

# Symptom development and timing of menarche: a longitudinal study.

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**ABSTRACT** *Self-reported somatic complaints among females were studied cross sectionally at age 15 and 43, and longitudinally between these two ages. Specifically, the relationship between symptom development and timing of menarche was considered. The sample consisted of 477 females representing the general Swedish population. All the included symptoms were significantly related to each other at both ages. There was also longitudinal correlational stability for all the studied symptoms. Moreover, symptoms at age 15 were associated with early menarcheal timing. Pattern analyses of the studied symptoms were conducted with roots in person-oriented methodology. These results revealed structural as well as individual stability in patterns of symptom reporting for nearly 30 years. That is, similar configurations of symptoms were found at both ages, and it was the same females who reported being either symptom free or reporting a high symptom load at both ages. At age 15 fewer early maturing females than expected by chance reported being symptom free, whereas more of the late maturing females than expected reported being symptom free. However, there were no effects of menarcheal timing on symptom reporting at age 43.*

**Key words:** biological maturation, somatic complaints, methodology, females

## Introduction

Adolescence is a period of change in several areas: cognitive, social, emotional, as well as physical (Forman, 1993). During the process of adaptation to new challenges and demands at this time of development, there is an increase in the amount of stress put upon the individual (Seiffge-Krenke, 1995), which has an impact on health (Simmons, Burgeson and Carlon-Ford, 1987). Such a health effect might in turn have long-lasting consequences that affect adult health status (Bardone, Moffitt, Caspi, Dickson, Stanton and Silva, 1998; Robins and Price, 1991), which suggests possible stability between adolescent symptomatology and later health.

In this context, timing of transition into adolescence, referred to as pubertal timing or biological maturation (Stattin and Magnusson, 1990; Petersen, Sirigiani and Kennedy, 1991; Rierdan and Koff, 1991; Susman, Dorn and Chrousos, 1991; Caspi, Lynam, Moffitt and Silva, 1993; Ge, Conger and Elder, 1996; Graber and Brooks-Gunn, 1996; Graber,

Lewinsohn, Seeley and Brooks-Gunn, 1997; Ge, Conger and Elder, 2001; Stice, Presnell and Bearman, 2001) is believed to be an important factor, because it is presumed to influence how different individuals experience or negotiate with such transition (Rutter, 1989; Graber and Brooks-Gunn, 1996). Experiencing transition earlier than others might be a potential risk factor for non-successful adaptation, as the adolescent has less time than most others to develop the skills needed for an appropriate way of handling the new situations and corresponding situational demands with which she or he is faced (Stattin and Magnusson, 1990; Graber and Brooks-Gunn, 1996).

The above has been referred to as the early timing hypothesis (Petersen and Taylor, 1980), which states that, despite the same chronological age, differences in pubertal timing may be more influential in not only social but also emotional development and adaptation than transition to puberty per se (Petersen and Taylor, 1985; Buchanan, Eccles and Becker, 1992).

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Previous research has confirmed this hypothesis, showing that pubertal timing is of importance for adaptation during adolescence. Various studies have shown that the risk associated with early pubertal timing is especially pronounced among females. Early maturing girls have been found to report elevated levels of internalizing, mental symptoms and psychosomatic complaints and also more externalizing problem behaviours in adolescence than girls with on-time or late pubertal timing (Aro and Taipale, 1987; Stattin and Magnusson, 1990; Petersen et al., 1991; Rierdan and Koff, 1991; Susman et al., 1991; Caspi et al., 1993; Bergman and Wångby, 1995; Ge et al., 1996; Graber et al., 1997; Ge et al., 2001; Stice et al., 2001).

Although much is known about the effects of early pubertal timing on girls in adolescence there are only a few studies of possible long-term effects into adulthood. Moreover, previous studies have mainly concentrated on psychosocial factors, and those have found early maturing females to have lower educational and occupational status (Stattin and Magnusson, 1990), to have more children, and to have had their first child earlier than on-time and late maturing girls (Udry and Cliquet, 1982; Stattin and Magnusson, 1990; Manlove, 1997).

Turning to the existing body of literature on pubertal timing in relation to symptom reporting, much is also known about its effects throughout the adolescent years. However, few studies have followed subjects long enough to evaluate the persistence of pubertal timing effects on health in terms of symptom reporting. Thus, there is an important gap in knowledge with respect to transition to puberty and subsequent health development.

Adolescent females with a high initial symptom load might be more susceptible to subsequent symptoms as the experience of symptoms early in life is presumed to lead to a developmental trajectory marked by high symptom levels (Petersen et al., 1991; Susman et al., 1991). This is supported in a study by Wångby (1997), conducted on the same sample as the present study, showing longitudinal stability in somatic complaints among girls between ages 10 and 16. From the above follows that individual stability in symptom reporting over time could be expected. That is, girls with a high symptom load in adolescence would also report a high frequency of symptoms in middle age, whereas girls with a low

symptom load in adolescence would still report low frequencies in middle age. Moreover, it could be expected that early maturing females in particular might be at a heightened risk for a subsequent poor health development since they report higher levels of various symptoms in adolescence than on-time and late maturing females.

In order to study individual differences in the development of symptom reporting as related to timing of puberty, the person-oriented approach (see, for example, Bergman and Magnusson, 1997) was adopted, involving simultaneous analysis of some of the most commonly reported somatic complaints in both adolescence and middle age at the level of the individual. It follows from the assumptions underlying this approach that combined information on several simultaneously operating factors at the level of the individual is essential in order to understand and explain individual development and functioning (Magnusson, 1988). Further, only a limited number of frequent value patterns of the operating factors under study are expected to occur; finding these typical configurations of values is important because they are assumed to characterize the system under study at specific points in time. Longitudinally, typical configurations define the trajectories that individuals take, which in turn might be studied in terms of stability and change over time (for example, El-Khoury, 2001). For detailed discussions and examples of the use of the person-oriented approach, see Bergman, Magnusson and El-Khoury (2003).

The aim of the present study was to investigate the development of somatic symptoms from adolescence to middle age among females as related to pubertal timing, defined as time of menarche. However, as many commonly reported somatic complaints without a medically stated etiology are known to be associated with a depressive symptomatology, it was considered appropriate to study the associations between somatic complaints and indications of depression in separate analyses.

Firstly, it was hypothesized that early maturing females would report elevated symptom levels in adolescence as compared with on-time and late maturing females. In middle age, the effect of menarcheal timing on symptom reporting was expected still to be evident. Moreover, we expected a high level of structural as well as individual stability over time in the symptom profiles obtained. That is, it was

assumed that the same value combinations of symptoms would emerge at both time points, and that it would be the same individuals manifesting with these patterns. Finally, high levels of somatic symptoms at both ages were expected to be associated with indications of depression, as common somatic complaints might be regarded as proxies for general distress.

## Method

### *Sample*

Data were taken from the longitudinal research programme Individual Development and Adaptation (Magnusson, 1988). The programme covered all children who attended the third grade (age 10) in a Swedish representative urban community in 1965.

As pubertal timing was a main focus of investigation it was important that all females were of the same chronological age so that differences in age would not influence the results. Thus, only those females born in 1955 were originally included ( $N = 590$ ). Complete information on all the studied symptoms were obtained for 473 cases. Imputation analysis of at most one missing value resulted in four more cases being added, resulting in a total of 477 females at age 15. These 477 females defined the sample on which residue analysis and consequently cluster analysis were performed.

At age 43, complete data were obtained for 439 females. Imputation analysis for at most one missing value resulted in five more cases being added. Residue analysis, however, resulted in six cases being removed. Thus, 438 females formed the sample on which cluster analysis at age 43 was conducted.

The overlap between the two samples at ages 15 and 43, respectively, consisted of 352 females. Thus, these 352 cases constitute our longitudinal sample. There were no differences in symptom reporting at age 15 between those females who were not included in the analysis at age 43 (due to longitudinal attrition) and those who remained in the sample.

### *Variables*

At age 15, girls were administered a self-report questionnaire addressing timing of menarche and also a symptom check-list. A six-point scale was used to assess the age at which the girls reached menarche (1 = menarche before age 10; 2 = between ages 10–11;

3 = between ages 11–12; 4 = between ages 12–13; 5 = after age 13; 6 = not had first menarche yet). A variable called 'menarcheal timing' was formed. This variable distinguished between early, normal and late menarche. The new variable ranged between 1 = early menarche (before age 12); 2 = normal menarche (between ages 12 and 13); and 3 = late menarche (after age 13). Table 1 shows the distributions of the age 15 and 43 samples with regard to menarcheal timing. Self-reports of age at menarche have been used extensively within the IDA programme (Stattin and Magnusson, 1990; Bergman and Wångby, 1995) and have been shown generally to exhibit high reliability and validity (Caspi and Moffitt, 1991; Shisslak, Renger, Sharpe, Crago, McKnight, Gray, Bryson, Estes, Parnby, Killen and Taylor, 1999).

The instrument for the assessment of the symptoms at age 15 was a checklist covering commonly reported mental as well as physical symptoms in the general adolescent population, although no standardized checklist was used. In the symptom checklist the girls were asked to report how often they had experienced a variety of symptoms during the past year. The selected symptoms were those that were most frequently reported by the age 15 sample, and for which there was corresponding information at age 43. The symptoms included in the person-oriented analyses were headaches, stomach problems, and sleeping difficulties. Moreover, the frequency of sadness was included in order to investigate the possible associations with somatic complaints. Symptom frequency was measured on a five-point scale (1 = never; 2 = about once per semester; 3 = about once a month; 4 = weekly, and 5 = daily).

At age 43, the subjects completed a symptom checklist in which they were asked to report how often they had experienced a variety of symptoms during the past month. The symptoms measured by the checklist included the most common physical and mental symptoms reported by females in the general population (Lindén, 1991; Tibblin, Bengtsson, Furunes and Lapidus, 1991; Krantz and Östergren, 1999). The symptoms selected for the present study were headaches, stomach problems, and sleeping difficulties. Moreover, sadness was also included at age 43. Symptom frequencies were measured on a four-point scale (1 = daily; 2 = several times per week; 3 = only a few times; and 4 = never).

Table 2 shows for all the included symptoms the percentages of women reporting a symptom of any frequency both at ages 15 and 43.

#### Statistical analyses

Person-oriented strategies and methods were primarily used for the study. For a detailed discussion of the rationale behind the person-oriented approach and its methodological implications see, for example, El-Khoury (2001) or Bergman, Magnusson and El-Khoury (2003), where guidelines concerning the strategies employed in the analyses described below are given. Unless otherwise indicated, all of the analyses referred to below were performed using procedures in SLEIPNER 2.0 (Bergman and El-Khoury, 1998).

For this study, cluster analytic techniques were used to identify, at each age separately, homogenous classes of individuals characterized by similar patterns of symptoms. The similarity between two patterns was assessed by computing the averaged squared Euclidean distance between them. Prior to conducting cluster analysis, however, preparatory analyses were performed on each data set separately. Partial dropout was handled by imputing missing values using a 'nearest neighbour' approach, which was also used for the identification and removal of

outliers at each age. For the purpose of finding an age-specific classification of symptoms, Ward's method – a hierarchical agglomerative technique, was used with special attention paid to achieving a well functioning classification (Gordon, 1996; Milligan, 1996; Bergman, 1998; El-Khoury, 2001). For instance, for each age group, the results of cluster analysis were tested against a null hypothesis of no relationships in the data. This was achieved by using a simulation procedure where the cluster analysis was repeated a number of times on a partially artificial data set obtained by modifying the original data randomly while keeping constant marginal frequencies for the included variables.

Structural as well as individual stability and change over time were also studied in the sense of typical patterns, and the movement of individuals between these typical patterns – those defining absence of problems, one problem present, or multi-problem configurations. This was done by cross-tabulating, in a contingency table, the two age-specific samples with respect to the cluster solutions obtained at each age, and assigning a probability value to the difference between the observed and the expected frequencies in each cell of that table.

Next, each of the two age-specific classifications obtained in the cluster analyses were cross-tabulated

**Table 1.** Frequencies and percentages of the 15 and 43 age samples with regard to menarcheal timing

|                 | Age 15 sample | Age 43 sample |
|-----------------|---------------|---------------|
| Early menarche  | 158 (33.2%)   | 118 (33.6%)   |
| Normal menarche | 194 (41.0%)   | 141 (40.0%)   |
| Late menarche   | 123 (25.9%)   | 93 (26.4%)    |
| Total           | 475           | 352           |

**Table 2.** Percentages (RF) of women reporting a symptom of any frequency at ages 15 and 43, and corresponding means (M) and standard deviations (SD)

|    | Age 15 |     |      | Age 43 |     |      |
|----|--------|-----|------|--------|-----|------|
|    | RF     | M   | SD   | RF     | M   | SD   |
| HA | 82%    | 2.8 | 1.06 | 80%    | 2.9 | 0.73 |
| SP | 83%    | 2.6 | 1.07 | 49%    | 3.3 | 0.89 |
| SD | 85%    | 3.0 | 1.18 | 46%    | 3.4 | 0.80 |
| SA | 78%    | 2.4 | 1.05 | 72%    | 3.5 | 0.73 |

Note. HA = headaches, SP = stomach problems, SD = sleeping difficulties, and SA = sadness.

against the menarcheal-timing variable. Thus, at age 15 we were able to explore the possibility of any typical or atypical relationships between the concluded classes of symptoms and menarcheal age, and further, to contrast these with those found for age 43 between menarcheal age and the concluded classes of symptoms for that age. For the exact analysis of single cells in the obtained contingency tables, the EXACON procedure was employed. See also Bergman and El-Khoury (1988).

Finally, we repeated the same procedure as above, exchanging menarcheal timing with the variable sadness, as an indicator of mental symptoms, in order to investigate the possible relationships between the obtained classifications of somatic symptoms and the simultaneous presence of depressive symptomatology.

## Results

### *Relationships between symptoms*

Table 3 shows correlations between all the included symptoms, cross-sectionally as well as longitudinally, and, further, between the symptoms at each age and menarcheal timing. At both ages 15 and 43, all possible relationships between the included symptoms were significant. Longitudinally, correlational stability was also found for a majority of the symptoms – headaches, stomach problems, sleeping difficulties, and sadness. The highest, longitudinal correlational stability was found for sadness, and

between sleeping difficulties at age 15 and sadness at age 43. With regard to menarcheal timing, significant correlations were found for all the studied symptoms at age 15, with early maturing girls showing higher symptom frequencies than on-time or late maturing girls. However, the results did not show any association between symptom reporting at age 43 and menarcheal timing.

### *Profiles of symptoms at ages 15 and 43*

The seven-cluster solution was chosen for the age 15 data set and the six-cluster solution for the age 43 data set. The explained error sum of squares (EESS) for a given solution refers to the percentage variance explained by the given cluster solution, and is defined in Bergman and El-Khoury (1998) as satisfactory when it is 67% or above. The obtained EESS for the data set at age 15 corresponding to the 7-cluster solution was 67%. The corresponding EESS for the data set at age 43 was 71%. Table 4 shows the means for the included variables – centroids – corresponding to these solutions along with the standard deviations of the variables involved at age 15. Also shown in Table 4 are coefficients of homogeneity defined for each cluster as the average within-cluster average squared Euclidean distance. Corresponding details for the age 43 cluster solutions are shown in Table 5. It can be seen from Table 4 and Table 5 that the homogeneity for the single clusters varied between

**Table 3.** Spearman rank order correlations between the studied symptoms and menarcheal timing

|        | Age 15 |        |        |        | Age 43 |        |        |        |        |
|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|
|        | HA     | SP     | SD     | SA     | HA     | SP     | SD     | SA     | MT     |
| Age 15 |        |        |        |        |        |        |        |        |        |
| HA     |        | 0.66** | 0.67** | 0.27** | 0.10*  | 0.11** | 0.12** | 0.09*  | 0.15** |
| SP     |        |        | 0.71** | 0.35** | 0.09*  | 0.12** | 0.11** | 0.08   | 0.33** |
| SD     |        |        |        | 0.35** | 0.05   | 0.09*  | 0.10*  | 0.15** | 0.09*  |
| SA     |        |        |        |        | 0.04   | 0.05   | 0.09*  | 0.18** | 0.03   |
| Age 43 |        |        |        |        |        |        |        |        |        |
| HA     |        |        |        |        |        | 0.76** | 0.76** | 0.27** | 0.02   |
| SP     |        |        |        |        |        |        | 0.77** | 0.31** | 0.01   |
| SD     |        |        |        |        |        |        |        | 0.48** | 0.02   |
| SA     |        |        |        |        |        |        |        |        |        |

Note. Two-tailed significance: \*  $p < 0.05$ ; \*\*  $p < 0.01$ ; HA = headaches, SP = stomach problems, SD = sleeping difficulties, SA = sadness, and MT = menarcheal timing.

0.28 and 1.11 for the age 15 solutions and between 0.00 and 0.96 for the age 43 solutions. Taken together for each age separately, the above indicates that the cluster solutions were satisfactory.

Figures 1 and 2 offer a graphical presentation of the centroids. For these figures the means of the variables – centroids – were z-transformed. To each z-transformed centroid, a constant of 2 was added for the data set at age 15 and a constant of 4 for the data

set at age 43. Thus, the dotted lines in these figures represent standardized mean values for the total sample.

Three single symptom clusters were found: cluster B comprised girls who were characterized by frequent stomach problems ( $n = 97$ , 20%), cluster D girls characterized by frequent sleeping difficulties ( $n = 39$ , 8%), and the girls in cluster G were high in headaches ( $n = 42$ , 9%).

**Table 4.** The centroids, standard deviations, number of cases and homogeneity coefficient in each cluster of the seven-cluster solution at age 15 in terms of the original scale

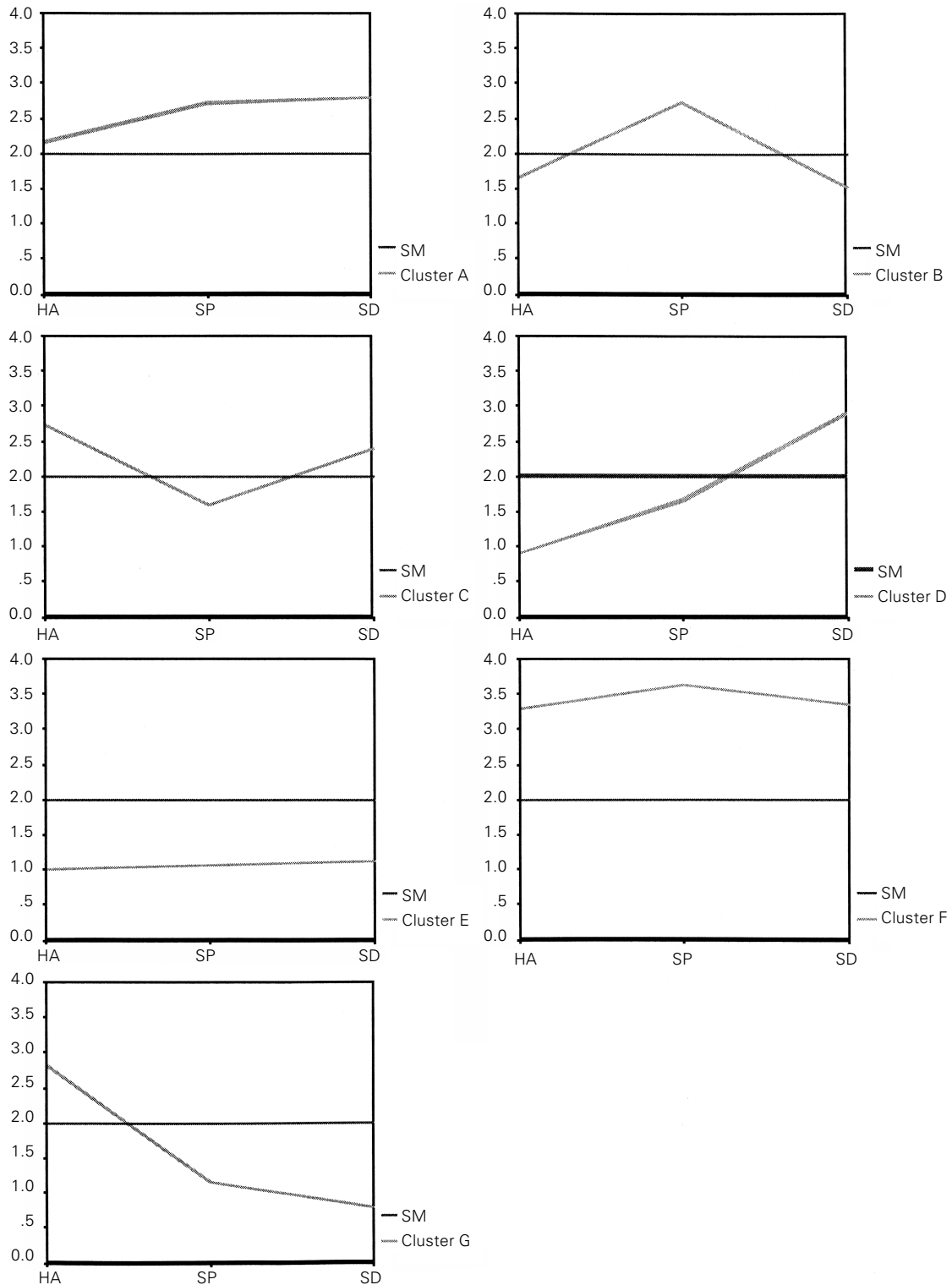
|           | HA           | SP           | SD           | N   | HC   |
|-----------|--------------|--------------|--------------|-----|------|
| Cluster A | 2.93<br>0.25 | 3.31<br>0.46 | 3.17<br>0.38 | 58  | 0.28 |
| Cluster B | 2.40<br>0.86 | 3.34<br>0.57 | 1.89<br>0.56 | 97  | 0.93 |
| Cluster C | 3.55<br>0.69 | 2.08<br>0.61 | 2.77<br>0.73 | 101 | 0.93 |
| Cluster D | 1.61<br>0.49 | 2.17<br>0.60 | 3.28<br>0.55 | 39  | 0.61 |
| Cluster E | 1.70<br>0.46 | 1.53<br>0.76 | 1.49<br>0.87 | 97  | 0.47 |
| Cluster F | 4.13<br>0.55 | 4.27<br>0.76 | 3.74<br>0.87 | 43  | 1.11 |
| Cluster G | 3.64<br>0.65 | 1.64<br>0.65 | 1.14<br>0.35 | 42  | 0.65 |

HA = headaches, SP = stomach problems, and SD = sleeping difficulties.

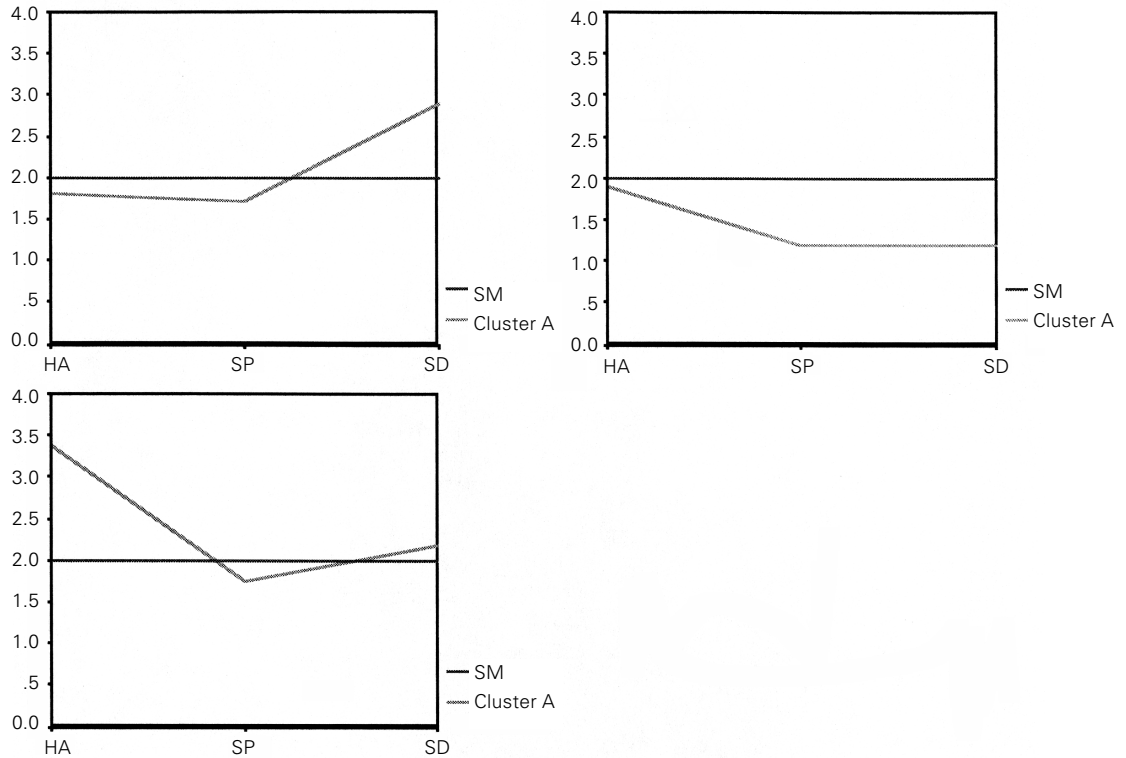
**Table 5.** The centroids, standard deviations, number of cases and homogeneity coefficient in each cluster of the 6-cluster solution at age 43 in terms of the original scale

|           | HA           | SP           | SD           | N  | HC   |
|-----------|--------------|--------------|--------------|----|------|
| Cluster H | 2.70<br>0.72 | 3.43<br>0.49 | 2.36<br>0.82 | 65 | 0.96 |
| Cluster I | 1.00<br>0.00 | 1.21<br>0.41 | 1.15<br>0.36 | 73 | 0.20 |
| Cluster J | 1.90<br>0.29 | 2.26<br>0.44 | 1.00<br>0.00 | 53 | 0.19 |
| Cluster K | 1.93<br>0.24 | 1.49<br>0.50 | 2.34<br>0.59 | 95 | 0.44 |
| Cluster L | 2.00<br>0.00 | 1.00<br>0.00 | 1.00<br>0.00 | 84 | 0.00 |
| Cluster M | 3.07<br>0.26 | 1.52<br>0.50 | 1.77<br>0.72 | 68 | 0.56 |

HA = headaches, SP = stomach problems, and SD = sleeping difficulties.



**Figure 1.** Cluster means (centroids) of the seven-cluster solution based on the age 15 sample. The means are expressed in z-transformed scores based on the total sample at age 15 and to which a constant of 2 was added. Dotted lines represent the standardized mean values in the studied variables for the total sample. HA = headaches, SP = stomach problems, and SD = sleeping difficulties.



**Figure 2.** Cluster means (centroids) of the six-cluster solution based on the age 43 sample. The means are expressed in z-transformed scores based on the total sample at age 43 and to which a constant of 4 was added. Dotted lines represent the standardized mean values in the studied variables for the total sample. At age 15, cluster E consisted of girls who were symptom free ( $n = 97$ , 20%). HA = headaches, SP = stomach problems, and SD = sleeping difficulties.

One cluster with two frequent symptoms was found: cluster C comprised girls who were above average with regard to headaches and sleeping difficulties, but below average on stomach problems ( $n = 101$ , 21%).

Two multi-problem clusters were found: cluster A, which comprised girls who were slightly above average in headaches, and high on stomach problems and sleeping difficulties ( $n = 58$ , 12%); and cluster F which was very high on all symptoms ( $n = 43$ , 9%).

In sum, it was most common to report high frequencies in both headaches and sleeping difficulties, followed by being symptom free or having frequent stomach problems. In addition, it was uncommon to report only sleeping difficulties or only headaches as well as having high frequencies in all the studied symptoms. Thus, among adolescent females, headaches and sleeping difficulties tend to

operate simultaneously at the level of the individual, whereas stomach problems more often emerge as a single symptom.

At age 43, two symptom-free clusters – clusters with symptom levels below average – were found, namely: cluster I ( $n = 73$ , 16%) and cluster L ( $n = 84$ , 19%).

Two clusters were found with one symptom above average but average or below average on the other reported symptoms: females in cluster J reported stomach problems ( $n = 53$ , 12%), and cluster K females ( $n = 95$ , 21%) reported sleeping difficulties.

Cluster M ( $n = 68$ , 15%) was characterized by being high on headaches and slightly above average in sleeping difficulties, but below average in stomach problems. Finally, cluster H ( $n = 65$ , 15%) comprised females reporting very high frequencies in all three symptoms.



Taken together, it was shown that 35% of the study population at age 43 reported being symptom free, followed by reporting high frequencies only in sleeping difficulties.

#### *Structural stability of symptom profiles over time*

The structural stability of the symptom patterns was investigated between ages 15 and 43 by pairwise matchings of the centroids in the studied symptoms from the obtained cluster solutions from both time points. The averaged squared Euclidean distance (ASED) of the centroids at ages 15 and 43, respectively, from the closest cluster at age 15, indicated that four pairs of clusters showed considerable structural stability over a time period of nearly 30 years (ASED varying between 0.05 and 0.34). These were clusters B-J, E-I, F-H, and C-M. Two pairs (D-K and G-L) displayed less structural stability, but were still comparable. Thus, somatic complaints combined together in almost the same manner in these clusters despite age. However, the clusters showed lower symptom problem level over the period of 30 years.

#### *Individual stability of symptom profiles over time*

The stability of the symptom patterns at individual level was investigated between ages 15 and 43 by cross-tabulating the obtained cluster solutions from both time points. Figure 3 presents a graphical illustration of only the significant longitudinal movements between those clusters described by high versus low symptom levels between both ages. The flow between the high symptom clusters as well as between the low symptom clusters between both ages was observed 1.5 and 2.0 more times, respectively than would be expected by chance alone, as illustrated by the arrows. In contrast, the flow between the high and low clusters between both ages was observed 1.8 and 4.7 fewer times, respectively than expected by chance alone, as illustrated by the dotted arrows.

The results showed that more females than expected (hypergeometric probability  $p < 0.05$ ) from the symptom free cluster E at age 15 were found in the symptom free cluster I at age 43 (observed: 18; expected: 12.4,  $p < 0.05$ ). Moreover, more females than expected from the high symptom cluster F at age 15, were found in cluster H at age 43, also described by high frequencies in all the studied symptoms (observed: 9; expected: 4.1,  $p < 0.05$ ). In

contrast, fewer females than expected from the symptom-free cluster E at age 15 were found in the high symptom cluster H at age 43 (observed: 6; expected: 10.7,  $p < 0.05$ ) and fewer females than expected from the high symptom cluster F at age 15 were found in the symptom-free cluster I at age 43 (observed: 1; expected: 4.7,  $p < 0.05$ ).

#### *Symptom profiles and timing of menarche*

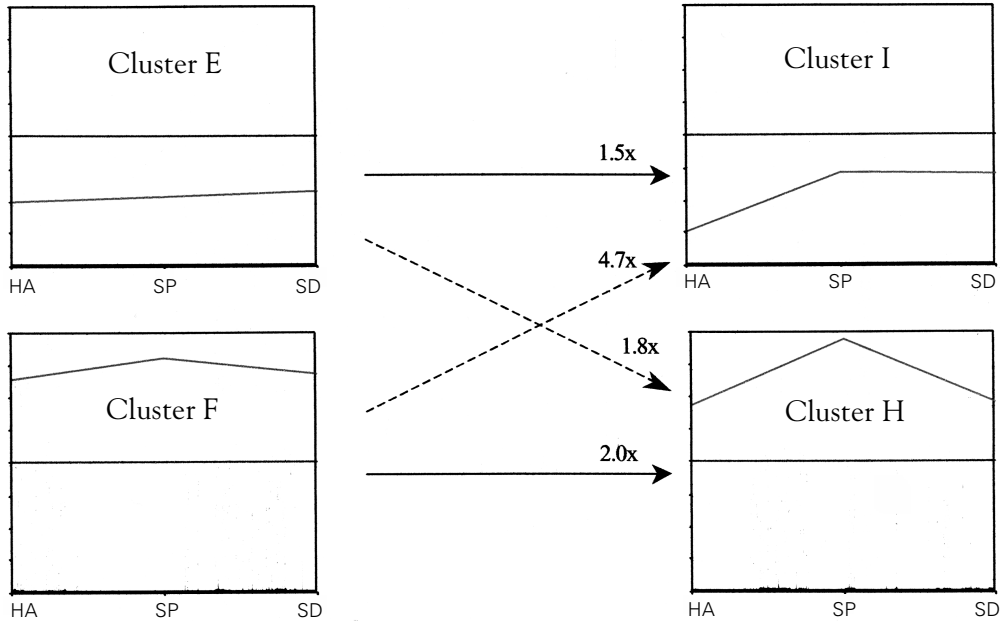
The seven-cluster solution at age 15 and the six-cluster solution at age 43 were each cross-tabulated against timing of menarche.

At age 15, it was confirmed that girls with late menarcheal timing were significantly more frequently found in the symptom-free cluster E (observed: 34; expected: 25.0,  $p < 0.05$ ) than expected by chance. Further, significantly fewer than expected of the late-maturing girls were found in cluster B, which was described by stomach problems (observed: 19; expected 26.0,  $p < 0.05$ ) and in cluster G, described by frequent headaches (observed: 5; expected 10.5,  $p < 0.05$ ). Moreover, significantly fewer of the girls with normal menarcheal timing were found in cluster E, which was the cluster showing the highest symptom levels (observed: 11; expected: 17.4,  $p < 0.05$ ). Finally, the hypothesis that fewer girls with early menarcheal timing than expected would be found in cluster E (observed: 19; expected: 32.6,  $p < 0.000$ ), whereas significantly more than expected of them would be found in cluster F, characterized by very high frequencies in all the studied symptoms (observed: 24; expected: 14.4,  $p < 0.001$ ) was confirmed.

Testing for the same relationships at age 43, no significant associations between symptom patterns and timing of menarche were found.

#### *Symptom profiles and sadness*

When studying the possible associations between the obtained cluster solutions at age 15 and the simultaneous presence of sadness, two clusters were focused upon, namely the symptom-free cluster E, and cluster F, which was described by high frequencies in all the studied symptoms. It was found that significantly more females than expected from cluster E reported not having been sad at any time during the past year (observed: 46; expected: 22.8,  $p < 0.000$ ), whereas fewer of them than expected reported having been sad on a frequent basis during the past year (several times per month: observed: 20; expected: 36.1,  $p < 0.001$ ,



**Figure 3.** Significant longitudinal movements between ages 15 and 43. Numbers attached to the end of the arrows indicate how many times more the flow was observed as compared to what would be expected from chance alone. Numbers attached to the end of the dotted arrows indicate how many times less the flow was observed as compared to what would be expected by chance alone. An exact one-tailed hypergeometric test was used:  $p < 0.05$ . HA = headaches, SP = stomach problems, and SD = sleeping difficulties.

weekly: observed: 3; expected: 10.0,  $p < 0.005$ , daily: observed: 0; expected: 2.8,  $p < 0.05$ ).

When turning to cluster F, comprising girls with high symptom levels, the pattern was reversed. The results revealed significantly fewer females than expected reporting low frequencies of sadness during the past year (not at all: observed, 4; expected: 9.9,  $p < 0.05$ , once or twice: observed: 3; expected: 10.8,  $p < 0.001$ ), whereas significantly more females than expected reported high levels of sadness (weekly: observed: 14; expected: 4.3,  $p < 0.005$ , daily: observed: 4; expected: 1.2,  $p < 0.05$ ).

At age 43, the symptom free Cluster I and Cluster J, as well as Cluster H, showing high symptom frequencies, were focused upon. A similar picture emerged as at age 15.

Significantly fewer females than expected from the symptom free clusters I and L reported having been sad during the past month (cluster I, weekly: observed: 1; expected: 4.3,  $p < 0.05$ , only occasionally: observed: 16; expected: 24.8,  $p < 0.01$ , cluster L, weekly: observed: 1; expected: 4.9,  $p < 0.05$ , only occasionally: observed: 17; expected: 28.5,  $p < 0.005$ ), whereas

significantly more of them than expected reported not having been sad at all (cluster I, observed: 56; expected: 41.8,  $p < 0.000$ ; cluster L, observed: 65; expected: 48.1,  $p < 0.000$ ).

In contrast, significantly more females than expected from the high-symptom cluster H reported high frequencies of sadness during the past month (daily: observed: 8; expected: 1.7,  $p < 0.000$ , several times per week: observed: 14; expected: 3.8,  $p < 0.000$ ), whereas significantly fewer of them reported never having been sad during the past month (observed: 16; expected: 37.2,  $p < 0.000$ ).

#### *Sadness and menarcheal timing*

At age 15, significantly fewer early maturing females than expected reported never having been sad during the past year (observed: 29; expected: 37.7,  $p < 0.05$ ), whereas significantly more females than expected with normal menarcheal timing only had been sad occasionally (observed: 61; expected: 50.2,  $p < 0.05$ ).

At age 43, there were no significant associations between sadness and menarcheal timing.

## Discussion

The purpose of the present study was to investigate, both separately as well as in concert, the development of commonly reported somatic symptoms in the general female population from adolescence to middle age, and in relation to timing of menarche.

The results of the variable-oriented analyses showed that all of the included symptoms were significantly related to each other at each age. Further, significant correlational, longitudinal stability was found for a majority of the studied symptoms between ages 15 and 43. Moreover, there were significant associations between all the included symptoms in adolescence and early menarcheal timing. However, no associations remained between menarcheal timing and symptoms at age 43. Longitudinal stability in somatic complaints was also found by Wångby (1997) when studying some of the same symptoms among females within the IDA program as in the present study. In that study, there was significant, although low, correlational stability in symptoms between ages 10, 13, 15, and 16. The highest stability was found between ages 15 and 16. However, the person-oriented analyses of patterns of symptoms in that study only showed stability between ages 15 and 16. One of the questions to which this gives rise is whether these results are accounted for by the short time span covered, or whether they reflect increasing stability with age.

From the results of the person-oriented analyses in the present study it can be concluded that there is significant individual stability over time in symptom reporting. Thus, not only is there significant correlational, longitudinal stability based on the total sample mean in the studied symptoms but, even more importantly, it is the same individuals presenting with either high or low frequencies of symptoms for over nearly 30 years. This supports the notion of increasing symptom stability with age. Moreover, there was also structural stability over time – that is, the same configurations of symptoms emerged at both time points, supporting the hypothesis that there is a limited number of frequent value combinations of operating factors within a given system, and these describe the trajectories that individuals take (see, for example, El-Khoury, 2001). This was represented in the present study by the findings that about the same number of homogenous clusters were obtained at both time points, and moreover, that

these were similar in the value profiles that emerged.

When analysing the cluster solutions in relation to timing of menarche it was found that, in adolescence, girls characterized by a value profile of low frequencies in all the included symptoms to a greater extent than expected belonged to the late maturing group, whereas fewer girls with early menarche than expected appeared in the clusters described by low symptom frequencies. The reversed pattern was also found in that girls with a profile of high symptom frequencies to a higher extent than expected by chance belonged to the early maturing group, whereas fewer of the late-maturing girls were found in these clusters. However, there were no remaining effects of menarcheal timing on symptom reporting in middle age, either from the variable-oriented analyses or from the person-oriented analyses.

Finally, it was found that sadness, as an indicator of depression, was associated with high frequencies of the studied symptoms at both ages. It was also related with early menarcheal timing in adolescence. However, no association between menarcheal timing and sadness in middle age was found. One possible explanation for these results is that somatic complaints might function as proxies for general distress. In this way, somatic complaints could serve as manifestations of the increased stress put upon adolescents, which is supported by the higher frequency of symptoms reported at this time of development. Moreover, as there was individual stability in symptom reporting for nearly 30 years, indicators of distress in terms of sadness could also represent a more general vulnerability to stress, which in turn could be regarded as a tendency for psychological vulnerability that has been associated with somatic complaints (Vassend, 1987; 1989; Spinhoven, Jochems, Linssen and Bogaards, 1991). Another possible explanation might be that the females presenting with a high symptom load over time might have certain personality characteristics known to be related with self-reported symptoms such as hypochondria and somatization (Barsky, Cleary and Klerman, 1992), neuroticism (Costa and McCrae, 1985) and negative affectivity (Watson and Pennebaker, 1989).

Taken together, the results of the present study support the early timing hypothesis in relation to health (Petersen and Taylor, 1985). Thus, the early maturing girls can be regarded as more psychosocially

vulnerable in adolescence resulting in higher symptom frequencies during this period of development, whereas the late-maturing girls have lower symptom frequencies. These results are in line with those of a study by Ge et al. (2001) who found a protective effect of late menarche on internalizing symptoms in adolescence. However, the generally quite high symptom frequencies reported in adolescence, in particular by early maturing females, seem to be of a temporary nature. Frequencies level off as the females grow older, possibly due to them learning the skills necessary for successful adaptation. This line of reasoning is supported in a study by Herman-Stahl and Petersen (1996) who found that poor coping skills are associated with symptomatology among adolescents. Another study showed that early maturing girls were at the highest risk of developing internalizing disorders during high school, whereas the effects of pubertal timing on symptoms had weakened by the age of 21 (Hayward, Killen, Wilson, Hammer, Litt, Kraemer, Haydel, Varady and Taylor, 1987).

One limitation of the present study is that we did not have measures based on medical examinations. Therefore, some of the symptoms reported might have been due to actual medical conditions, meaning that some of the females might have had poorer health than others. However, the symptoms included in this study are known to be rarely associated with any organic disease; rather, they are regarded as reflecting distress in person-environment interaction (Aro and Taipale, 1987; Krantz and Östergren, 1999), which is supported by the hypothesis that early maturing girls are more vulnerable to various stressors during adolescence. Another limitation is that we did not have the possibility to control for body mass, known to be associated with menarcheal timing (Fredriks, Van Buuren, Jeurissen, Dekker, Verloove-Vanhorick and Wit, 2003; Frontini, Srinivasan and Berenson, 2003; Koziel and Jankowska, 2002). Future studies should try to isolate the impact of body mass, if any, on symptom reporting as related to menarcheal timing. Moreover, we did not capture all points across the lifespan, and important changes could have taken place before and between assessments. This, for instance, might include the possibility that some of the early maturing females with high frequencies of symptoms in adolescence might have shown high frequencies

even prior to their menarcheal debut. Indeed, this is suggested by Wångby's study of symptom stability between late childhood and adolescence (1997).

In conclusion, there was individual stability in symptom reporting for over nearly 30 years. Further, early menarcheal timing could be regarded as one factor that helps identify females at risk of poor health at a young age. However, the effect of early menarche on symptom reporting does not extend into middle age. Instead, it seems to be the experience of somatic symptoms per se in combination with indicators of depression that puts young females at risk for poor health development. Thus, if a higher symptom load of the sort discussed here is interpreted as indicating poor health then the results from the present study point to the possibility of identifying females already at risk at an early age. Mellner and Lundberg (2003) have indeed shown that self-reported symptoms predict self-rated, as well as physician-rated, general health status. However, in order to prevent poor health and to promote good health it is important to investigate other tentative factors involved, and to obtain an increased understanding of how they operate together in this developmental process.

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