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Is obesity actually non-communicable?

Cain C. T. Clark

Introduction

Obesity, broadly speaking, is characterised by having a body-mass index above 30 kg m$^2$, and described as a non-communicable risk factor. The proportion of adults in the United Kingdom (UK) that are overweight or obese has risen from 57.6% to 68% in men, and from 48.6% to 58% in women between 1993 and the present day. This represents an estimated economic burden of £27 billion, with government predictions stating that almost half of the UK population could be obese by 2050, with an associated cost of £50 billion a year. Obesity levels in the UK (and worldwide) are consistently acknowledged as, and accepted to be an epidemic. Moreover, when defining an epidemic, its’ severity and initial rate of increase depend upon the value of the Basic Reproduction Number ($R_0$), defined as the average number of new ‘infections’ generated. If $R_0 > 1$ an epidemic will occur and if $R_0 < 1$ it will die out. Therefore, given the consistent rise in weight status over recent decades, obesity could be considered to be highly communicable.

Familial and Social Transmission

Although literature is equivocal on the heritability of obesity, Whitaker, et al. demonstrated that parental obesity more than doubles risk of adult obesity in under 10’s, whilst children without obese parents represent a very low risk. In a seminal investigation, Christakis, et al. highlighted the potential of familial transmission of obesity. For instance, should one sibling become obese, they are at a 40% higher risk of becoming obese.

In addition to familial transmission of obesity, societal ties are asserted to impact on the spread of an obesogenic environment. Overweight youth have been shown twice as likely to have overweight friends, supporting the network theory of homophily, in addition to weak associations between social position and weight status. Christakis, et al. examined person-to-person infection of obesity in 12,067 people over three decades of follow up analyses, and highlighted discernible clusters of obese persons at every time-point. Furthermore, a person’s chance of becoming obese increases 57% if a friend becomes obese at any given interval. Notwithstanding, the veracity of Christakis, et al. assertions have been questioned by Cohen-Cole, et al., although the datasets used for interpretation had “several important differences…” (Cohen-Cole, et al., pp 1383). Further evidence to support the notion of the
communicability of obesity has been assimilated. Bagrowicz, et al. 7 noted that indices of body image and self-perception measured pre- and post-altering their social environment resulted in dissatisfaction with body size and self-image in less than two months. Verbatim reports suggest the influence of a highly obesogenic population was the cause. Further, Trogdon, et al. 8 indicate that friends’ weight is significantly and strongly correlated with an adolescent’s own weight, even after controlling for factors such as; demographics, smoking status, birth weight, and own parental and household characteristics including parental obesity, with females being more susceptible to transmission.

**Conclusion**

*The Good:* the communicability appears to operate in an omnidirectional fashion, with exercise and health conceivably conferring the same level of infection or spread. *The Bad:* the feasibility of controlling an epidemic critically depends on the value of the Basic Reproduction Number and the timeframe in which a person is contagious. We face an unprecedented truth that a person may be ‘contagious’ through the entire life course. *The Ugly:* network phenomena are evidently relevant to the physiological and behavioural tenets of obesity. We therefore hope to raise the ‘ugly’ question that, should a reclassification of obesity to being socially-communicable be considered and adopted by clinicians, scientists and key-stakeholders, further taking this communicability into account during treatment and intervention alike?

**References**

