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The influence of chronic conditions and the environment on pubertal development. An example from medieval England

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ABSTRACT

Adolescence is a unique period in human development encompassing sexual maturation (puberty) and the physical and psychological transition into adulthood. It is a crucial time for healthy development and any adverse environmental conditions, poor nutrition, or chronic infection can alter the timing of these physical changes; delaying menarche in girls or the age of peak height velocity in boys. This study explores the impact of chronic illness on the tempo of puberty in 607 adolescent skeletons from medieval England (AD 900–1550).

A total of 135 (22.2%) adolescents showed some delay in their pubertal development, and this lag increased with age. Of those with a chronic condition, 40.0% (n = 24/60) showed delay compared to only 20.3% (n = 111/547) of the non-pathology group. This difference was statistically significant. A binary logistic regression model demonstrated a significant association between increasing delay in pubertal stage attainment with age in the pathology group.

This is the first time that chronic conditions have been directly associated with a delay in maturation in the osteological record, using a new method to assess stages of puberty in skeletal remains.

1. Introduction

Today, adolescence (from the Latin 'adolescere', or "to grow up") is defined by the World Health Organisation (1993) as a period between the ages of 10–24 years. Adolescence is a crucial period in terms of healthy development when individuals gain 50% of their weight and 20% of their final adult stature (World Health Organisation, 1993). This adolescent growth spurt is a uniquely human phenomenon and encompasses sexual maturation (puberty), thus marking a period of physiological and psychological transition into adulthood (Hochberg and Belsky, 2013). Adolescent health is receiving increasing attention in modern populations, with their unique behaviour directly linked to morbidity. Alcohol abuse, drug-taking and sexual activity increases the risk of unwanted pregnancies, sexually transmitted diseases and deaths due to motor vehicle accidents and suicides (Grunbaum et al., 2003). Well-nourished and healthy adolescent females are seen as the key for overall population well-being, with the nutritional levels of women before their first pregnancy having a direct impact on the development and birth-weight of their child and its subsequent survival (EWECWG, 2015). Despite the importance of this period of human development, it has received scant attention in bioarchaeology.

Pubertal changes occur as the result of stimulation of the hypothalamic–pituitary–gonadal (HPG) axis and the hypothalamic–pituitary–adrenal (HPA) axis (Louis et al., 2008). The latter, although active during infancy, becomes latent during childhood and is reactivated by the release of gonadotropine into the hypothalamus during adolescence, stimulating the gonads. Sex steroid induced accelerated growth is in turn, reliant on the presence of insulin-like growth factors produced by the pituitary gland (or the GH/IGF-1 axis) (Mason et al., 2011). Within a year of peak height velocity (PHV) being reached, girls will menstruate indicating that the ovaries are capable of secreting sufficient levels of oestrogen. Sometime later, the brain emits neuro-endocrine signals in a 26-day cycle that stimulates the pituitary glands to release the hormones needed for ovulation (Zacharias and Wurtman, 1969). In boys, the presence of gonotrophins stimulate the release of androgens (testosterone and androstenedione), a stage known as 'gonadarch' (Louis et al., 2008). The HPA axis is independent of HPG and signals adrenal activation, eventually stimulating the formation of secondary sexual characteristics (Louis et al., 2008), such as breast development in females, and the apperance of facial hair and voice 'breaking' in males.

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Many factors have been shown to influence the onset of puberty, including genetic disorders, ancestry, social status, nutrition, exposure to pollutants, extreme physical exercise, psychological stress, increased or reduced body mass, and chronic illness (Louis et al., 2008). Successful progression through the pubertal stages relies on the height and weight of the individual, which in turn is influenced by nutrition and the absence of chronic disease (Proos and Gustafsson, 2012). Hochberg and Belksky, (2013) interpret this relationship in terms of evolutionary biology, demonstrating phenotypic adaptive plasticity in favourable environmental conditions that allow females to achieve the extremes of their genetic range for maturation. In less favourable conditions, females may delay sexual maturation until they have reached the required body weight, providing for later fecundity, fertility and greater longevity. In terms of health longer term, the importance of pubertal growth on peak bone mineral density is demonstrated by the observation that women who experience delayed menarche by as little as two years, are more vulnerable to bone loss before the age of 40 years and potentially, early osteoporosis (Karapanou and Papadimitriou, 2010).

Adolescence is a time of increased susceptibility to chronic infections such as tuberculosis and leprosy, due to exposure to new diseases associated with a riskier lifestyle, and the maturation of the immune system (Marais et al., 2005; Patil, 2013). The greater propensity for adolescents over younger children to develop chronic tuberculosis for instance, is explained by Marais et al. (2005). They point to the destructive containment of pathogens, characteristic of a maturing immune response, as producing an oxygen-rich environment that allows *M. tuberculosis* to flourish, producing adult-type cavitations in the lung tissue. Chronic bowel conditions such as Crohn's disease and colitis often manifest in early adolescence and are associated with a delay in puberty development, and in particular delay boys entering peak height velocity of the adolescent growth spurt (Proos and Gustafsson, 2012). This is though to be due to the combined influence of poor nutrition and inflammation on the GH/IGF-1 axis (Mason et al., 2011). Juvenile idiopathic arthritis is another condition that often develops around 11–14 years, and is associated with a delay in puberty due to chronic inflammation, with menarche often occurring two years later than in healthy adolescents (Tsatsoulis et al., 1999; Umlawska and Prusek-Dudkiewicz, 2010). Stini (1985: 213) also argued that the difference in immune system capability between males and females arises during adolescence as female bodies prepare for pregnancy.

While the presence of chronic infections is known to delay pubertal attainment in modern populations, this has yet to be assessed in archaeological samples. The development of new osteological techniques to identify stages of puberty provides the potential for us to explore this important period of human development in much greater detail than ever before (Shapland and Lewis, 2013, 2014). Recent analysis of 645 skeletons aged between 10 and 25 years from medieval England provided the first direct evidence for the age of the adolecent growth spurt and onset of puberty between the seventh to sixteenth centuries (Lewis et al., 2015). While both males and females began puberty around 10–12 years, as today, the tempo of pubertal growth was longer, with females achieving menarche between 13 and 16 years, and individuals being as old as 16–20 years before they were fully mature. In London, most females were not achieving menarche until they were 17 years of age, and many were dying between 22 and 25 years, before they have reached full maturation. These delays indicate that the age at which children achieve each maturational stage in adolescence may be useful as an indicator of environmental stress.

During that study it was noted that a number of children identified as being behind their peers in pubertal stage attainment also suffered from a chronic condition, including tuberculosis, treponemal disease, osteomyelitis, rickets, bowel infection, arm paralysis and severe congenital spinal curvature (Lewis et al., 2015). The current study explores this association in more detail to assess the impact of chronic conditions on the tempo of the adolescent growth spurt, peak height velocity and menarche in urban medieval England.

Fig. 1. Location of the cemetery sites.

2. Materials and methods

Of the original study sample of 994 adolescent individuals (10–25 years) analysed by Shapland and Lewis (2013, 2014),), 607 were preserved well enough for a pubertal stage and the presence or absence of a pathology to be observed with confidence and were included in the analysis. This medieval urban sample comprised skeletons from four cemetery sites in England dating from AD 900–1550; St Peter's Church, Barton-on-Humber (or Barton, $n = 109$), a combined York sample of St. Helen-on-the-Walls and Fishergate House ($n = 71$), with the majority derived from St. Mary Spital, London ($n = 427$) (Fig. 1). The adolescents from Barton were considered to be relatively affluent in comparison to those from York and London (Tyszka, 2006), and it is likely the London sample contained a more diverse migrant group (Connell et al., 2012). As the focus was on the physical development of individuals with chronic conditions rather than geographical location or social status, the samples were combined to represent one large urban medieval sample.

2.1. Age and sex determination

In order to investigate the influence of skeletal pathology on adolescent development in the largest sample possible, individuals were each assigned a 'minimum' age (i.e. 14+ years, 17+ years etc.) based on the lowest estimates derived from their dental age or extent of skeletal maturation. Age was assessed independently of pubertal status, with any marker used to provide a pubertal stage