, so BMI should be interpreted with caution in this group. Some other population groups, such as Asian and older people, have comorbid risk factors that would be of concern at different BMIs (lower for Asian adults and higher for older people). Healthcare professionals should use clinical judgement when considering risk factors in these groups, even in people not classified as overweight or obese using the classification in recommendation.
Assessment of the health risks associated with overweight and obesity in adults should be based on BMI and waist circumference as follows.

<table>
<thead>
<tr>
<th>BMI classification</th>
<th>Waist circumference</th>
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<tbody>
<tr>
<td></td>
<td>Low</td>
</tr>
<tr>
<td>Overweight</td>
<td>No increased risk</td>
</tr>
<tr>
<td>Obesity I</td>
<td>Increased risk</td>
</tr>
</tbody>
</table>

For men, waist circumference of less than 94 cm is low, 94–102 cm is high and more than 102 cm is very high.

For women, waist circumference of less than 80 cm is low, 80–88 cm is high and more than 88 cm is very high.

Adults should be given information about their classification of clinical obesity and the impact this has on risk factors for developing other long-term health problems.