

The Secular Trend to Earlier Puberty in Boy Choristers A Literature Review

*Professor Martin Ashley
Edge Hill University*

What is puberty?

Puberty is commonly described in the medical literature as the process of sexual maturation through which reproductive competence is attained (Mantzoros et al, 1997; Herman-Giddens, et al, 2001). Puberty is also associated with significant increases in height, weight, muscular strength, vital capacity, and the “breaking” of the voice. The term “breaking” is increasingly resisted by phoniaticians, voice researchers and singing teachers, but remains the commonly used term for that time when a boy chorister’s career ends. Given the direct association of this event with puberty, puberty and possible changes in its timing are matters of primary concern to those whose work and interest is with boy choristers.

During puberty there are changes to the length and tissue composition of the vocal folds and the size of the larynx. The neck also increases in relative volume, which is associated with a lower relative larynx position and consequent enlargement of the entire vocal tract. Similar changes occur in the vocal apparatus of girls but are more marked and obvious for boys, as are the increases in muscular strength and vital capacity. There is marked sexual dimorphism with regard to the thyroid cartilage which grows to an elongated shape in boys and a more rounded shape in girls. This results in the clearly visible protrusion of the so called “Adam’s apple” in boys at later stages of puberty and accommodates the greater fold length associated with greater voice drop in boys.

Puberty is not considered to be a single event but is almost universally defined by five stages identified and described by Tanner (1978). So-called “Tanner staging” relies on two sets of five descriptive photographs in which the growth of pubic hair and characteristic and predictable changes to the size and morphology of the genitalia are employed as visual indicators. These continue to be cited with a high degree of frequency in the medical literature, though they are subjective in character (Euling et al, 2008). They provide a descriptive reference point against which other measurable quantities may be compared and have been shown to be closely associated with similarly staged changes in pitch and quality of the voice (Harries *et al*, 1998). Puberty is also a time for both sexes of neurological change and significant brain development, associated with cognitive, emotional and psycho-social change that is no less significant than the physical changes. These psycho-social changes are associated with a second peak in the social construction of gender identity (the first of which occurs at age 7 – 8, see Skelton *et al*, 2009) in which boys develop a transitory hyper-sensitivity to issues of masculinity. The voice has been shown to play a critical role in this process (Ashley, 2008).

Puberty is thought to be caused by the action of a small neuronal cluster within the hypothalamus which results in secretion of gonadoliberin, a stimulant that controls the release of gonadotrophins from the pituitary gland. A sequence of chemically driven gonadal and adrenal activation is thus consequent upon the events within the hypothalamus which appear to be the trigger of puberty. In boys, the stimulation of gonads results in the production of dihydrotestosterone within the testes. This in turn stimulates the secondary sexual characteristics, of which laryngeal development is one. This action of the testes is clearly important as in boys the very first external sign of pubertal onset is an increase in testicular volume to above 3ml. This occurs in most cases before the first appearance of

pubic hair. The appearance of this 3ml testicular volume is now considered to be the most reliable marker of pubertal onset (Largo & Prader, 1983) and the testes continue to grow rapidly during pubertal development, reaching an adult size of up to 25ml by Tanner Stage 5. It is probable that this rapid testicular enlargement, which is accompanied by darkening and wrinkling of the scrotal skin gives rise to the popular myth amongst boys that “your voice breaks when your balls drop”.

Is there a secular trend?

The term “secular trend” is used frequently in the medical literature and is understood in the present context to mean a gradual, unidirectional trend in measurements such as stature or pubertal onset over time. The direction of the trend is variously toward greater stature, earlier onset of puberty and earlier attainment of pubertal climax. Almost all authorities are agreed that the timing of puberty is dependent upon both genetic and environmental factors. Research has tended to concentrate on environmental factors for understandable reasons. However, a key question remains unanswered and this is whether there is a genetically determined range for the timing of puberty which represents the optimal phenotype. This question is extremely important because it would inform two other questions of fundamental significance to the present discussion:

- Has the secular trend slowed or approached a terminal point in certain populations because the optimal phenotype has been achieved?
- Would it be more accurate to state, not that puberty is coming sooner, but that in the late nineteenth and early twentieth century it was delayed by environmental factors such as poor nutrition?

The latter question is of some interest since there may have been a secular trend in the reverse direction between previous periods during which composition for boys’ voices flourished but which predate present day medical records. A reverse secular trend was found to be associated with conflict in Croatia and Bosnia (Amselem *et al*, 2007).

Popular discussions of puberty tend both to exaggerate and to sensationalise the issue, often linking early puberty to popular discourses as “toxic childhood” (Palmer, 2006) which encourage “moral panics” (Cohen, 1987; Males, 2002; Altheide, 2009) about precocious sexuality in tabloid newspapers. The medical literature is far more cautious and conservative. Summarising the available evidence, the recent large scale Cambridge Primary Review concludes that the secular trend has been significant in the past, but that the case for its current continuance is unsubstantiated. The review, moreover, acknowledges that changes in attitudes, behavior and appearance may be greater than biological changes, and suggests that this encourages a subjective belief that puberty is coming earlier (Alexander *et al*, 2010: 92 – 93).

The Cambridge Primary Review, however, says more about girls than boys, and this undoubtedly reflects the general position in the medical literature. Unfortunately, sexual dimorphism appears to be significant and it can be stated with confidence that the volume of research on girls significantly outweighs that on boys, the reason being that much of the research is driven by concerns about the reproductive health of populations. The ready availability of data on girls has tempted some writers to infer trends in boys from girls but the extent of dimorphism is such that this is clearly not appropriate (Euling, Sherry *et al*, 2008).

The Euling *et al* paper is one of a small number of expert review panels set up to consider all the available evidence and must, therefore, be considered to be amongst the most authoritative sources available. Its conclusion was that

... on the basis of few studies and reliability issues of some male puberty markers, the current data for boys are insufficient to evaluate secular trends in male pubertal development.

Another large scale expert review was that of Amselem et al (2007) and this similarly concludes that the sum of available evidence demands further research and does not justify a confident conclusion that puberty is invariably coming sooner for all boys. This review gives particular attention to the wide variations in pubertal timing across ethnic groupings, social classes and geographical regions. A crucial point is that it is inappropriate to draw a conclusion for a specific population (i.e. English chorister boys) from the synthesis of data that are both diverse and relatively rare.

In general the medical literature searched through Medline, Web of Knowledge and Scopus appears to suggest the following:

- Findings for the timing of puberty in boys are less obvious than for girls in the diverse longitudinal studies that have been conducted.
- Several studies on specific populations (e.g., Denmark) showed that there did appear to be a downward trend in the onset of puberty in boys; usually related to Body Mass Index (BMI). However, other larger-scale secular studies of this topic showed only small and/or non-significant results. There was no clear evidence in non-regional populations to suggest a definite downward trend in puberty onset in boys.
- The largest and most prevalent downwards trend in puberty onset appears to be found in the United States. Although several of the regional studies also show a downward trend, they do not seem to have matched the extent of the US downward trend.
- Environmental factors such as nutritional status, chronic diseases, migration to a healthy environment, frequent infectious diseases, pollution, war and exposure to insecticides are all thought to influence pubertal onset.
- A large portion of the literature was concerned with puberty timing for children with developmental disorders and was more focused on menarche in girls than puberty signs in boys. Most of the research associated with both boys and girls found more significant results with girls.

It seems not unreasonable to state that a sufficiently large scale study of those populations that are specific to chorister singing has yet to be undertaken. Until the time when such a study yields results, the balance of evidence suggests a cautious and conservative approach that takes account of the subjective pressures to believe that puberty is coming sooner for choristers. The least conservative study identified was that of Liu et al (2000) who were concerned that the reference standards set by Tanner (1978) might be out of date and set out to establish new reference points through a study of changes in height velocity in 2432 Swedish children. Their conclusion was that there had been a downward secular trend which now justified new standards. Mean age of onset in boys was found to be 9, with peak height velocity occurring at 13.5 years. This represents an advance of onset of over 1.5 years and about 0.5 years at climax, which seems relatively extreme and was not generally found in other studies. Height velocity is also a less accurate and reliable method of assessment than testicular volume.

Nutritional status

Some researchers have speculated that environmentally induced variability in pubertal timing may occur because the body has a genetic survival mechanism that will initiate reproduction sooner when a population is threatened. This may or may not be so, but differences across time periods, geographical regions and heterogeneity with geographical

regions are both established (Amselem et al, 2007). Lejarraga *et al* (1976) found no trend in Argentina during the decades preceding their study whereas Castellino *et al* (2005) found that both boys and girls in Italy were experiencing pubertal onset one year earlier than the reference data provided by Tanner during the 1960s. Krawczynski *et al* (2003) studied the secular trend in Poland over eight different periods from 1880 to the present. During the twentieth century the 1950s and 70s saw the greatest trend, and the 1940s the least. They concluded that there had been an irregular and unstable trend toward increased height and weight during the twentieth century, but that the secular trend in Poznan, Poland has now ceased.

Vercauteren (2003) provides data to suggest that the trend in Belgium has now stopped and suggests that this is accounted for by the stabilization of nutritional status in an advanced Western democracy. Cameron (1979), however, concluded that the secular trend had ended in London during the 1960s for similar reasons. As it may have started again, this is clearly an area of uncertainty. Attention in the Western world has now shifted from stunted stature to obesity. Loesch *et al* (2000) note that in Australia, the secular trend for height has significantly slowed, whilst that for weight has speeded up. Whereas previously under-nutrition appears to have accounted for delayed puberty, the nutritional content of so-called “fast foods” which accounts for obesity appears to be having other effects. Most studies do agree that for girls, there does appear to be a clear trend toward earlier puberty that is associated with increasing adiposity. However, the apparently reliable relationship between obesity and menarche for girls has not been similarly established in puberty timing for boys (Kahl *et al*, 2007). Some studies conclude that obesity has little effect on boys’ pubertal timing (Denzer *et al*, 2007), some that it advances it (Heger *et al*, 2008; Sørensen *et al*, 2010), others that it retards it (Biro *et al*, 2006).

There was a flurry of publication in the late 1990s and early 2000s following investigation of the so called “critical mass hypothesis” associated with the adipocyte derived hormone, leptin (Clayton & Trueman, 2000; Colledge, 2004; Amselem et al 2007.) This important hormone appears to be associated with the transmission to the brain of information about the body’s energy stores and is thus implicated in the relationship between body mass index (BMI) and pubertal onset. The “critical mass” hypothesis maintains that sexual maturation is regulated by chemically transmitted signals about body height and adipose cell content. According to Amselem et al (2007) leptin deficiency could be the primary cause of delayed puberty and menarche in undernourished children. This would support the possibility that certain English populations are simply recovering from delayed pubertal onset in the past. Unfortunately, this has once again only been reliably established as the case for girls. Boys appear to experience leptin fluctuations in a different way. In boys the increase is interrupted in early puberty when testosterone and lean body mass increase, whereas in girls, leptin and body fat mass continue to increase during puberty (Rogol, 2000).

Mantzoros et al (1997) did find that serum leptin levels rose by 50% in a sample of eight healthy Caucasian boys just before pubertal onset and then decreased to baseline values of the initiation of puberty, remaining stable for two years. They concluded that circulating leptin levels are an important signal responsible for triggering the onset of puberty in boys. Mills *et al* (1986) excluded obese boys from their study and found that puberty occurred earlier in boys who had been significantly heavier than the mean at the ages of six months, two years and four years. Boys who had matured earlier by the age of 14 had all been taller and possessed greater muscle mass than other boys prior to six years of age. This confirmed earlier results of Douglas & Simpson (1964). These studies do suggest that there are other important variables that might be masked by a failure to control for obesity. If there an evolutionary advantage of larger, more muscular boys reaching reproductive maturity sooner exists, this could be relevant for chorister studies.

Whether or not obesity should be considered a pathology that justifies the exclusion of obese boys from chorister studies is indeed a significant point. The UK itself has consistently been one of the most unequal societies in Europe (Wilkinson & Pickett, 2009). Over the last 150 years, English working class boys have gone from a state of malnutrition that rendered them on average 22cm shorter than upper class boys in 1890 to a present day state of obesity. The Newsom report of 1963 showed a difference of seven pounds in the mean weight and one and quarter inches in the mean height between middle and working class boys. The two nutritional pathologies of malnutrition and obesity clearly have measurable consequences linked to changes over time that happen differently according to social class. In 2006, the House of Commons Public Accounts Committee on tackling obesity recorded that 17% of working class children under the age of 11 were obese as against 12% of the children of professional and managerial parents. The middle classes, from which most English chorister boys are drawn, are thus less susceptible to changes attributable to nutrition and stature than the working classes and this is a factor that needs to be controlled in studies.

Endocrine disruption

Endocrine-disrupting chemicals (EDCs) have also been targeted by some researchers, much more in the USA than in Nordic countries (Juul *et al*, 2006). Euling *et al* (2008b) however are confident that the alleged role of EDCs this remains controversial. Nebesio & Pescovitz (2005) reach a similar conclusion about endocrine disruption to the more general conclusion about the secular trend, that the data supporting a role for products containing agents such as estrogen in disturbing the timing of puberty in humans are mostly speculative. Sharpe (2006) reports on clinical investigations driven by concerns about low sperm counts in young men and the possibility that this is the consequence of EDC action on early testicular development. His team concluded that there is currently no definitive evidence that exposure of humans to environmental chemicals can induce testicular dysgenesis and/or impair masculinisation.

There may also again be a social class dimension to this since middle class parents may be more likely to restrict their children's access to such chemicals. More than one other study has also established a relationship between earlier puberty and domestic stress. Boys living without fathers at age 14 experience earlier puberty than boys in stable families according to Bogaert (2006). Boys living with step fathers may experience puberty earlier than boys living in more stable families (Arim *et al*, 2007). No general conclusion can be drawn from this, but the findings demand further exploration. The degree to which problems of nutrition and stress might affect the working more than the middle classes means that it is unwise to assume that any data drawn from wide populations will be a reliable guide to the conditions under which choristers develop and reach puberty. Physical and demographic data specific to the populations from which choristers are drawn need to be obtained before any confident statements can be made about the secular trend with regard to the singing of English boy choristers.

Another factor that must be considered is that the relationship between stature increase and pubertal timing is not straightforward. Karlberg (2002) is amongst those who have found that the two trends of stature increase and pubertal onset are not strongly correlated. A genetic influence is also evident in twin studies. Two studies by Juul *et al* which note differences between the USA and Denmark may be particularly important. The study published in 2006 concluded that Danish boys were significantly taller than in 1964, but that their pubertal timing had not altered (Juul *et al*, 2006). However, the study published in 2007 did find a statistically significant downward trend in age at voice "break" of Danish boys (Juul *et al*, 2007), although the age range quoted of 13.9 – 14.6 years for 2003 would not impact greatly on a significant portion of choristers. Similarly to Mills *et al*, Juul *et al* concluded that the heaviest quartile of eight year old boys had significantly greater risk of "early" voice break

than the lightest eight year olds. These studies, however, did not appear to control for obesity in the way that Mills *et al* did. Their conclusion that pre-pubertal BMI affects the timing of boys' puberty is therefore significant but not conclusive. The fact that Denmark appears to be catching up the USA with regard to unhealthy children may be indicative of the possibility that the secular trend may not yet have peaked in all Western European states, but might be slowed down by improvements in nutrition. The Juul study, however, is directly contradicted by a contemporary German study (Kahl *et al*, 2007) which not only gives 15.1 as the median for "voice low" but states that the onset of puberty is not significantly earlier than in other European studies. Buyken *et al* (2009) appear to advance this area of knowledge through a longitudinal study of German boys and girls which suggests that pre-pubertal BMI is not critical for pubertal onset but results in earlier attainment of the later stages of puberty.

Sun *et al* (2005) found slight evidence of a secular trend in non-Hispanic American white boys between 1966-70 births and 1988-94 births, but no corresponding trend in black boys. The more significant complication in this study was that whilst the white boys appeared to experience pubertal onset and Stages 2-4 sooner, Stage 5 actually came later. Findings such as these, if confirmed by other studies, are particularly relevant for choristers since what little serious scientific research as exists on boy treble voices suggests that a clear differentiation needs to be drawn between pubertal onset and pubertal climax. There is little doubt that most senior boys in English cathedral choirs will be at Cooksey Stage II or Stage IIa. They have thus passed pubertal onset and it is probable that this has been the case since at least the fifteenth century. The Williams (2010) study confirmed this in some detail for "Boy H". Some choristers I assessed in my earlier research were at stage 3 and clearly struggling as a result.

The empirical task

The recent study of Willis & Kenny confirms that an average speaking pitch of 195Hz is the critical point for chorister voices. This compares closely with my own previous work that concluded that 185Hz was the critical point. The difference is small and if the average of 190Hz is taken there is a clear empirical task. The mean age and mean speaking pitch of a sufficiently large cohort of Y8 boys (the year in which the majority of choristers leave the choir) during the school summer term must be established. If the mean speaking pitch is found to be lower than 190Hz, then a serious case for reviewing the impact of pubertal status on choristers is established, regardless of the inconclusive medical evidence reviewed here. This alone, however, will not establish the reality and extent of the secular trend, for it may be the case that choristers have always continued with treble singing past the 190Hz point. If this is so, a review of how choristers are taught and deployed is called for.

References

Alexander, R., Pugh, G., Hodgson, H. and Margrave, R. (2010) *The Primary Review: children, their world, their education*. London: Routledge

Altheide, D. (2009) Moral panic: From sociological concept to public discourse. *Crime, Media, Culture*, 5 79-99.

Amselem, S., Carel, J., De Roux, N., MacCari, S., Prevot, V. and Susane, C. (2007) *Growth and Puberty: secular trends, environmental and genetic factors*. Paris: Institut national de la sante et de la recherche medicale.

Arim, R., Shapka, J., Dahinten, V. and Willms, J. (2007) Patterns and correlates of pubertal development in Canadian youth: effects of family context. *Canadian Journal of Public Health*, **98** (2) 91-96.

Ashley, M. (2010) Technique or Testosterone? an empirical report on changes in the longevity of boy singers. *Journal of Singing*, (November/December) .

Ashley, M. (2008) *Teaching Singing to Boys and Teenagers: the young male voice and the problem of masculinity*. Lampeter: Mellen.

Biro, F., Khoury, P. and Morrison, J. (2006) Influence of obesity on timing of puberty. *International Journal of Andrology*, **29** (1) 286-290.

Bogaert, A. (2005) Age at puberty and father absence in a national probability sample. *Journal of Adolescence*, **28** (4) 541-546.

Buyken, A., Karaolis-Danckert, N. and Remer, T. (2009) Association of prepubertal body composition in healthy girls and boys with the timing of early and late pubertal markers. *American Journal of Clinical Nutrition*, **89** (1) 221-230.

Cameron, N. (1979) The growth of London schoolchildren 1904 - 1966: an analysis of secular trend and intra-county variation. *Annals of Human Biology*, **6** (6) 505-525.

Castellino, N., Bellone, S., Rapa, A., Vercellotti, A., Binotti, M., Petri, A. and Bona, (2005) Puberty onset in Northern Italy: a random sample of 3597 Italian children. *Journal of Endocrinol Investigation*, **28** (7) 589-594.

Clayton, P. and Trueman, A. (2000) Leptin and puberty. *Archives of Disease in Childhood*, **83** (1) 1 - 4.

Cohen, S. (1987) *Folk Devils and Moral Panics: the creation of mods and rockers*. Oxford: Blackwell.

Colledge, W. (2004) GPR54 and puberty, *Trends in Endocrinology and Metabolism*, **15** (9) 448 - 453.

Cooksey, J. (2000) Voice transformation in male adolescents. In: L. Thurman and G. Welch (eds) *Bodymind and Voice - foundations of voice education*. Iowa City, IA: The VoiceCare Network. 718-738.

Cooksey, J. (1993) Do adolescent voices 'break' or do they 'transform'? *Voice*, **2** (1) 15-39.

Cooksey, J. and Welch, G. (1998) Adolescence, singing development and national curricula design. *British Journal of Music Education*, **15** (1) 99-119.

Cooper, I. and Wikstrom, T. (1962) Changing Voices. *Music Educators Journal*, **48** (4) 148-151.

Curwen, J. (1899) *The Boys' Voice: a book of practical information on the training of boys' voices for church choirs*. 3rd ed. London: J. Curwen.

Denzer, C., Weibel, A., Mucic, R., Karges, B., Sorgo, W. and Wabitsch, M. (2007) Pubertal development in obese children and adolescents. *International Journal of Obesity*, **31** (10) 1509-1519.

Douglas, J. and Simpson, H. (1964) Height in relation to puberty, family size and social class: a longitudinal study. *The Millbank Memorial Fund Quarterly*, **42** (3) 20-34.

Euling, S., Herman-Giddens, M., Lee, P., Selvan, S., Juul, A., Sorensen, T., Dunkel, L., Himes, J., Teilmann, G. and Swan, S. (2008) Examination of US puberty-timing data from 1940 to 1994 for secular trends: panel findings. *Pediatrics (supplement edition)*, **121** S172-S191.

Euling, S., Selevan, S., Pescovitz, O. and Shakkebaek, N. (2008) Role of environmental factors in the timing of puberty. *Pediatrics (supplementary edition)*, **121** S167-S171.

Harries, M., Walker, J., Williams, D., Hawkins, S. and Hughes, A. (1997) Changes in the male voice at puberty. *Journal of Laryngology and Otology*, **112**, 451 - 454 .

Heger, S., Körner, A., Meigen, C., Gausche, R., Keller, A., Keller, E. and Kiess, W. (2008) Impact of weight status on the onset and parameters of puberty: analysis of three representative cohorts from central Europe. *Journal of Pediatric Endocrinol Metabolism*, **21** (9) 865-867.

Herman-Giddens, P., Wang, L. and Koch, G. (2001) Secondary sexual characteristics in boys: estimates from the national health and nutritional examination survey III, 1988 - 1994. *Archives of Pediatric Adolescent Medicine*, **155** 1022-1028.

Howard, F. (1895) *The Child-Voice in singing: treated from a physiological and a practical standpoint and especially adapted to schools and boy choirs*. New York: H.W. Gray/Novello.

Kahl, H., Schaffrath, R. and Schlaud, M. (2007) Sexual maturation of children and adolescents in Germany. Results of the German Health Interview and Examination Survey for Children and Adolescents (KiGGS). *Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz.*, **50** (5-6) 677-685.

Karlberg, J. (2002) Secular trends in pubertal development. *Hormone Research*, **57** 19-30.

Krawczynski, M, Walkowiak, J. and Krzyzaniak. A. (2003) Secular changes in body height and weight in children and adolescents in Poznan, Poland, between 1880 and 2000. *Acta Paediatrica*, **92** 277 - 282.

Largo, R. and Prader, A. (1983) Pubertal development in Swiss boys. *Helvetica Paediatrica Acta*, **38** (3) 211-228.

Lejarraga, H., Cusminsky, M. and Castro, E. (1976) Age of onset of puberty in urban Argentinian children. *Annals of Human Biology*, **3** (4) 379-381.

Liu, Y., Wikland, K. and Karlberg, J. (2000) New reference for the age at childhood onset of growth and secular trend in the timing of puberty in Swedish. *Acta Paediatrica*, **89** (6) 637-643.

Loesch, D., Stokes, K. and Huggins, R. (2000) Secular trend in body height and weight of Australian children and adolescents. *American Journal of Physical Anthropology*, **111** (4) 545-566.

Males, M. (2002) *Framing Youth: 10 Myths About the Next Generation*. Monroe ME: Common Courage Press.

Mantzoros, C., Flier, J. and Rogol, A. (1997) A longitudinal assessment of hormonal and physical alterations during normal puberty in boys. V. rising leptin levels may signal the onset of puberty. *Journal of Clinical Endocrinology and Metabolism*, **82** (4) 1066-1070.

Mills, J., Shiono, P., Shapiro, L. C., P. and Rhoads, G. (1986) Early growth predicts timing of puberty in boys: results of a 14-year nutrition and growth study. *Journal of Pediatrics*, **109** (3) 543-547.

Nebesio, T Pescovitz, O. (2005) Historical Perspectives: Endocrine Disruptors and the Timing of Puberty. *Endocrinologist*, **15** (1) 44- 48

Ong, K., Ahmed, M. and Dunger, D. (2006) Lessons from large studies on timing and tempo of puberty (secular trends and relation to body size): The European trend. *Molecular and Cellular Endocrinology*, **254-255** 8-12.

Palmer, S. (2006) *Toxic Childhood: how the modern world is damaging our children and what we can do about it*. London: Orion.

Sharpe,R. (2006) Pathways of endocrine disruption during male sexual differentiation and masculinisation. *Best Practice & Research Clinical Endocrinology & Metabolism*, **20** (1) 91 - 110

Skelton, C., carrington, B., Francis, B., Hutchings, M., Read, B. and Hall, I. (2009) Gender matters in the primary classroom: pupils' and teachers' perspectives. *British Educational Research Journal*, **35** (2) 187-204.

Sørensen, K., Aksglaede, L., Petersen, J. and Juul, A. (2010) Recent changes in pubertal timing in healthy Danish boys: associations with body mass index. *Journal of Clinical Endocrinol Metabolism*, **95** (1) 263-270.

Stalhammar, B. (2003) Music Teaching and Young People's Own Musical Experience. *Music Education Research*, **5** (1) 61-68.

Sun, S., Schubert, C., Liang, R., Roche, A., Kulin, H., Lee, P., Himes, J. and Chumlea, W. (2005) Is sexual maturity occurring earlier among U.S. children? *Journal of Adolescent Health*, **37** (5) 345-355.

Swanson, F. (1977) *The Male Singing Voice Ages Eight to Eighteen*. Cedar Rapids, LA.: LaurancePress.

Tanner, J. (1978) *Foetus into Man: physical growth from conception to maturity*. London: Open Books.

Tanner, J. and Whitehouse, R. (1976) Clinical longitudinal standards for height, weight, height velocity, weight velocity, and stages of puberty. *Archives of Disease in Childhood*, **51** 170-179.

Wayman, J. (2009) *Identification of the Adolescent Male Voice: unchanged vs. falsetto*. M.Mus Ed. Graduate Faculty, Texas Tech University. Unpublished.

Welch, G., Himonides, E., Papageorgi, J., Saunders, T., Rinta, C., Stewart, C., Preti, J., Lani, M. V. and Hill, J. (2009) The National Singing Programme for primary schools in England: an initial baseline study. *Music Education Research*, **11** (1) 1-22.

Williams, J. (2010) *The implications of intensive singing training on the vocal health and development of boy choristers in an English cathedral choir*. PhD. University of London, Institute of Education. Unpublished.

Williams, J. (2007) Letter to *Journal of Singing*, the Journal of the American National Association of Teachers of Singing. *Journal of Singing*, **64** (1).

Willis, E. & Kenny, D. (2008) Relationship between weight, speaking fundamental frequency and the appearance of phonational gaps in the adolescent male changing voice.. *Journal of Voice*, **22** (4) 54 - 74.