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Factors affecting onset of puberty in Denizli province in Turkey

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The relationship between the possible factors affecting pubertal onset and pubertal timing was investigated in the Denizli province in Turkey.

A total number of 3311 subjects (1562 girls, 1749 boys) aged 6-16.5 years participated in this study. Body mass index (BMI) was calculated. Pubertal stages were assessed according to methods of Marshall and Tanner. Testicular volume was determined using Prader orchidometer. Menarcheal age was recorded. All parents and students completed different questionnaires on demographic variables affecting pubertal timing such as socioeconomic conditions, psychosocial factors, exercise, nutritional status, chronic diseases, migration and birth weight. Using distribution percentiles of pubertal stages according to age, the relation between pubertal timing and factors affecting puberty was investigated.

There was no significant association between exercise, birth weight, migration, chronic disease, and socioeconomic status and age of puberty onset. Menarcheal age of overweight and obese girls was significantly lower than that of girls with normal weight. In-family stress was the cause of early puberty in girls and of delayed puberty in boys.

Key words: pubertal timing, menarche, obesity, migration, socioeconomic environment, intrauterine growth retardation, stress, chronic disease.

There is wide variation in the normal onset and rate in which a child progresses through puberty, and there are many conditions that may affect this normal process. This variability involves genetic factors, as indicated by the studies on heritability of menarcheal age[1]. Other factors such as ethnicity, nutritional conditions, and secular trends have been shown to influence the physiological range in age at the onset of puberty[1]. Secular trends appear to influence the physiological range in pubertal onset. The reasons for this secular trend relate to the decline in the severity and frequency of illness and to a better health and nutritional status of the general population. These factors, in turn, are the result of improvements in medical care and socioeconomic conditions[2].

A number of studies have noted the association between increased body mass index (BMI) and earlier onset of puberty or menarche in the United States and in Europe[2-5]. The same association between adiposity and earlier pubertal maturation does not appear in boys[6].

Factors such as adoption, migration from developing to industrialized countries, and rural-urban migration are important determinants of nutritional status and growth of adolescents from developing countries because of the improvement in socioeconomic status and behavioral changes to which they contribute[7-10].

Different stresses, such as acute or chronic illnesses and adverse physical or psychological conditions, are known to depress the hypothalamic-pituitary-gonadal system[11]. There

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is growing interest in the potential role of psychological factors in growth and maturation. In particular, the influence of familial stress upon growth and maturation has been studied somewhat extensively. Several reports indicate accelerated sexual maturation in association with a stressful family environment[12,13].

Delayed onset of puberty and reduced pubertal growth spurt are often reported in patients suffering from chronic diseases. The causes of abnormal puberty can be divided into two groups: firstly, the adverse effects due to the disease itself; and secondly, the adverse effects due to drug therapy, namely glucocorticoid therapy[14]. Pubertal delay could be the result of under-nutrition, emotional deprivation, excess increase in protein degradation, accumulation of toxic substances, stress, and the secondary effects of therapy[15-17].

Epidemiological evidence relating intrauterine growth to pubertal maturation is sparse. In the general population, some authors found no significant correlation between birth weight and menarcheal age, whereas others reported that thin newborns enter puberty earlier[18-20].

In this study, we investigated the role of possible etiologies that might affect onset of puberty in schoolchildren living in the Denizli province in Turkey.

Material and Methods

A cross-sectional school-based study was performed between March and May 2005. The study included schoolchildren in grades 1 to 8 in primary schools in the Denizli province. The target population of the survey was all 116,229 children attending primary schools.

Denizli is located in the west of Turkey and is the second principal city in the Aegean part of the country. The population of Denizli was cited as 850,029 in 2000. From 1980 to 2000, the population employed in the industry, services, construction, and agricultural sectors increased by 119%, 84.2%, 42.7%, and 32.9%, respectively. Per capita gross national product was US$ 2807 in 2000.

A multistage stratified clustered sampling design was employed. It has been hypothesized that the age of pubertal onset might occur one year earlier considering the secular trend. The age of pubertal onset was predicted as 9±0.5 in girls and 10.5±0.5 in boys. Using the formula below, clustered sample was calculated, and it was decided to attain a target value of at least 1218 girls and 1648 boys.

$$n = \frac{\bar{N}\,(t_{1-\alpha})^2 \times (x)^2}{S^{\bar{2}}\,(N-1) + (t_{1-\alpha})^2 (x)^2}$$

All of the urban and rural regions of Denizli were included in the study. Primary schools were randomly selected. Subsequently, one class from every eligible grade from each school was randomly selected and all students from the selected classes were included in the sample.

A total of 3311 subjects (1562 girls, 1749 boys) aged 6 -16.5 years from both urban and rural schools participated in this study. Before the initiation of the study, a detailed explanation was given to all teachers, participants and their parents. Approval of the local authorities was obtained. Informed consent was obtained from a parent. Weight and height were measured and BMI was calculated. Body weight was measured to the nearest 0.1 kg with a balance scale (Baurer, PS 07), and height was measured to the nearest 0.1 cm with a stadiometer (Hyssna Limfog, AB) with subjects lightly dressed and without shoes. BMI was calculated as weight (kg) divided by height square (m^2). The degree of obesity was quantified using Cole's reference data[21]. Pubertal stages were assessed by clinical examination according to methods of Marshall and Tanner[12,13]; breast stages (B1-5) in girls, genital stages (G1-5) in boys, and pubic hair (PH1-5) in both sexes were evaluated[22,23]. To increase the reliability of visual assessment of B2, it was evaluated both by inspection and palpation. When two breasts of an individual were not in the same stage of development, the stage of the more advanced side was recorded. The stage of genital development (G) in boys was based on direct palpation measurement of testicular volume (TV), which is usually considered to be an important indicator in the assessment of pubertal development rather than on inspection of changes in scrotal skin and enlargement of the testes[24]. Not to cause cremaster reflex, testis examination was done while the subject was standing and in appropriate room temperature. TV was determined using a Prader orchidometer[25]. This orchidometer consists of 12 models of rotation ellipsoids with volumes of 1, 2, 3, 4, 5, 6, 8,

10, 12, 15, 20, and 25 ml. The registered volume was the volume of rotation ellipsoid palpated to the nearest volume to that of the testis examined. If the two testes were not identical, the larger was chosen to determine TV. A testis volume of 1-3 ml was assessed as G1, 4-8 ml as G2, 10-15 ml as G3, 15-20 ml as G4 and greater than 25 ml as G5 (25,26). Fourteen boys did not accept for their TV to be measured; thus, TV could be measured in 1735 boys.

The onset of puberty was accepted as breast development at Tanner stage 2 (B2, appearance of a breast bud) for females and a testicular volume equal to or greater than 4 ml for males[25,27]. The age at menarche was determined by asking each girl when she had her first menstruation period. Menarcheal age was recorded.

The measurements of height and weight and examination of puberty were performed by trained staff. Breast and pubic hair development for each girl were assessed by one female pediatrician, and genital and pubic hair development for each boy were assessed by one male pediatrician. To validate the accuracy of the examination of puberty, pubertal staging in 50 students was done by two observers. One of the observers was a pediatric endocrinologist and the other was a pediatrician well trained in pubertal staging. Inter-observer differences were tested using kappa analysis. In girls, kappa statistics (agreement corrected for chance) were 0.902 for pubic hair stage and 0.806 for breast development. In boys, kappa statistics were 1.000 for pubic hair stage and 0.953 for genital development. Inter-observer adjustment was evaluated as very good and both pediatricians were able to assess pubertal stages with a high degree of accuracy.

All parents completed a questionnaire including 49 questions on demographic variables affecting pubertal timing such as socioeconomic conditions, psychosocial factors, exercise, nutritional status, chronic disease, migration and birth weight. Similarly, all students completed a questionnaire including 10 questions on demographic variables affecting pubertal timing. Categorization of socioeconomic class was based on the occupation and education status of parents by applying the Boratav index[28]. High- or low-grade exercise status was described as exercising at least three times weekly or less. Categorization of psychosocial factors was based on family dynamics. These included death of one or both parents, separation or divorce of parents, parental marital conflict, parent-child conflict, and in-family violence. Nutritional status was classified according to BMI. Migration status was queried regarding whether it was from rural region to urban region or vice versa or from one city to another. Chronic disease was accepted to exist when a recurrent infection, gastrointestinal, renal, respiratory or endocrine disease, chronic anemia, immunodeficiency or eating disorder was present.

The relation between possible factors affecting puberty and the age at the onset of puberty was investigated using distribution percentiles according to ages of pubertal stages. To achieve an adequate number of subjects statistically, subjects whose age of puberty onset was below 10p were grouped as early puberty and subjects whose age of puberty was above 90p as delayed puberty. Distribution of the group between these two was described as normal puberty. The relation between possible factors affecting puberty and these three groups was investigated and compared.

Statistics

SPSS for Windows version 13.0 was used for analyses. Data were used to calculate means, standard deviations (SD), medians, ranges and percentiles of ages for sexual development in girls and boys. One-way ANOVA was used to compare the relation between the mean age for menarche and weight status. Comparison of possible factors affecting pubertal onset to pubertal timing and comparison of menarcheal age to gestational age and birth weight were made using X^2 test.

Results

When B2 in girls and G2 in boys were considered as the first signs of puberty, mean age percentile values for B2 and G2 were 10p: 7.19, 50p: 9.13, 90p: 11.10 years and 10p: 10.31, 50p: 11.80, 90p: 13.60 years, respectively. Pubertal timing in girls and boys is shown in Table I. According to percentile distribution, 21 girls out of 659 were diagnosed as early puberty (below 10 p) and 49 girls as delayed puberty (above 90 p). Early and delayed puberties were observed in 40 and 34 out of 429 boys, respectively.

Table I. Pubertal Timing in Girls and Boys

	Girls		Boys	
	n	%	n	%
Early puberty	21	3.2	40	9.3
Normal puberty	589	89.4	355	82.8
Late puberty	49	7.4	34	7.9
Total	659	100	429	100

The mean age at menarche was 12.41±0.92 (9-15) years. Menarcheal age was significantly lower in girls with BMI above the median compared with girls with BMI below the median (12.11 vs.12.53 years; p<0.0001) (Table II). No statistically significant difference was found between gestational age, birth weight and menarcheal age (p>0.05).

Table II. Mean Age for Menarche According to Weight Status

Weight	n	Mean age ± SD (year)
Normal	229	12.53±0.88*
Overweight	68	12.05±0.89
Obese	9	12.11±1.36*
Total	306	12.41±0.92

*p<0.0001.

The relation between exercise, birth weight, migration, chronic disease, socioeconomic status and onset of puberty was not significant (p>0.05). In-family stress was the cause of early puberty in girls and of delayed puberty in boys (p<0.05) (Table III).

Discussion

Under physiological conditions, factors affecting the genetic control of hypothalamic functions are predominant in determining the individual variations in the timing of pubertal onset. In pathological conditions, however, these variations can involve different genetic susceptibilities and the interaction of environmental factors. Environmental effects on the mechanism of pubertal onset may start during intrauterine life. In the general population, some authors found no significant correlation between birth weight and menarcheal age[20], while others reported that thin newborns entered puberty earlier[18,19]. Lienhardt[29] described delayed onset of puberty in short children born with intrauterine growth retardation. Boys with intrauterine growth retardation had a reduced pubertal growth. In some other studies, it was reported that both boys and girls who were shorter or thinner at birth had an earlier onset of puberty[30,31]. It has been known that infants born small-for-gestational age had much lower levels of neonatal and childhood serum leptin, insulin-like growth factor-I and insulin-like growth factor binding protein-3[32-34]. It can be speculated that this subnormal neonatal hormone profile at birth may have a long-term imprinting effect on the postnatal timing of puberty, although the mechanism for this is unclear. No significant difference was found between birth weight and puberty onset in either sex in our study. Some studies have shown an earlier menarcheal age in girls with low birth weight or intrauterine growth retardation[18,30]. Although birth weight alone was not significantly related to age at menarche, girls who were relatively long and thin at birth attained menarche approximately six months earlier than did girls who were short and light[18]. Menarcheal ages of girls born small- or large-for-gestational age were comparable to those born with normal weights in our study.

Table III. The Relationship Between the Possible Factors Affecting Pubertal Onset and Pubertal Timing

Possible factors affecting pubertal onset	Pubertal timing in girls	Pubertal timing in boys
Violence of mother to offspring	Early*	Late*
Violence of father to offspring	NS	Late*
Violence of father to mother	Early*	NS
Parental-marital conflict	Early*	NS
Parental separation or divorce	NS	Late*
Birth weight	NS	NS
Weight status	NS	NS
Chronic disease	NS	NS
Socioeconomic status	NS	NS
Migration	NS	NS

*p<0.05; NS: Non-significant.

Many studies have examined the relationship between BMI and timing of maturation[3-5,35,36]. Leptin serves as a metabolic signal for puberty to progress, and appears to be a permissive factor rather than the trigger for the onset of puberty. Leptin levels are greater in blacks, even after adjustment for pubertal stage and fat mass, leading to speculations that the greater body fat in prepubertal black girls may increase the likelihood for early onset of puberty[3,37]. The same association between adiposity and earlier pubertal maturation does not appear in boys[4]. No statistically significant difference was found between the age of puberty onset and the weights, whether normal or overweight, of children of either sex in our study. However, menarcheal age of obese and overweight girls was significantly lower than that of girls with normal weight. This finding was similar to that reported in the literature.

In developing countries, socioeconomic factors are often the main determinants of nutritional status and growth from childhood to adulthood throughout adolescence[38,39]. Socioeconomic factors play a unique role in the secular trend of menarcheal age, and menarcheal age decreases as socioeconomic status improves[19,40,41]. Lindgren[42] showed that the relation of social class to age of pubertal stage did not differ significantly. The relation of the age of puberty onset and menarcheal age to socioeconomic status of the family was not significant in our study.

Every child with any chronic disease could present with delayed puberty (due to recurrent infections, immunodeficiency, gastrointestinal disease, renal disturbances, respiratory illnesses, chronic anemia, endocrine disease, eating disorders, exercise and a number of miscellaneous abnormalities)[17]. The degree to which growth and pubertal development are affected in chronic illness depends upon the type of disease and individual factors, as well as on the age at illness onset, its duration and severity. The earlier its onset and the longer and more severe the illness, the greater the repercussions on growth and pubertal development. The mechanisms that trigger the start of physiological puberty remain unknown. Although malnutrition is probably the most important mechanism responsible for delayed puberty, emotional deprivation, toxic substances, stress and the side effects of chronic therapy, among others, have been implicated in the pathophysiology of delayed puberty[14]. No significant association was demonstrated between the age of puberty onset and the presence of a chronic illness in girls in our study. The frequency of chronic disease in boys with delayed puberty was higher than that in boys who had normal pubertal timing, but this difference did not reach a significant level.

Longitudinal research has largely supported the hypotheses that socioemotional stressors are associated with earlier pubertal timing in girls[43]. Greater family conflict predicted earlier menarche[44]. Ellis and Garber[43] showed that a history of mood disorders in mothers predicted earlier pubertal timing in daughters, and this relation was fully mediated by dyadic stress and biological father absence. Hulanicka and co-workers[45] reported that girls exposed to familial distress (these include death of one or both parents, separation or divorce of the parents, a single-mother family, prolonged illness of a member of the family, presence of social deviations such as alcoholism of one or both parents, and criminal records) are more likely to have an early puberty, which is associated with a short final stature. Our study revealed early puberty in girls was more prevalent in families with parental - marital conflict, physical violence of father to mother and of mother to offspring. Tschann[46] reported that pubertal timing was not related to emotional distress in either sex. Paternal alcoholism led to a delay of male pubertal onset, as suggested by the hypothesis that stress activates the hypothalamic-pituitary-adrenal axis, and inhibits the hypothalamic-pituitary-gonadal axis[47]. We found delayed puberty was noted in boys living in families with parental - marital conflict and parental separation or divorce.

It is well known that intensive exercise during the peripubertal period results in growth and pubertal delay[17]. This is particularly seen in those sports or activities where a low weight is beneficial. Athletes also constitute a high-risk group for developing eating disorders. At the same time, it was thought that the stress involved with strenuous exercise could inhibit or alter the gonadotropin-releasing hormone (GnRH) pulse generator[48]. Highly trained male athletes, like their female counterparts, may have a deficiency of hypothalamic GnRH[49]. On the contrary, it was concluded in another study that the body composition of young male

athletes is not necessarily affected, and there is no determined effect of intense training on their physical and pubertal development[50]. With respect to age of puberty onset, no significant difference was determined in either sex in our study between the group exercising 3 times or more a week and the group without this exercise frequency.

Following an initial report from Sweden, sexual precocity has been described in children migrating from developing countries, primarily through international adoption[1,51]. Urban populations are, on an average, richer than rural populations and have better living conditions, since wealth and economic, sanitary and social infrastructures are concentrated in cities. However, the gap between poor and rich is much greater in urban areas. These differences in wealth and living conditions, as well as differences in diet and food intake, between urban and rural environments are often related to differences in nutritional status. It was associated with earlier puberty and better nutritional status[10]. Endocrine-disrupting chemicals are widespread environmental substances that have been introduced by man and may influence the endocrine system in a harmful manner[52]. Migration may interrupt exposure to endocrine disrupters and precocious puberty might then result from withdrawal of their negative feed-back effect and/or from accelerated hypothalamic maturation. Our study showed no significant difference in either sex statistically between the age of puberty onset and migration status of the family. However, most of the migrated families had not made important geographic displacements; they were from village to city Centrum or from district to Centrum, all of which occurred within the borders of the city. For this reason, it was concluded that the effect of migration between countries on the age of puberty onset that is described in the literature does not refer to this type of migration.

In conclusion, there was no significant association between age of puberty onset and exercise, birth weight, migration, chronic disease, and socioeconomic status. Girls with a higher BMI have earlier menarcheal age. The most important factor affecting pubertal timing has been found to be in-family stress.

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