

# Obesity: a preventable risk factor for large joint osteoarthritis which may act through biomechanical factors

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Osteoarthritis (OA) is a common degenerative disease of joints. The major clinical features are pain and stiffness, leading to a decline in physical function, which may ultimately require joint replacement surgery. As no cure exists, current medical intervention focuses on symptomatic relief. Moreover, as no cure is imminent, preventable risk factors for the onset and progression of the disease are of great interest. Obesity is the main preventable risk factor that has been identified. Given that obesity is modifiable by conservative treatment such as weight loss, its potential importance in reducing the incidence of OA cannot be underestimated.

Examination which examined serum cholesterol, uric acid, body fat distribution, and blood pressure among 3885 American adults aged 45–74 found no link between these metabolic factors and the association between obesity and knee OA.<sup>9</sup> However, other metabolic factors involved in the pathogenesis of knee OA may not yet be identified or studied in this setting. For instance, there is emerging evidence that serum leptin is associated with bone area, which may have implications for musculoskeletal diseases, including OA.<sup>16</sup> Nonetheless, it does not appear that the risk of large joint OA is increased by central fat distribution, as is the case for cardiovascular disease and diabetes.<sup>3,7</sup> Unlike these diseases, the risk of large joint OA is greater with increased body mass index, but not with any particular patterns of weight distribution. These findings strengthen the likelihood of a stronger biomechanical rather than any known metabolic component in the pathogenesis of knee OA.

Osteoarthritis (OA) is a common degenerative disease of joints.<sup>1</sup> The major clinical features are pain and stiffness, leading to a decline in physical function. Considerable pain and disability results from large joint OA, which is the major cause of joint replacement surgery and cost to the community. The focus of current medical intervention is on symptomatic relief, given that no cure for the disease exists. As curative intervention is unlikely in the near future, both the prevention of disease onset and progression of the disease are of great importance. Obesity is the main preventable risk factor that has been identified in large joint OA.<sup>2,3</sup> Given that obesity is modifiable by conservative treatment such as weight loss, its potential importance in reducing the incidence of large joint OA cannot be underestimated.

Longitudinal data have shown that obesity is a powerful risk factor for the development of knee OA,<sup>2</sup> with one twin study finding a 9–13% increased risk for the onset of the disease with every kilogram increase in body weight.<sup>4</sup> In addition, obesity is also a risk factor for the progression of radiological OA.<sup>5,6</sup> Despite the significant risk imposed by obesity, the mechanism by which the excess weight influences disease onset and progression is unclear. Metabolic factors<sup>7–12</sup> and increased biomechanical load across articular cartilage<sup>13–15</sup> have been postulated as possible disease inducing mechanisms.

Several studies have examined a metabolic link between obesity and OA with conflicting results. Whereas some authors have suggested that metabolic factors are associated with obesity and OA,<sup>3,11</sup> many studies have not supported a metabolic link in the pathogenesis of the disease.<sup>7–9,12</sup> The National Health and Nutrition

Although the gait characteristics of people with knee OA have been relatively well described and include decreased knee range of movement<sup>14</sup> and a larger than normal knee adduction moment,<sup>13</sup> there is a paucity of studies examining the influence of obesity on adult biomechanics. One study suggested that obesity was a risk factor for the progression of knee OA in those with varus knees, but not those with valgus alignment.<sup>17</sup> Despite the likelihood of the knee adduction moment, which concentrates load to the medial tibiofemoral compartment, representing one of the most important biomechanical variables in the pathogenesis of knee OA, no study has examined its relation to obesity directly. Nonetheless, it is intuitive to suggest that added weight would increase joint reaction forces, which may adversely affect joint cartilage. Interestingly, one study that showed a significant effect of varus angle on progression of knee OA suggested that most of the effect of knee angle on the risk of progression of knee OA could be explained by obesity.<sup>17</sup> It has also been speculated that obesity increases subchondral bony stiffness,<sup>18</sup> making bone less adept at coping with impact loads. The increased stiffness of bone may subsequently redistribute a greater force across the underlying articular cartilage, increasing its vulnerability to degenerative changes. In support of this, we have shown that, in normal, healthy subjects, adductor forces are associated with an increase in the area of tibial bone rather than the amount of knee cartilage.<sup>19</sup> This suggests that such biomechanical factors may affect the bone as an early step in the

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pathogenesis of OA. We have also recently shown that tibial bone area increases in subjects with OA and that one risk factor for this is obesity (unpublished data).

Although the association between obesity and large joint OA is probably mediated by a biomechanical component, it is unlikely to be the sole means by which obesity contributes to the pathogenesis of OA. Given that the disease is more common in women and exists in non-weight bearing joints such as the hand,<sup>20</sup> it would appear that factors other than biomechanical contribute to the pathogenesis of OA. Several studies have identified a genetic predisposition toward OA.<sup>21, 22</sup> However, despite these significant genetic findings, the mechanism expressing the familial contribution toward disease is unclear. Only one study has examined and provided preliminary evidence that the genetic component involved in the pathogenesis of OA may manifest as altered biomechanics.<sup>23</sup> This study showed that the offspring of people with medial tibiofemoral OA walked with a less than normal degree of foot rotation, which may ultimately predate disease. Nonetheless, it is also plausible that the genetic component in OA is mediated by altered metabolic factors, such as those that result in obesity.

Given that obesity is associated with the onset and progression of OA, weight loss represents an important preventive strategy. The Framingham study showed that weight control significantly affected the risk of developing knee OA.<sup>24</sup> Women who reduced their body mass index by two units or more reduced the odds for developing OA by greater than 50%. Another study similarly found that the risk of knee OA increased by 35% for every 5 kg weight gain.<sup>3</sup> Symptomatic relief from knee OA and an improved health related quality of life have also been shown in people with established knee OA who were able to reduce their body weight.<sup>24, 25</sup> Although the mechanism for these benefits are unclear, emerging data on the mechanisms of knee pain including bone oedema and chondral defects, and the possible effects of obesity on these, may help our understanding of this.<sup>26</sup>

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