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A Life Course Study about Menarche Made Feasible by the Health and Demographic Surveillance System in Matlab, Bangladesh

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Introduction

This study is about the age at menarche (a biomarker) of adolescent girls in Matlab, Bangladesh, and the relative impact of contemporary and early childhood nutritional determinants. Application of the life course perspective requires analyses of longitudinal data. The study involves the follow-up of 707 under-five children who were enrolled in a study on persistent diarrhoea conducted in Matlab in 1988-1989 by Baqui. The follow-up survey took place in 2001. By that year the under-fives had grown up to adolescents, aged 12 to 16 years. Tracing the adolescents was feasible because of the Health and Demographic Surveillance System, which is maintained in Matlab. The results reveal that the relative high median age at menarche (15.1 years) is associated with stunting in early childhood and particularly in adolescence. The study is an example of the further integration of the life course approach to reproductive health research within the discipline of demography.

Background: a life course approach to the study on menarche

The life course can be seen as the period from conception to death that encompasses the totality of experiences of life at a given time in history (Berger 1996). The emergence of the life course paradigm in demographic studies can also be observed in other disciplines among which including epidemiology, for which the research group of Barker is among the most salient exponents (Barker 1992; 1993; 1998; Eriksson et al. 1999; Barker et al. 2001). The shift from the 'life style paradigm' to the 'early life experience paradigm' was a major scientific revolution (Robinson 1992) and has also expanded to the field of women's health (see, for instance, Kuh and Hardy 2002) and reproductive health.

The first important experience in a woman's reproductive career is the menarche, the first menstruation. A late menarche may ‘run in the family’. There is some evidence of genetic predisposition on timing of menarche, which is among others grounded on the finding that age at menarche of mothers and their daughters is positively correlated (Gray 1993, p. 220). However, the predictive power of age at menarche of a girl’s mother is small (Graber et al. 1995). Genetic factors probably account for approximately 10 to 15 per cent of the observed variation in age at menarche (Gray 1993, p. 220). It is widely recognised that nutritional status is one of the most important non-genetic determinants of menarche (Riley et al. 1993). In general, better nourished girls reach menarche earlier than undernourished girls (WHO 2003). A review of studies on the determinants of menarche reflects a development that runs parallel to the aforementioned shift towards the adoption of the

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life course approach. In the early 1970s, timing of menarche was believed to be ‘triggered’ by a certain critical weight (Frisch and Revelle 1969; 1971), but the evidence for this relationship was weak (Trussell 1980). In later studies, other anthropometric indices such as adolescent height, mid-upper arm circumference and Body Mass Index were (also) positively associated with menarche (see, for instance, Delgado et al. 1985; Linhares et al. 1986; Maclure et al. 1991; Koprowski et al. 1999). More recently, there is growing support for the possibility that timing of menarche may be set *in utero* or early in life but may be modified by changes in body size and composition in childhood (Silva et al. 2003), a line of thinking which relates to the 'foetal origins of disease hypothesis' of Barker. However, as yet there are only a handful of reports that hint at foetal determinants of age at menarche (Rich-Edwards 2002) and the mechanisms involved are still unclear (Cole 2000).

Nutritional status in early life, a sensitive period

Among the population of Bangladesh malnutrition has been prevalent for generations as a result of which individuals are both prenatal as well as postnatal exposed to nutritional micro and macro deficiencies. In addition, in Bangladesh there is a well-documented list of factors (notably infectious diseases, diarrhoea, adverse environmental conditions and behaviours) which contribute to the ‘chain of risks’ impacting nutritional anthropometry in the negative. This chain of risks is an alternative pathway - that of *cumulative causation* - which describes how experiences in early life increase the likelihood of future events which in turn lead to a change in the risk of adult diseases and which can be advantageous or detrimental in their effect (Kuh and Ben-Shlomo 1997).

Within the Bangladeshi population stunting seems to have become an embodied[4] trait. The overall picture in Bangladesh is one of widespread malnutrition, particularly among women and children (Ross et al. 1996; ICDDR,B 2002; WHO 2003). For instance, 58 per cent of the under-fives in Bangladesh (1993-1995) is underweight, 55 per cent is stunted, 73 per cent is anaemic and 78 per cent has vitamin A deficiency (WHO 2003, p. 8). In addition, Bangladesh has the highest rate (50 per cent) of children born full-term with a weight at birth below 2500 grams as a result of intrauterine growth retardation (Pojda and Kelley 2000, pp. 3-4). Such a high prevalence of LBW is an *intergenerational* problem (Pojda and Kelley 2000, p. 2).

Gestation and early childhood are *sensitive* or *critical* for the adolescent stage in life. A delay in early life growth can hardly be stopped or reversed and after the age of two years the potential for catch-up growth[5] is indeed limited when such children remain in poor environments (Gillespie and Flores 2000, p. 2). If environmental conditions improve, particularly in terms of favorable nutrient intake, the period of adolescence could be *a window of opportunity* to catch up early life growth faltering (WHO 2003, p. 10), although evidence of complete catch-up in developing countries is limited (WHO 2003, p. 22). In one of the few studies on this, undertaken in Guatemala, catch-up growth was reported to take place in adolescents, but they did not fully negate the growth retardation of early childhood (Martorell et al. 1995). Also within developmental psychology such special time windows have been identified. Reviewing multi-dimensional development processes, Jenniskens and Verduin (1998,

[4] Embodiment describes how extrinsic factors experienced at different life stages are inscribed into an individual’s body functions or structures. This may be through a developmental process associated with critical periods (Kuh et al. 2005).

[5] Catch-up growth is defined as the recovering of a delay in growth (Silventoinen 2000).

p. 17) outline four points in life at which it is not possible to catch up on deficits (also called ‘points of no return’, i.e. interventions cannot make up the deficit), respectively at birth, at 12 months (brain development), at 3 years (height and mental development) and adolescence (behaviour).

Studying the influence of nutritional status in early life, notably in early childhood, on age at menarche involves analyses of longitudinal data. To our knowledge, there have been only few of such longitudinal studies conducted in developing countries, for instance in Guatemala (Martorell and Scrimshaw 1995; Stein et al. 2003), in Gambia (Ceesay et al. 1997; Moore et al. 2001), and in Indonesia (Kusin and Kardjati 1994; Alisjabana and Kusin 2003).

Aim, data and methods

This study was carried out among 12 to 16-year old adolescent girls in Matlab, a rural area Bangladesh. We aim to study their age at menarche in relation to contemporary and early childhood (i.e. up to the age of five years) nutritional status, and recalled age of their mothers. More specifically, we aim to study whether the expected age at menarche can be predicted on the basis of a given nutritional status profile in adolescence and/or early life, i.e. in early childhood and at birth. Timing of menarche and its nutritional determinants is studied by means of lifetable techniques and the Cox regression model. Here we account for censored cases, i.e. girls who were still premenarcheal at the time of interview. The study is the result of a collaboration between the Centre for Health and Population Research (ICDDR,B) in Bangladesh, the Netherlands Interdisciplinary Demographic Institute (NIDI) in the Hague and the Population Research Centre (PRC) of Groningen University in Groningen, the Netherlands. This paper is part of a broader PhD-research on adolescents' reproductive health in rural Bangladesh that has recently been concluded (Bosch 2005).

For nearly four decades the ICDDR,B has been carrying out research in Matlab. The area is known for its rich research history, reflected in the elaborate collection of data. Virtually all inhabitants of Matlab are enrolled in a Health and Demographic Surveillance System (HDSS), maintained by ICDDR,B. The basis of HDSS is a so-called Registration IDentification (RID) number. Our study involves the follow-up of 707 under-five children who were enrolled in an earlier research on persistent diarrhoea, conducted in Matlab in 1988-1989, by Baqui, a pediatrician affiliated with ICDDR,B (Baqui 1990; Baqui et al. 1992; 1993a; 1993b). Linking data about nutritional status in early childhood to data about menarche and nutritional status in adolescence was feasible only because of the aforementioned RID number. At the start of the baseline study, April 1988, the youngest child enrolled was less than one month old, whereas the oldest child was almost five years old. The under-five children have been measured a variable number of times, with a maximum of 14, within an approximate two-year period. We indicated early childhood anthropometry by an average figure based on the available number of measurements (1 to 14). The follow-up study took place in 2001. By that year (most of) the under-fives had grown up to be adolescents, aged 12 to 16 years. We succeeded in surveying the majority (569) of the adolescents, among which including 255 girls.

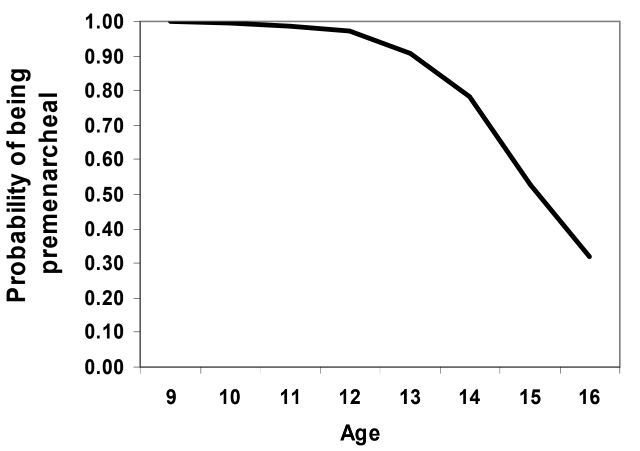
Data on menarche were collected by retrospective recall among the adolescent girls. The recall method of reporting age at menarche may not be optimal, but is usually the only source of available information (Graham et al. 1999, p. 259). Accuracy of short-term recall among adolescent girls was relatively high in a study of Koo and Rohan (1997, pp. 61-64), where 66 per cent was able to recall the age at menarche correctly. In addition, we asked every adolescent girl's mother in retrospect to recall her age when she menstruated for the first time. An event such as the first menstruation is usually not easily forgotten, but having to recall an event over a long time is far from easy, although some - relatively old - studies of Damon et al. (1969) and Livson and McNeill (1962), referred to by Becker (1993), point out that recall errors are fairly random, i.e. show "no systematic bias in retrospective reports". Nevertheless, extra caution while analysing these data is needed.

Nutritional status in early childhood and in adolescence is in this study assessed on the basis of international standards of anthropometric indices (combinations of measurements) such as weight-for-age (undernutrition), height-for-age (stunting), mid-upper arm circumference (MUAC) and body mass index (BMI) that indicates thinness (WHO 1995). Nutritional status is analyzed by using the nutritional anthropometry program 'NutStat' of EPI info 2000 (version 1.1.2; CDC 2000). Two different reference populations of the United States Centre for Health Statistics (US NCHS) were applied: the CDC/WHO reference population of 1978 to the nutritional analyses of the under-fives in 1988-1989 and the CDC reference population of 2000 to the nutritional analyses of the same population in adolescence in 2001. The rationale for this selection was that these two reference populations are as close as possible in time to the year of measurement of anthropometry of the study population in childhood and adolescence respectively. Details about the collection of data and analysis have been reported on elsewhere (Bosch 2005).

Results

Among the 255 12 to 16-year-old girls in our sample many had not yet reached menarche: the proportion of *postmenarcheal* girls increased from 7 per cent among 12-year-old girls to 81 per cent among 16-year-old girls. Over half, 52 per cent, of the *postmenarcheal* girls reached menarche at an age of 14 years or older, the cut-off point of what could be defined as a 'late' menarche when compared with contemporary Western countries. In addition, also 52 per cent of the *premenarcheal* girls was 14 years or older and therefore, according to this definition, 'late' with the reaching of menarche. This latter figure can be regarded as a lower limit as it may increase if *premenarcheal* girls who are currently 12 or 13 years old do not reach menarche before their 14th birthday. We found neither a significant correlation between a mother's age at menarche and menarche status of her adolescent daughter (all girls included), nor a significant correlation between a mother's age at menarche and the age at menarche of her *postmenarcheal* daughter. *Lifetable* analyses, which allow for censoring, revealed that the (expected) median age at menarche among the girls in our sample is 15.1 years (Figure 1).

Figure 1: Survival funtion for adolescent girls showing the probabilty of being premenarcheal by age (in years), Matlab 2001



Reaching menarche at the age of 14 years or older may be detrimental for reproductive health *if* such a relatively 'late' menarche is followed shortly by the birth of the first child and the girl's height is low. This notion is grounded in the recognition that a) height and pelvic size are correlated; and b) at the time of reaching menarche girls have approximately 4 per cent more height and 12 to 18 per cent more pelvic growth ahead of them (WHO 1991, p. 6). In our sample are 31 and 46 per cent of the adolescent girls *moderately* (between -3 and -2 SD) and *severely* (<-3 SD) underweight in comparison to a well-nourished reference population of the same age and sex (CDC 2000). The corresponding proportions of stunting are 40 per cent (*moderate*) and 28 per cent (*severe*) respectively. In absolute terms, some of the girls in our sample are at risk because their weight and height fall below the cut-off points below which obstetric risks increase (for a height less than 145 cm and a weight lower than 45 kg there may be an obstetric risk; WHO 2003, pp. 22-23). The adolescent girls in our sample are likely not to have completed growth but their height and weight seem also to be sub-normal because of malnutrition. *If* the 16-year-old girls in our sample married and became pregnant soon afterwards, 83 and 23 per cent respectively would be at risk in terms of obstetric cut-off points for weight and height.

By means of descriptive analyses, lifetables and Cox regression models, we reviewed further a) the stage in life (adolescence, early childhood or birth) that is most important with respect to the influence of nutritional status on menarche attainment; and b) within this stage, the type of nutritional status indicator (underweight, stunting or BMI) that has the strongest effect. In the univariate model we observed that every contemporary and early childhood nutritional indicator that we included in the analyses had a significant effect on the rate of menarche when considered separately. This also reflects the extent of *collinearity* between the independent variables. Illustrating the effect of contemporary stunting on menarche attainment yielded the followed results: *severely* (<-3 SD) and *moderately* (between -3 and -2 SD) stunted adolescent girls have a rate of menarche that is respectively 16 and 42 per cent of the rate for girls who are *not stunted* (>-2 SD) in adolescence (the reference group). This means that girls who are *severely* stunted in adolescence have the highest age at menarche. Significant effects on the rate of menarche were also found for *severely* underweight and stunting in early childhood.

After controlling for the respective early childhood predictors (i.e. childhood underweight, childhood stunting and recalled birth weight), it appeared that adolescent stunting stood out as the most important (significant) determinant of age at menarche (Table 1).

Table 1 **Cox regression multivariate model predicting the rate of menarche, Matlab 1988-2001**

Variables	Categories	Model 1 (B)	Model 2 (B)	Model 3 (B)	Model 4 (B)	Model 5 (B)
Adolescent underweight	not underweight: >-2 SD (ref.)	-	-	-	-	
	moderately underweight: between -3 and -2 SD	-0.265	-0.414	-0.383	-0.059	
	severely underweight: <-3 SD	-0.808	**-1.107***	**-1.028***	-0.602	
Adolescent stunting	not stunted: >-2 SD (ref.)	-	-	-	-	-
	moderately stunted: between -3 and -2 SD	**-0.727****	**-0.746****	**-0.884****	**-1.103****	**-1.440*****
	severely stunted: <-3 SD	**-1.132****	**-1.208****	**-1.504****	**-1.590***	**-2.893*****
Adolescent CED according to BMI Z-scores	not CED: >-2 SD (ref.)	-	-	-	-	
	moderately CED: between -3 and - 2 SD	-0.290	-0.238	-0.241	0.125	
	severely CED: <-3 SD	-1.057	-0.977	-1.034	-1.708	
Childhood underweight	not underweight: >-2 SD (ref.)		-			-
	moderately underweight: between -3 and -2 SD		0.215			0.106
	severely underweight: <-3 SD		0.511			0.593
Childhood stunting	not stunted: >-2 SD (ref.)			-		-
	moderately stunted: between -3 and -2 SD			-0.061		-0.151
	severely stunted: <-3 SD			0.584		0.499
Recalled birth weight	> 2000 grams (ref.)				-	-
	<= 2000 grams				-0.704	-0.771
-2 Log Likelihood		773.854	771.796	769.149	296.764	300.168
Overall score Chi-square		**58.985*****	**61.116*****	**61.678*****	**35.938*****	**33.450*****
df		6	8	8	7	7
Change from previous step (Chi-square)		**57.570*****	**59.175*****	**61.822*****	**37.430*****	**34.026*****

* Significant at the 0.05 level
** Significant at the 0.01 level
*** Significant at the 0.001 level

However, we believe that we cannot conclude that *only* adolescent stunting impacts on menarche attainment and that adolescent weight (or weight-for-age) and nutritional status in childhood are not factors of importance. Given that in extreme situations (famine, diet, physical exercise) menstruation (temporarily) stops (Napieralski and Devine 1998, p. 3) there seems to be a minimum of nutritional intake for reproductive functioning. Such a minimum - or to use Frisch and Revelle's term (1971), "critical weight" - is also likely to be present for the first menstruation, menarche. Given the strong correlation that we found between stunting in early childhood and adolescence, adolescent stunting still resonates from the effect of stunting in early childhood. Following this line of thinking, the effects of nutritional status of the previous generation (the adolescent girls' mothers) should also not be ruled out as a factor possibly (indirectly) influencing age at menarche. Maternal height did not have a significant effect on the rate of menarche, but it appeared to be an important determinant of adolescent stunting. The results indicated that adolescent girls who were malnourished according to anthropometry as an under-five child are more likely to reach menarche 'late' (i.e. 14 years or older when this event occurs) as compared to their well-nourished counterparts.

Concluding notes

As stated by Napieralski and Devine (1998, p. 4), "age at menarche is beyond our control". Given our results, however, this remains to be seen. In our study we conclude that menarche is indeed reached 'late' for a considerable proportion of the girls due to low contemporary nutritional status, particularly indicated by *severely* stunting, which was in turn associated with malnutrition in early childhood. A girl who finds herself on the threshold of adolescence may indeed not be able to alter the timing of her coming menarche. However, for malnourished girls in early childhood (particularly in infancy), but possibly also in late childhood, i.e. between the ages of 6 to 12 years (a population under-addressed in contemporary research), it may still be feasible to improve nutritional status and catch up part of the deficit of early childhood growth. On the basis of stunting profiles in particular, monitoring systems should be formulated, aiming at singling out those pre-adolescent girls who run a risk of reaching their menarche 'late' so as to programme urgent nutritional aid for them. It is worth noting that maintenance of proper nutritional status from birth onwards also adds to mental well-being in later life (see, for instance, Lachance 1995; Gillespie and Flores 2000).

The significance of a life course study on timing of menarche within the discipline of demography relates also to future transitions embedded within the reproductive career. A comparison of the data on age at *menarche* with data published in other sources on age at *first birth* in Bangladesh - 63 per cent of the girls in this country has given birth by the age of 20 years (Population Reference Bureau 2000, p. 21) - yields a relatively small time gap between these two events. Also, apparently irrespective of their social and cultural diversity, adolescents start sexual activity at about the same age in both developed and developing countries (UNFPA 1997). It is as yet not known exactly how a pregnancy in an adolescent girl interferes with her own growth and reproductive maturation process (Riley et al. 1993). There are indications that catch up early life growth faltering in adolescent girls is characterised by a growth that continues longer than usual, while growth velocity does not change (Riley 1994; Silventoinen 2000). This may have serious implications for adolescent girls' reproductive health, because then reproductive organs (for example, the pelvis) take longer to reach maturity (height is correlated to pelvic size; WHO 1991). Consequently, these girls would typically reach physical 'readiness' for childbirth also at a later stage. These observations have implications for *gynaecological* age, the time in years since menarche (Becker 1993). Young gynaecological age (immaturity of the young mother-to-be) may thus not only jeopardize the course and outcome of the pregnancy, but it is likely to increase the risk of obstructive labor as well, endangering both the life of the adolescent mother and that of her baby. Gynaecological and biological age (indexed by nutritional status) rather than chronological age seem more important to an adolescent girl's reproductive health status. More life course research is required in order to verify the validity of such a hypothesis.

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References

Barker, D.J.P. (ed.). 1992. *Fetal and infant origins of adult disease*. Papers written by The Medical Research Council Environmental Unite. University of Southampton; British Medical Journal.

Barker, D.J.P. 1993. *Fetal origins of adult disease division*. University of Southampton, School of Medicine. Document posted on http://www.som.soton.ac.uk/research/foad/Barker/asp.

Alisjahbana, A. and J.A. Kusin. 2003. The intergenerational cycle of low birthweight: a prospective study of the Tanjungsari cohort 1988-1989. Draft proposal.

Baqui, A.H. 1990. *Epidemiology of persistent diarrhoea in Bangladeshi children*. PhD thesis. Baltimore: Johns Hopkins University.

Baqui, A.H., Black, R.E., Sack, R.B., Yunus, M.D., Siddique, A.K. and H.R. Chowdhury. 1992. “Epidemiological and clinical characteristics of acute and persistent diarrhoea in rural Bangladeshi children”. *Acta Paediatr* (suppl) 381: 1-7.

Baqui, A.H., Black, R.E., Sack, R.B., Chowdhury, H.R., Yunus, M., and A.K. Siddique 1993a. “Malnutrition, cell-mediated immune deficiency, and diarrhoea: a community-based longitudinal study in rural Bangladeshi children”. *American Journal of Epidemiology* 137(3): 355-365.

Baqui, A.H., Sack, R.B., Black, R.E., Chowdhury, H.R., Yunus, M. and A.K. Siddeque. 1993b. “Cell-mediated immune deficiency and malnutrition are independent risk factors for persistent diarrhoea in Bangladeshi children”. *American Journal of Clinical Nutrition* 58: 543-548.

Barker, D.J.P. 1998. *Mothers, babies and disease in later life*. London: British Medical Journal Publishing Group.

Barker, D.J.P., Forsén, T., Uutela, A., Osmond, C. and J.G. Eriksson. 2001. “Size at birth and resilience to effects of poor living conditions in adult life: longitudinal studies”. British Medical Journal 323 (7324): 1273

Becker, S. 1993. “The determinants of adolescent fertility with special reference to biological variables”. In: Gray, R., Leridon, H. and A. Spira. *Biomedical Determinants of Reproduction.* Oxford: Clarendon Press, pp. 21-49.

Ben-Shlomo, Y. and D. Kuh. 2002. “A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives”. *International Journal of Epidemiology* 31: 283-293.

Berger, R.L. 1996. “Human behaviour throughout the life course”. In: Berger, R.L., McBran, J.T. and M.J. Rifxis. *Human behaviour: a perspective for helping X.* New York: Longman Publishers, pp. 129-169.

Bosch, A. 2005. *Adolescents' reproductive health in rural Bangladesh: the impact of early childhood nutritional anthropometry*. PhD thesis. Groningen: University of Groningen. Dutch University Press.

Centers for Disease Control and Prevention (CDC). 2000. *Epi Info 2000. NutStat, a nutritional anthropometry program*. Atlanta (Georgia): CDC, pp. 79-91.

Ceesay, S.M., Prentice, A.M., Cole, T.J., Foord, F., Weaver, L.T., Poskitt, E.M.E., Weaver, L.T. and R.G. Whitehead. 1997. “Effects on birth weight and perinatal mortality of maternal dietary supplements in rural Gambia: 5 years randomised controlled trial”. *British Medical Journal* 315: 786-790.

Cole, T.J. 2000. “Secular trends in growth”. *Proceedings of the Nutrition Society* 59: 317-324

Delgado, H.L., Hurtado, E. and V. Valverde. 1985. “Physical growth, age at menarche and age at first union in rural Guatemala”. *Ecology of Food and Nutrition* 16: 127-133.

Eriksson, J.G., Forsén, T., Tuomilehto, J., Winter, P.D., Osmond, C. and D.J.P. Barker. 1999. “Catch-up growth in childhood and death from coronary heart disease: longitudinal study”. *British Medical Journal* 318 (13 February): 427-431.

Frisch, R.E. and R. Revelle. 1969. “Variation in body weights and the age of the adolescent growth spurt among Latin American and Asian populations, in relation to calorie supplies”. *Human Biology* 41: 185-212.

Frisch, R.E. and R. Revelle. 1971. “The height and weight of girls and boys at the time of initiation of the adolescent growth spurt in height and weight and the relation to menarche”. *Human Biology* 43: 140-159.

Gillespie, S. and R. Flores. 2000. *1999-2000 Annual report. Eradicating malnutrition*. Document posted on http://www.ifpri.cgiar.org/pubs/books.

Graber, J.A., Brooks-Gunn, J. and M.P. Warren. 1995. “The antecedents of menarche age: heredity, family environment, and stressful life events”. *Child Development* 66: 346-359.

Graham, M.J., Larsen, U. and X. Xiping. 1999. “Secular trend in age at menarche in China: a case study of two rural counties in Anhui province”. *Journal of Biosocial Sciences* 31(2): 257-267.

Gray, R. 1993. “Biological factors other than nutrition and lactation which may influence natural fertility: a review”. In: Gray, R., Leridon, H. and A. Spira. *Biomedical Determinants of Reproduction.* Oxford: Clarendon Press, pp. 219-250.

ICDDR,B. 2002. *Annual Report 2001*. Dhaka: Centre for Health and Population Research.

Jenniskens, F. and K. Verduin. 1998. *Background information on developments since Cairo 1994*. Draft. Amsterdam: Royal Tropical Institute (KIT); Dutch Ministry of Foreign Affairs.

Koo, M.M. and R.E. Rohan. 1997. “Accuracy of short-term recall of age at menarche”. *Annals of Human Biology* 24(1): 61-64.

Koprowski, C., Ross, R.K., Mack, W.J., Henderson, B.E. and L. Bernstein. 1999. “Diet, body size and menarche in a multiethnic cohort”. *British Journal of Cancer* 79(April): 1907-1911.

Kuh, D. and Y. Ben-Shlomo (eds.). 1997. *A life course approach to chronic disease epidemiology*. Oxford: Oxford University Press.

Kuh, D. and R. Hardy (eds.). 2002. *A life course approach to women’s health*. Oxford: Oxford University Press.

Kuh, D., Ben-Shlomo, Y., Lynch, L., Hallqvist, J. and C. Power. 2003. “Life course epidemiology: glossary”. *Journal of Epidemiology and Community Health* 57: 778-783.

Kurdzielewicz, M., Rzepka-Gorska, I., Blogowska, A. and B. Kosmowska. 1999. “Age at menarche and the investigation of selected physical parameters in the course of menarche”. *Ginekol Pol* May 70(5): 345-348.

Kusin, J.A. and Sri Kardjati (eds.). 1994. *Maternal and child nutrition in Madura*, *Indonesia*. Amsterdam: Royal Tropical Institute (KIT).

Lachance, P.A. 1995. “Recommended dietary allowance for growth, development and performance”. *Asia Pacific Journal of Clinical Nutrition*. Supplement 1 to volume 4: 7-12.

Linhares, E.D.R., Round, J.J. and D.A. Jones. 1986. “Growth, bone maturation and biochemical changes in Brazilian children from two different socioeconomic groups”. *American Journal Clinical Nutrition* 44: 552-558.

Maclure, M., Travis L.B., Willet, W. and B. MacMahon. 1991. “A prospective cohort study on nutrient intake and age at menarche”. *American Journal of Clinical Nutrition* 54: 649-656.

Martorell, R. and N.S. Scrimshaw (eds.). 1995. “The effects of improved nutrition in early childhood: the institute of nutrition of Central America and Panama (INCAP) follow-up study”. *Journal of Nutrition*. Supplement to volume 25(4s): 1027-1138.

Martorell, R., Schroeder, D.G, Rivera, J. and H.J. Kaplopwitz. 1995. “Patterns of linear growth in rural Guatemalan adolescents and children. Supplement: the INCAP Follow-up study”. *Journal of Nutrition*. Supplement to volume 25(4s): 1060-1067.

Moore, S.E., Collinson, A.C. and A.M. Prentice. 2001. “Immune function in rural Gambian children is not related to season of birth, birth size, or maternal supplementation status”. *American Journal of Clinical Nutrition* 74(6): 840-847.

Napieralski, J.A. and C.M. Devine. 1998. *Childhood life events and the risk of breast cancer.* Fact sheet number 8. New York: Cornwell University.

Pojda, J. and L. Kelley. 2000. Low birthweight: a report based on the International low birthweight symposium and workshop held on 14-17 June 1999 at the ICDDR,B. ACC/SCN Nutrition Policy Paper 18. Geneva: World Health Organisation.

Population Reference Bureau. 2000. *The world’s youth 2000: data sheet*. Washington DC: Measure Communication.

Rich-Edwards, J. 2002. “A lifecourse approach to women’s reproductive health”. In: Kuh, D. and R. Hardy (eds.). *A life course approach to women’s health*. Oxford: Oxford University Press, pp. 23-43.

Riley, A.P. 1994. “Determinants of adolescent fertility and its consequences for maternal health, with special reference to rural Bangladesh”. *Annals New York Academy of Sciences* 18(709): 86-100.

Riley, A.P., Samuelson, J.L. and S.L. Huffman. 1993. “The relationship of age at menarche and fertility in undernourished adolescents”. In: Gray, R., Leridon, H. and A. Spira (eds.). *Biomedical Determinants of Reproduction*. Oxford: Clarendon Press, pp. 50-61.

Robinson, J.R. 1992. “Introduction”. In: Barker, D.J.P. (ed.). *Fetal and infant origins of adult disease*. University of Southampton. British Medical Journal, pp. 1-20.

Ross, J.L., Chowdhury, S.N.M. and T. Mirza. 1996. *Health, gender, sexuality. Bangladesh country report*. Dhaka: Centre for Health and Population Research.

Silva, dos Santos I., Stavola, de B.L., Mann, V., Kuh, D., Hardy, R. and W.E.J. Wadsworth. 2003. “Prenatal factors, childhood growth trajectories and age at menarche”. *International Journal of Epidemiology* 31: 405-412.

Silventoinen, K. 2000. *Body-height: determinants and associations with social position and adult health*. PhD thesis. Helsinki: University of Helsinki.

Stein, A.D., Barnhart, H.X., Hickey, M., Ramakrishnan, U., Schroeder, D.G. and R. Martorell. 2003. “Prospective study of protein-energy supplementation early in life and of growth of subsequent generation in Guatemala”. *American Journal of Clinical Nutrition* 78: 162-167.

Trussell, J. 1980. “Statistical flaws in evidence for the Frisch hypothesis that fatness triggers menarche”. *Human Biology* 52(4): 711-720.

UNFPA. 1997. *UNFPA and Adolescents*. New York: United Nations Population Fund.

WHO. 1995. “Adolescents”. In: WHO *Physical status: the use and interpretation of anthropometry*. Report of a WHO expert Committee. WHO Technical Report Series 854. Geneva: World Health Organisation, pp. 263-311.

WHO. 2003. *Adolescent nutrition in South-East Asia: a review of the current situation*. Draft. Geneva: World Health Organisation; New Delhi: Regional Office of South-East Asia.