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Life-course and cohort trajectories of mental health in the UK, 1991-2008 – a multilevel age-period-cohort analysis

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Abstract

There is ongoing debate regarding the shape of life-course trajectories in mental health. Many argue the relationship is U-shaped, with mental health declining with age to mid-life, then improving. However, I argue that these models are beset by the age-period-cohort (APC) identification problem, whereby age, cohort and year of measurement are exactly collinear and their effects cannot be meaningfully separated. This means an apparent life-course effect could be explained by cohorts. This paper critiques two sets of literature: the substantive literature regarding life-course trajectories in mental health, and the methodological literature that claims erroneously to have 'solved' the APC identification problem statistically (e.g. using Yang and Land's Hierarchical APC – HAPC – model). I then use a variant of the HAPC model, making strong but justified assumptions that allow the modelling of life-course trajectories in mental health (measured by the General Health Questionnaire) net of any cohort effects, using data from the British Household Panel Survey, 1991-2008. The model additionally employs a complex multilevel structure that allows the relative importance of spatial (households, local authority districts) and temporal (periods, cohorts) levels to be assessed. Mental health is found to increase throughout the life-course; this slows at mid-life before worsening again into old age, but there is no evidence of a U-shape – I argue that such findings result from confounding with cohort processes (whereby more recent cohorts have generally worse mental health). Other covariates were also evaluated; income, smoking, education, social class, urbanity, ethnicity, gender and marriage were all related to mental health, with the latter two in particular affecting life-course and cohort trajectories. The paper shows the importance of understanding APC in life-course research generally, and mental health research in particular.

Keywords

Research Highlights

- Previous research has suggested mental health takes a U-shaped life-course

- It is argued that this is a result of confounding with cohort effects

- Controlling for cohorts, mental haelth worsens throughout the life-course

- Associations with covariates (e.g. marriage) are found, that vary with age/cohort

- The paper has important methodological implications for wider life-course research

This paper considers longitudinal and life-course effects on mental health. How mental health varies, between social groups, as individuals age, and over time, is of interest to researchers examining the causes of psychiatric illness and mental distress more generally, and their public health implications.

This poses methodological challenges that are central to this paper. As a result of the age-period-cohort (APC) identification problem, it is impossible to predict APC trajectories accurately without making assumptions regarding at least one of APC (Bell & Jones, 2013b; Glenn, 2005). Other sources of dependency, particularly spatial dependency, should also be considered. Given these challenges, a multilevel model is presented which develops the Hierarchical APC (HAPC) model (Yang & Land, 2006, 2013), overcoming its recently exposed flaws (Bell & Jones, 2014b, c; Luo & Hodges, 2013) to model APC effects on mental health robustly. The HAPC model treats periods and cohorts as contexts in which individuals reside, and is here extended to incorporate other contexts, including spatial contexts such as households and geographical areas.

This paper challenges the view that, over the life-course, the trajectory of mental health is U-shaped (Blanchflower & Oswald, 2008; Lang et al., 2011) – worsening through young adulthood until mid-life, then improving into old age. In the analysis presented here – using British Household Panel Survey (BHPS) data – no such U-shape is found, suggesting potential health benefits of old age, including retirement, are overwhelmed by problems like dementia and loneliness. It is argued the U-shape finding resulted from a failure to control for cohort effects appropriately. The paper also explores how longitudinal and life-course trajectories may vary across individuals with different characteristics, e.g. income, education, ethnicity and marital status, and thereby contributes to the wider substantive literature on mental health.

The paper starts with a general overview of the literature on mental health, before considering the APC identification problem and, subsequently, how this relates to the literature on mental health over the life-course. This is followed by an explanation of the methods used and the results found.

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Mental health

Mental health can be defined as "a state of well-being in which an individual realizes his or her own abilities, can cope with the normal stresses of life, can work productively and is able to make a contribution to his or her community" (WHO, 2014). It is more than simply an absence of symptoms and diagnoses (depression, anxiety, stress, insomnia, etc.), including more subjective, non-clinical criteria, and a "full spectrum of mental health states", from positive (wellbeing) to negative (illness) (Weich et al., 2011:23). It is influenced by socio-economic, spatial and dynamic factors, which interact in complex ways.

In a review, Fryers et al. (2003) found socio-economic status, unemployment, education, income, and material living standards were all predictors of mental health, particularly of persistent depression (see also Lorant et al. (2003)). Many of these associations are complex, only occurring within certain groups. For example, urbanity (Verheij, 1996) and socio-economic status (Weich & Lewis, 1998a) has been found to predict mental health particularly for women and the elderly.

Where you live also affects your mental health. McKenzie et al. (2002) argue that the social network in which you interact (your 'social capital') is important in predicting mental disorders like schizophrenia. Weich et al. (2002) consider the built environment, finding that deck-access and recently built housing are associated with depression. Weich et al. (2005) also examined the importance of the household, finding similar levels of depression among cohabiting individuals. Larger-scale spatial units appear less important, with minimal differences between neighbourhoods in mental health (Propper et al., 2005; Weich et al., 2003). Larger-still geographical scales of analysis may be important; local authority districts (LADs) are units by which public health funding is now distributed in the UK (Department of Health, 2012), which could be an important mechanism by which spatial differences arise. Evidently, geography may matter at some scales more than others.

Mental health is dynamic. Psychiatric disorders have onsets and recoveries and can be chronic or more temporary in nature (Weich & Lewis, 1998a:9), whilst their predictors also vary over time. Thus, Weich and Lewis (1998b) find that poverty and unemployment effect recovery from, but not the onset of, mental disorders. Benzeval and Judge (2001) find that long-term poverty has a bigger effect on mental health than short term poverty. Lindstrom et al. (2014) find that risk factors accumulate through the life-course, with factors in childhood adding to contemporary factors to affect mental health later in life. Regarding changes over the life-course, Musick and Bumpass (2012) find that the positive association of marriage and mental wellbeing dissipates through the life-course, whilst Jorm (1999) finds the association between mental disorders and smoking is smaller in old age.

The Age-Period-Cohort identification problem

A key methodological conundrum when considering temporal facets of mental health is the APC identification problem. Whilst this has been part of the literature for decades (Glenn, 1977; Mason et al., 1973; Ryder, 1965), serious misunderstandings remain across the social sciences (Bell & Jones, 2014b). This section clarifies these misunderstandings before considering their relevance to understanding changing mental health.

The differences between age, period, and cohort effects is explicated by this fictional dialogue by Suzuki (2012:452):

A: I can't seem to shake off this tired feeling. Guess I'm just getting old. [Age effect]

B: Do you think it's stress? Business is down this year, and you've let your fatigue build up. [Period effect]

A: Maybe. What about you?

B: Actually, I'm exhausted too! My body feels really heavy.

A: You're kidding. You're still young. I could work all day long when I was your age.

B: Oh, really?

A: Yeah, young people these days are quick to whine. We were not like that. [Cohort effect]

In summary, age effects result from individuals growing older, period effects result from factors specific to the year of measurement, and cohort effects result from similarities between individuals born contemporaneously (e.g. due to common factors affecting them in their formative years).

However, APC are exactly co-linear, such that the value of one can be found if you know the values of the other two:

$$Period = Age + Cohort$$

(1)

As shown previously (Bell & Jones, 2014b:336-337), this means an apparent effect of age could fully or in part be the result of combined period and cohort processes. Imagine that mental health is determined solely by cohort and age effects, each of value 2:

$$Mental\ Health = (2 * Age) + (2 * Cohort)$$

(2)

Substituting (1) into (2), the following data generating processes produce *identical* dependent variables:

$$Mental\ Health = (1 * Age) + (1 * Period) + (1 * Cohort)$$

$$Mental\ Health = 2 * Period$$

(3)

Therefore one cannot tell, from a given dataset alone, which of the above processes produced the outcome variable, and thus the true size of (or even presence of) an age effect. A model attempting to differentiate all three linear effects cannot be estimated because of exact collinearity, whilst controlling for only two of APC risks confounding with the third. This problem is "in the population, not just in the sample… [meaning it] cannot simply be solved by manipulating the data or the model"

(Bell & Jones, 2014b:338), unlike other problems of (inexact) collinearity, where collecting more data could be a solution.

Regardless, many have attempted to solve the identification problem statistically (Mason et al., 1973; Robertson & Boyle, 1986). Some group one of APC to break the exact collinearity – producing results that arbitrarily depend on the chosen grouping (Glenn, 1976; Osmond & Gardner, 1989). Others use more complex statistical legerdemain. Here I consider the Hierarchical APC (HAPC) model (Yang & Land, 2006, 2013), since it is adapted for use in this paper. This cross-classified multilevel model is designed for repeated-cross-sectional data, and treats periods and cohorts as contexts in which individuals reside (figure-1). It can be specified as a 'micro' (individual-level) and 'macro' (higher-level) equation as follows:

$$y_i = \beta_{0i} + \beta_1 Age_i + \beta_2 Age_i^2 + e_i$$

$$\beta_{0i} = \beta_0 + u_{Cohort} + u_{Period}$$

$$e_i \sim N(0, \sigma_e^2), \qquad u_{Cohort} \sim N(0, \sigma_{u1}^2), \qquad u_{Period} \sim N(0, \sigma_{u2}^2)$$

(4)

[Figure-1 about here]

i represents individuals; a residual is associated with each cohort group (u_{Cohort}) and period (u_{Period}), variances of which are estimated (σ_{u1}^2 and σ_{u2}^2 respectively). In contrast, the age effect is estimated as a polynomial function (here linear and quadratic), with parameters β_1 and β_2. It is argued that, because age effects are estimated differently to period and cohort effects, and because age effects are estimated non-linearly, the identification problem is solved:

> "The underidentification problem of the classical APC accounting model has been resolved
>
> by the specification of the quadratic function for the age effects."
>
> (Yang & Land, 2006:84)

"An HAPC framework does not incur the identification problem because the three effects are not assumed to be linear and additive at the same level of analysis"

<div align="right">(Yang & Land, 2013:191)</div>

Unfortunately this is incorrect; simulation studies show the HAPC model can produce incorrect estimates (Bell & Jones, 2014b, c; Luo & Hodges, 2013). This is unsurprising given the argument above; the HAPC model logically cannot do what is claimed (Fienberg, 2013; Glenn, 1976, 2005).

Failing to understand the ubiquity of the identification problem, and the impossibility of a mechanical solution to it, has led to numerous misleading findings. This problem is widespread in research on mental health, as shown next.

Longitudinal and life-course effects on mental health

Given the impossibility of separating APC effects, when modelling life-course or longitudinal effects one must make assumptions regarding at least one of APC to estimate their effects robustly. In some research areas there may not be a theoretical basis on which to base such assumptions, and they cannot be confirmed empirically. However with mental health, strong assumptions can be justified. Following Spiers et al. (2011) I argue that it is unlikely that there would be any linear (or higher polynomial) period trends in mental health. This is not to say that periods never matter – hypothetically, there could be a decline in mental health with an economic recession, or an increase during national celebrations. However there is no reason to expect a continuous trend across periods affecting all ages. Cohorts, through the nature of individuals' upbringings, more plausibly explain how changes in mental health could occur over time.

The reader may question this assumption – it cannot be confirmed empirically, relying instead on the researcher's intuition (ideally reinforced by theory). However such discussion is absent from previous research into life-course effects on mental health. Whilst some mention cohort and/or period effects,

the consequences of the identification problem on life-course effect-estimates are rarely recognised. Usually, the identification problem is left unconsidered.

The shape of life-course trajectories in mental health, both in terms of mental illness and mental wellbeing, has attracted debate involving researchers across social science and medical disciplines, although there is a limited social-science and economic theory to underpin this debate (Blanchflower and Oswald, 2008:1735). Much of this research argues that life-course trajectories are U-shaped, with mental health declining to mid-life and then improving with old age. To some extent this fits common conceptualisations of 'midlife crises', whereby individuals become unsatisfied with unmet aspirations (Schwandt, 2013) and attempt to rekindle their youth, often accompanied by mental distress such as anxiety and depression; this research has attracted media attention (Economist, 2010). There are theoretical and anecdotal reasons to believe midlife crises exist and can negatively affect individuals' wellbeing and health more generally, but uncertainty remains. Some argue that stereotypes of midlife crises are based on changing cultural age-related norms, and no mid-life change occurs (Freund & Ritter, 2009:582). Whilst midlife crises are generally understood as discrete periods lasting a few years, rather than the U-shape across the whole life-course found by this research, the *a posteriori* U-shape is notable, particularly the dramatic improvement in mental health found in later life. Such an improvement could result from, for example, a lack of stress in retirement (Bosse et al., 1991), but one would expect this to be tempered, if not reversed, by loneliness (Wenger et al., 1996), or age-related mental illnesses such as dementia.

However, I argue previous analyses of both wellbeing and mental disorders fail to control appropriately for cohort effects. Some attempt to control for both periods and cohorts (Blanchflower & Oswald, 2008; Clark & Oswald, 2006), some control only for periods (Blanchflower & Oswald, 2009; Howden-Chapman et al., 2011) whilst others are cross-sectional analyses that cannot control for cohorts due to exact collinearity with age (Blanchflower & Oswald, 2011; Deaton, 2008; Lang et al., 2011). Papers finding no U-shape (Frijters & Beatton, 2012; Kassenboehmer & Haisken-DeNew, 2012)

tend to be fixed effects analyses, controlling for all individual level variability using dummies or de-meaning (Bell & Jones, 2014d). Because cohort is an unchanging attribute of individuals, this controls for cohort effects (unless periods are additionally controlled).

Methodology

Extension of the HAPC model

Whilst the HAPC model does not work as claimed, the model presents a compelling conceptualisation of APC. The multilevel framework is intuitive and it can easily be extended to incorporate other random levels (e.g. spatial settings). However, first certain assumptions need to be made so that the results found are non-arbitrary. As argued above, one can assume that there are no continuous period trends, and thus equation-4 can be extended by including a cohort polynomial in the fixed part of the model:

$$y_{i(j_1 j_2)} = \beta_{0i} + \beta_1 Age_i + \beta_2 Age_i^2 + e_i$$

$$\beta_{0i} = \beta_0 + \beta_3 Cohort_i + \beta_4 Cohort_i^2 + u_{Cohort} + u_{Period}$$

$$e_i \sim N(0, \sigma_e^2), \qquad u_{Cohort} \sim N(0, \sigma_{u1}^2), \qquad u_{Period} \sim N(0, \sigma_{u2}^2)$$

(5)

β_3 and β_4 estimate the continuous cohort trend; u_{Cohort} estimates cohort-level differences around that trend. Additionally, an interaction between cohort and age should be included (Miyazaki & Raudenbush, 2000). This does not, as others claim (for example see Yang & Land, 2013:291), act as a period effect; rather it allows for different cohort groups to have different age effects (Bell & Jones, 2014a). Such a model has been used elsewhere (Chen et al., 2010; McCulloch, 2014; Shaw et al., 2014; Yang, 2007; Yang & Lee, 2009), but without the advantage of estimated period and cohort random effects.

Other structural levels can also be included. With panel data (rather than repeated-cross-sectional data), it is important to include an individual level, accounting for dependency within individuals (as suggested by Suzuki, 2012); age becomes an observation-level variable. Other spatial levels can be included, building the structure from three levels (figure-1) to six (figure-2). Additional covariates, and interactions involving those covariates, can be included. Finally, the random part of the model can be extended to include random slopes – e.g. allowing the age trend to vary between individuals. Equation-5 can thus be extended to:

$$y_i = \beta_{0i} + \beta_{1i}Age_i + \beta_2 Age_i^2 + \sum_6^p \beta_p X_{pi} + \sum_6^p \beta_{Cp}(X_{pi} * Cohort_i) + \sum_6^p \beta_{Ap}(X_{pi} * Age_i) + e_i$$

$$\beta_{0i} = \beta_0 + \beta_3 Cohort_i + \beta_4 Cohort_i^2 + u_{LAD} + u_{Household} + u_{Period} + u_{Cohort} + u_{Individual(0)}$$

$$\beta_{1i} = \beta_1 + \beta_5 Cohort_i + u_{Individual(1)}$$

$$u_{LAD} \sim N(0, \sigma_{u(5)}^2), \qquad u_{Household} \sim N(0, \sigma_{u(4)}^2), \qquad u_{Cohort} \sim N(0, \sigma_{u(3)}^2), \qquad u_{Period} \sim N(0, \sigma_{u(2)}^2),$$

$$\begin{bmatrix} u_{Individual(0)} \\ u_{Individual(1)} \end{bmatrix} \sim N\left(0, \begin{bmatrix} \sigma_{u1I}^2 & \\ \sigma_{u1Iu1S} & \sigma_{u1S}^2 \end{bmatrix}\right), \qquad e_i \sim N(0, \sigma_e^2)$$

(6)

[Figure-2 about here]

Here, i refers to observations (the lowest level) and not individuals. X_{pi} represents a series of p-5 covariates, with parameter estimates β_p, and their interactions with age and cohort are estimated by β_{Ap} and β_{Cp} respectively; β_5 estimates the age-by-cohort interaction effect. The random age slopes are specified by allowing β_{1i} to vary by residual $u_{Individual(1)}$, which has a variance estimated as σ_{u1S}^2, along with the intercept's variance (σ_{u1I}^2) and their covariance (σ_{u1Iu1S}). Other levels – LAD, Household, Cohort, Period and Observation – each have an associated set of residuals, and estimated variance.

Data

Data come from the BHPS, a representative survey of individuals tracked from 1991-2008 (The BHPS ended in 2008, replaced by a new dataset, *Understanding Society* - analysis of that dataset is beyond this paper's scope). Our dependent variable, measuring mental health, is derived from the General Health Questionnaire (GHQ-Goldberg & Williams, 1988). Respondents answer the following 12 questions, on a 4-point scale coded from 0-3 (0 representing the best health and 3 the worst):

Have you recently:

- been able to concentrate on whatever you are doing?

- lost much sleep over worry?

- felt that you are playing a useful part in things?

- felt capable of making decisions about things?

- felt constantly under strain?

- felt you couldn't overcome your difficulties?

- been able to enjoy your normal day to day activities?

- been able to face up to your problems?

- been feeling unhappy and depressed?

- been losing confidence in yourself?

- been thinking of yourself as a worthless person?

- been feeling reasonably happy, all things considered?

These scores are summed, creating a 37-point scale that measures general mental health, with higher scores indicating worse health (Taylor et al., 2010). This acts as a predictor of being a psychiatric case, but includes both positive and negative elements of mental health, including more transient problems not requiring treatment. Whilst specific facets of health cannot be distinguished with this measure, it is ideal for this paper, where mental health is conceived more generally than psychiatric illness.

We include control variables in our model, described in table-1, which were identified in the literature as important predictors of mental health (see above). This is done in a somewhat exploratory way given that the theoretical base for such covariates is limited. There is debate about the inclusion of such controls, because the direction of causality is unclear (Blanchflower & Oswald, 2009; Glenn, 2009): whilst marriage could affect mental health, health could also affect the likelihood of marriage. Including such controls has value, partly because including interactions with those covariates can reveal, albeit in an exploratory way, how age and cohort trends differ for different demographics, which has already been considered for a number of the covariates in previous work (see above). We also present models including only age, cohort and gender; substantively the results do not differ.

[Table-1 about here]

Our data also include identifiers for individuals, household-years and LADs – which are included as random variables, alongside periods and cohorts (the latter grouped into 5-year intervals to reduce dependency between each cohort unit).

Modelling strategy

Modelling was conducted in MLwiN v2.30 (Rasbash et al., 2014) with MCMC estimation (Browne, 2009). Models were run for 50,000 iterations, following a 2000 iteration burn-in, which was sufficient for all parameters to converge to a non-trending distribution, with an effective sample size of >400. Hierarchical centring was used to accelerate convergence (Browne, 2009:401).

We implement a bottom-up modelling strategy, starting with a simple model and building complexity (see table-2). Model-1 is a 2-level model, with observations nested within individuals. Age and cohort polynomials were included as far as significant (up to a cubic term), alongside an interaction between the age and cohort linear terms. A gender dummy variable, and interactions between gender and age/cohort polynomials, were also included (again to polynomial orders as high as significant). The model was then extended from two to six levels (model-2), with each level added consecutively, and

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their significance tested using the Deviance Information Criterion (DIC-Spiegelhalter et al., 2002). Covariates were added (model-3), followed by random slopes (model-4), the latter testing (a) whether the cohort random effects differ between genders (adding another dimension to the gender-by-cohort interaction terms), and (b) whether the age effect varies across individuals. At this stage, any non-significant effects were removed (model-5) on the basis of a Bayesian p-value of >0.05 for fixed effects and a substantial decline in the DIC for random effects. Next, interactions were included, between each of the covariates, and age/cohort (model-6), to allow for possible differences in life-course and longitudinal effects by demographic characteristic. These were retained if the effects of the ($X_{pi} * Age_i$) and ($X_{pi} * Cohort_i$) interactions were jointly significant. This model was used to create the graphs below, but the results did not substantively differ from other models.

Within- and between-individual effects

A problem with multilevel models (or 'random effects' models) is bias when covariates are correlated with (higher-level) residuals (Wooldridge, 2002:257). This is because covariates can have different effects at different levels of analysis, often termed between- and within-individual effects (Bell & Jones, 2014d). However using a variant of the formulation suggested by Mundlak (1978) mitigates this problem by specifying within and between effects explicitly. Thus, in model-7, the effect of each observation-level covariate was split into within- and between-individual components, using the individual-mean-centred variable for the within effect and the individual mean for the between effect.

Three points should be noted here. First, between effects can be biased by uncontrolled higher-level confounders (e.g. unmeasured attributes of individuals) – care must be taken when interpreting these coefficients. Second, effects are separated into only two levels (individuals and observations) – there could remain additional biases from correlations with other random effects. However, it was not feasible to estimate separate effects for each of our six levels for each covariate, and given that little variance is located at other levels, any bias would be minimal. Third, birth year (our measure of

cohort) is exactly collinear to the individual mean of age. Thus, the inclusion of cohorts in the model means that age effects are already the corrected within-individual effects (Bell & Jones, 2014b).

[Table-2 about here]

Results

All six levels were found to have significant variance (the DIC declined when each level was included). When all 6 levels were included in the model, the age-by-cohort interaction became insignificant and was omitted. Allowing the gender effect to vary at the cohort-group level improved the model only marginally (the DIC declined by <10, and the results of the more complex model were not substantively different); for the sake of simplicity these random terms were omitted from later models.

APC effects

Figure-3a shows the combined age and cohort effects on GHQ-score (i.e. the predictions based on the age/cohort parameter estimates in model-6). The U-shape found by others is to some extent visible, with mental health peaking around age 40, then declining (before rising in old age). However, this is an inappropriate test for life-course effects because it includes both age and cohort effects in the predictions. Instead, figures-3b and -3c show the age and cohort effects respectively, conditional on the other, and other covariates, with trends separated by gender. As can be seen, when cohort effects are controlled, there is no evidence of the U-shaped life-course trajectory. Mental health worsens throughout the life-course, and whilst this slows in mid-life, especially for men, there is little evidence of an improvement in mental health at any stage of life. The improvement apparent in figure-3a is in fact a result of a cohort effect (figure-3c), whereby those in later cohorts, particularly females, report worse mental health.

[Figure-3 about here]

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As well as life-course and cohort *trends* in mental health, the model also finds stochastic variation between cohort groups and periods. Figure-3c shows the cohort effect, including both the fixed quadratic trend and the random, cohort-level variation. There is significant variation between cohort groups around the overall trend. People born 1965-1974 (and to a lower level of statistical significance, 1930-34) had significantly better mental health than would be expected on the basis of the estimated quadratic trend, whilst those born in 1940-44 in general had worse mental health. The latter suggests a negative effect of World War II for those born during it, on their later life. The former is perhaps more surprising – it implies that recessions (e.g. those in the 1930s and 1970s) during an individual's formative years could improve that individual's subsequent mental health. Regarding periods, there are apparent differences between years, with generally better mental health reported in 1991 and 2003, and worse health in 1995 and 2000 (figure-3d). However, these effects are small compared to the cohort/age effects, meaning their substantive importance should not be overstated.

Stochastic individual and spatial effects

As well as period and cohort variances, the model also estimates variances for LADs, household-years, individuals and observations. Nearly half of the variation (49% based on model-2) occurs within individuals once age, cohort and gender are controlled – in other words an individual's mental health varies considerably year-to-year. The majority of the remaining variance is between individuals (41%), suggesting some individuals consistently have better mental health than others. A relatively large proportion of the variance (8%) occurs at the household-year level, in line with previous findings (Weich et al., 2005) that cohabiting individuals have similar levels of mental health. Finally, although statistically significant, LADs have only a small effect on mental health, accounting for 0.5% of the total variance, in line with Propper et al. (2005) and others who find limited spatial effects, and suggesting varying public health policies between LADs will have minimal effects on mental health. The period and cohort random effects are even smaller (with age and cohort *trends* controlled) – accounting for 0.1% and 0.2% of the variance, respectively.

In model-4 onwards, the linear component of the age effect was allowed to vary between individuals. Figure-4 shows the coverage intervals that result from allowing individuals to have different age slopes. Individuals vary greatly, and increasingly vary as they age – whilst some remain relatively mentally healthy (or improve in health), others deteriorate (perhaps because of age-related problems like dementia), producing a 'fanning out' of mental distress levels over the life-course.

[Figure-4 about here]

Other covariates, and their interactions with age/cohort

Several other covariates were included to evaluate their relationships with mental health, and their effect on life-course and cohort trajectories. It should be noted that these should be interpreted carefully, given the somewhat exploratory nature of the analysis. We are testing the significance of multiple variables, chosen with a relatively limited theoretic base, and so further theoretical work and testing should be undertaken to confirm the external validity of the following findings.

All the variables tested had significant effects on GHQ-scores (model-3 onwards). Individuals who are female, of lower social class, living in urban areas, non-white, poor, unmarried, smoke, and have only primary level education, in general have the worst mental health (at least as reported through the GHQ). There were no differences in mental health between different non-white ethnicities, nor between different education levels above primary education, and so these dummy variables were not included in the presented models.

Additionally, interactions between the age and cohort effects, and the above covariates, were evaluated to see how covariates' relationships with GHQ-score vary across cohorts and the life-course. Whilst only interactions with the linear age and cohort effects are presented here, interactions with higher-order polynomials of age and cohort were included in other models; whilst some of these were statistically significant, they did not alter the results substantively. The key significant findings are displayed in figure-5. The apparent benefits of marriage appear to be limited to the young, in line

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with the findings of Musick and Bumpass (2012): older people if anything have worse mental health if married. Among unmarried people there appears to be a weak U-shape trajectory (figure-5a). Controlling for these differential age effects, there are also differences in cohort trajectories between married and unmarried individuals; the benefits of marriage decrease over time and the increase in GHQ-score with cohorts is only evident for married individuals. Figure-5c shows that the beneficial effects of having more than primary education increases over the life-course (although this interaction is only marginally significant). Finally, figure-5d shows that the relationship between smoking and GHQ seems to be greater among more recent cohorts, with no differences between smokers and non-smokers in older cohorts; such a process could have produced the apparent age-smoking interaction found by Jorm (1999).

[Figure-5 about here]

The GHQ-Urbanity and GHQ-Income associations were also found to vary with age/cohort (the age and cohort interactions were jointly significant) but individually the interaction terms were non-significant, making it impossible to say which of the two effects drive these differences. Younger individuals appear less affected by urbanity and more affected by income than older people – but whether this is driven by an age or a cohort effect cannot be ascertained. No age/cohort interactions were found with either ethnicity or social class – these relationships appear constant across cohorts and the life-course.

One should not interpret these effects causally – reverse causality is possible, and there are potential confounders that could explain the apparent relationships. However, it remains valuable to consider how different populations (e.g. married and unmarried) differ in both their mental health, and their life-course and cohort trajectories. Statistical methods that attempt to consider reverse causality exist, but are beyond this paper's scope- e.g. the multilevel distributed lag model (Bell et al., 2014) gives an indication of causality for multilevel data.

Finally, model-7 divides the covariates' effects into within- and between-individual components. The within-individual effect of primary education was non-significant (unsurprisingly, given it varies little within individuals) as were the between-individual effects of the income-by-age and income-by-cohort interactions (these were removed from the model). That is, there is no evidence of an effect of gaining primary education in later life (only of having it from childhood), nor of generally having higher income (only of a change in income). There was no statistically significant difference in the between- and within-individual effects of social class and urbanity, so un-separated effects were retained (ethnicity and gender do not vary within individuals so only have between-effects). The effect of income on age/cohort trajectories appears to occur within-individuals – changes in income, rather than individuals' general level of income over the sample period, are related to mental health trajectories. Marriage appears to have both a within- and between-individual effect (both getting married, and being more pre-disposed to be married, are independently related positively to mental health). The effect of smoking appears to be predominantly (but not exclusively) a between-individual relationship; an individual's predisposition to smoke, rather than a change in the number of cigarettes smoked is related to GHQ-score (although the within-effect is also significant). It can be argued that significant within-effects are more suggestive of a causal effect (since they are net of any individual-level characteristics) although reverse causality remains a strong possibility; testing this conclusively would require quasi-experimental methods (e.g. natural experiments).

Discussion

The key contribution of this paper is to question the stylised fact of a consistent U-shaped trend in the life-course trajectory of individuals, with mental health problems peaking in mid-life. I argue these findings have been the result of a failure to understand the APC identification problem, and thus to appropriately control for cohort effects. Instead, mental distress appears to rise throughout the life-course: the negative effects of old age on mental health seem to outweigh the positives.

This is not to say that the U-shape is entirely without merit. First, here we examine general mental health, and do not differentiate between different facets of mental health. Different forms of health may have different life-course trajectories, with some (e.g. work-related stress) more likely to be U-shaped. Moreover, positive (wellbeing) and negative (illness) dimensions of mental health may have different trajectories (Hu et al., 2007). Second, an (albeit weak) U-shaped life-course trajectory was found for unmarried individuals. Finally, our study is limited to the UK and so its applicability to other contexts is uncertain; work using cross-national datasets, similar to that of Blanchflower and Oswald (2008) but with age and cohort properly specified, could help confirm this.

As well as allowing the life-course effect to be estimated accurately, the estimated cohort effects are themselves substantively interesting. Successive cohorts appear to have increasingly poor mental health, particularly among women, those who are married, and smokers. The general trend fits with theories about the increasing pace of modern life, but is not consistently found – e.g. Spiers et al. (2011) find no consistent cohort trend. In addition to the general trend, by modelling cohorts stochastically, evidence was found for better mental health among those brought up during recessions. This could be because individuals who are brought up experiencing hardship, but become better-off in later life, are more able to deal with problems than those without experience of hardship.

Finally, this article makes important methodological arguments that apply across disciplines. The APC identification problem can, and has, produced misleading results (for further examples see Bell & Jones, 2013a,b; Bell & Jones, 2014b,c). In this paper, continuous period trends have been assumed zero – an assumption that was not (and could not be) based on the data at hand. There remains space for explicit debate here, regarding in what situations period or cohort trends (or both) would be expected theoretically. This would be an improvement on the unstated and often unintended assumptions implicit in much life-course and longitudinal research, across the social sciences.

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Table-1: Description of variables used in this analysis.

Variable	Description	Mean/ Proportion	Range
Dependent variable			
General Health Questionnaire	Respondents answer 12 questions on a scale from 0-3, which are then summed to a single scale from 0-36.	11.2	0-36
Independent variables			
Age		46.0	18-100
Cohort	Birth Year	1954	1894-1990
Female	0=Male, 1=Female	0.54	
Household Social Class	Cambridge Scale, rescaled to 0-1 (Prandy, 1990)	0.34	0.0055-0.9999
Urban	0=Rural, 1=Urban (pop>10,000)	0.74	
Non-White	0=White, 1=Non-white	0.03	
Married	0=Not married, 1=Married	0.56	
Household Income	Net weekly household income, Equivalized using McClements 'before housing costs' scale, adjusted to January 2010 prices (Levy & Jenkins, 2012). In £1000s.	0.471	-0.058-10.3
Primary Education only	Education level, based on ISCED. 0=More than primary education, 1=Only has primary education (UNESCO, 2006)	0.26	
N cigarettes per day	In 10s of cigarettes	0.4	0-8.1
Random (structural) Variables			
Local Authority District	405/404 LADs		
Household-Year	113907/93168 household-years		
Year	18 years		1991-2008
Cohort Group	Birth year, grouped into 19 5-year intervals		1894-1899, 1900-1904, … 1980-1984 1985-1990
Individual	25883/21142 individuals		
Observations	194217/160927 observations		

Note: the two sample sizes for the random variables refer to their sample size with and without observations with missingness in the covariates.

Table-2: Model parameter estimates.

	1. Age, Cohort, Gender, 2-levels		2. Age, cohort, gender, 6-levels		3. With other covariates		4. With Random Slopes		5. More Parsimonious model		6. With interactions		7. Within/between-effects separate			
	β	SE	β	SE	β	SE	β	SE	β	SE	β	SE	β	SE	β	SE
Fixed Part													*Unseparated effects*			
Constant	11.023	0.052***	10.997	0.108***	10.981	0.123***	11.021	0.129***	10.980	0.120***	10.948	0.131***	10.991	0.120***		
Age	-0.009	0.005*	-0.008	0.009	-0.006	0.009	-0.005	0.009	0.003	0.008	-0.037	0.010***	-0.014	0.009+		
Age²	-0.002	0.000***	-0.002	0.001+	-0.003	0.001**	-0.003	0.001**	-0.001	0.000***	-0.002	0.000***	-0.002	0.000***		
Age³	0.000	0.000***	0.000	0.000***	0.000	0.000***	0.000	0.000***	0.000	0.000***	0.000	0.000***	0.000	0.000***		
Female	1.150	0.066***	1.131	0.064***	1.196	0.070***	1.172	0.087***	1.199	0.068***	1.178	0.069***	1.179	0.068***		
Cohort	0.007	0.004*	0.008	0.009	0.006	0.009	0.005	0.009	0.014	0.008*	-0.017	0.010+	0.007	0.010		
Cohort²	-0.001	0.000***	-0.001	0.001	-0.002	0.001**	-0.002	0.001*	-0.001	0.000***	0.000	0.000**	-0.001	0.000***		
Age*Female	0.020	0.006***	0.204	0.055***	0.018	0.006**	0.017	0.007**	0.016	0.006**	0.017	0.006**	0.017	0.006**		
Age²*Female	0.001	0.000***	0.001	0.000***	0.001	0.000***	0.001	0.000***	0.001	0.000***	0.001	0.000***	0.001	0.000***		
Age³*Female	-0.000	0.000***	-0.023	0.005***	-0.000	0.000***	-0.000	0.000***	-0.000	0.000***	-0.000	0.000***	-0.000	0.000***		
Cohort*Female	0.012	0.005**	0.013	0.005**	0.019	0.005***	0.019	0.006***	0.018	0.006***	0.021	0.006***	0.019	0.006***		
Age*Cohort	-0.002	0.001**	-0.001	0.002	-0.004	0.002+	-0.004	0.002+								
Social Class (Cambridge Scale, rescaled 0-1)					-0.783	0.132**	-0.775	0.132**	-0.778	0.134**	-0.728	0.133**	-0.319	0.136**		
Urban					0.104	0.054*	0.116	0.055*	0.115	0.055*	0.136	0.057**	0.111	0.058*		
Non-White					0.508	0.168**	0.478	0.165**	0.479	0.167**	0.484	0.165**	0.484	0.164**		
Age*Urban											0.002	0.007	0.001	0.006		
Cohort*Urban											-0.006	0.007	-0.007	0.007		
													Within effects		*Between effects*	
Primary Education only					0.475	0.066***	0.467	0.067***	0.467	0.069***	0.489	0.072***			0.386	0.079***
N cigarettes (10s per day)					0.351	0.024***	0.352	0.024***	0.352	0.024***	0.360	0.026***	0.135	0.035***	0.646	0.040***
Married					-0.427	0.041***	-0.466	0.042***	-0.466	0.042***	-0.362	0.045***	-0.399	0.059***	-0.398	0.074***
Income (£1000s/week, equivalized and adjusted)					-0.497	0.048***	-0.513	0.049***	-0.512	0.048***	-0.498	0.052***	-0.294	0.059***	-1.542	0.120***
Age*Married											0.046	0.006***	0.018	0.011+	0.063	0.007***
Cohort*Married											0.050	0.006***	0.031	0.011**	0.043	0.007***

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	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Age*Income					0.002 (0.009)	0.008 (0.013)	
Cohort*Income					-0.011 (0.009)	-0.003 (0.013)	
Age*Primary Ed					0.013 (0.007*)		0.010 (0.007+)
Cohort*Primary Ed					0.004 (0.007)		0.003 (0.007)
Age*N cigarettes					0.004 (0.003)	-0.003 (0.007)	0.009 (0.004**)
Cohort*N cigarettes					0.012 (0.003***)	0.008 (0.007)	0.010 (0.004**)
Random Part							
Local Authority District		0.159	0.139	0.124	0.125	0.123	0.121
Household-Year ‡		2.467	2.563	2.523	2.520	2.505	2.504
Year		0.015	0.016	0.016	0.018	0.017	0.017
Cohort Group							
(Intercept)		0.057	0.058	0.069	0.057	0.074	0.067
(Covariance)				-0.009			
(Female slope)				0.028			
Individual							
(Intercept)	12.497	12.171	11.632	10.518	10.528	10.559	10.461
(Covariance)				0.075	0.075	0.077	0.075
(Age slope)				0.007	0.007	0.007	0.007
Observation	16.996	14.553	14.369	14.126	14.128	14.126	14.121
DIC:	1120591	1113014	920797	918932	918941	918836	918743
N observations	194217	194217	160927	160927	160927	160927	160927

Note: + p<0.1; * p<0.05; ** p<0.01; ***p<0.001 (Bayesian p-values). All non-binary variables were centred on their grand mean. Full algebraic specification of these models can be found in an online appendix.

‡ Household-years were used, rather than households, because the BHPS only provides household-year identifiers.

Figure-1: Multilevel cross-classified structure of the HAPC model, with individuals nested within year of measurement, and cohort group. Note that the data are not structured in a strict hierarchy.
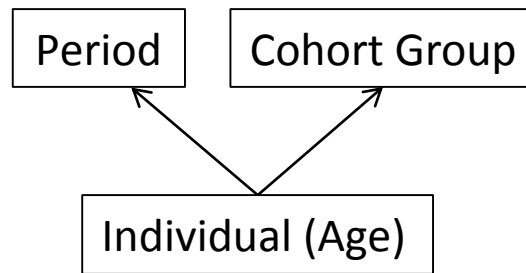

Figure-2 – Extension of the HAPC multilevel structure, incorporating spatial hierarchies and allowing for the model to be used with panel rather than repeated cross-sectional data.
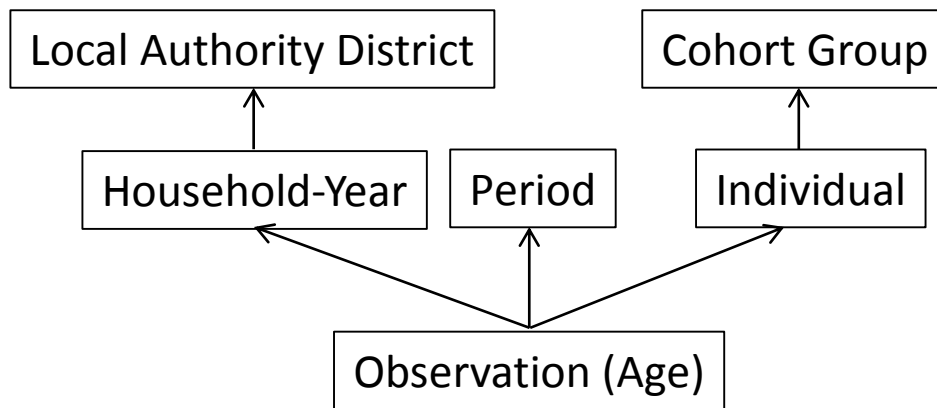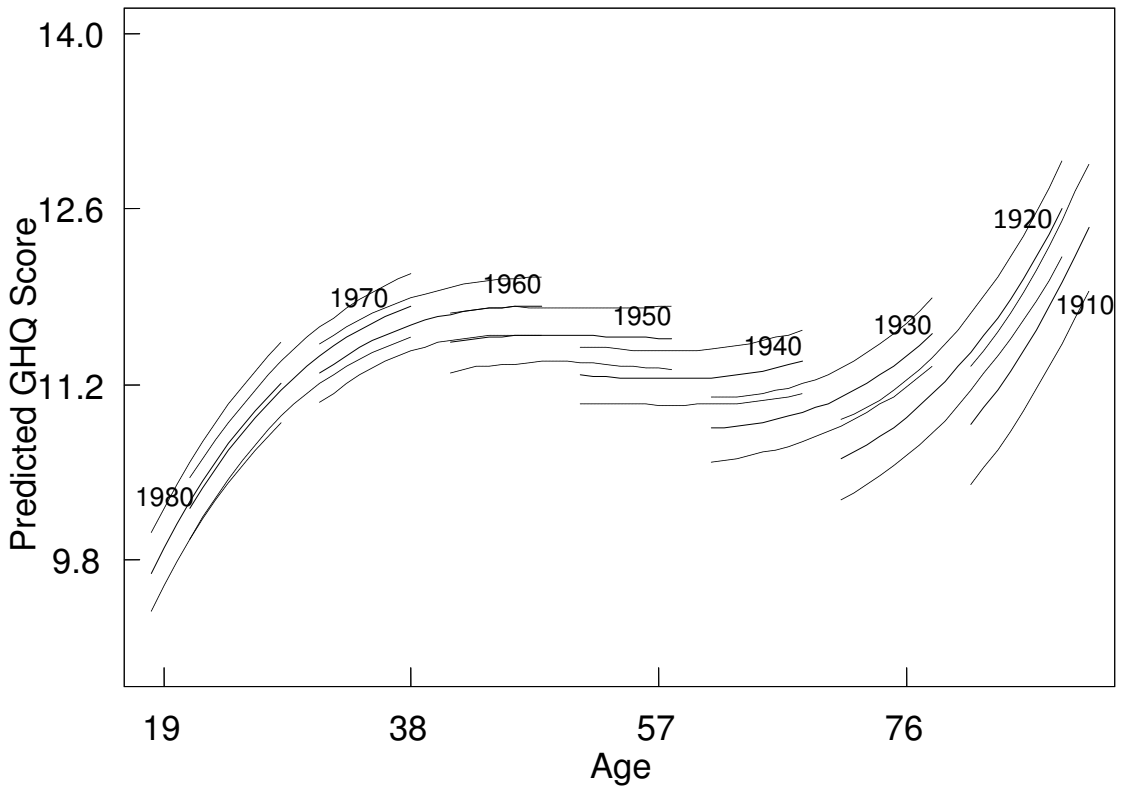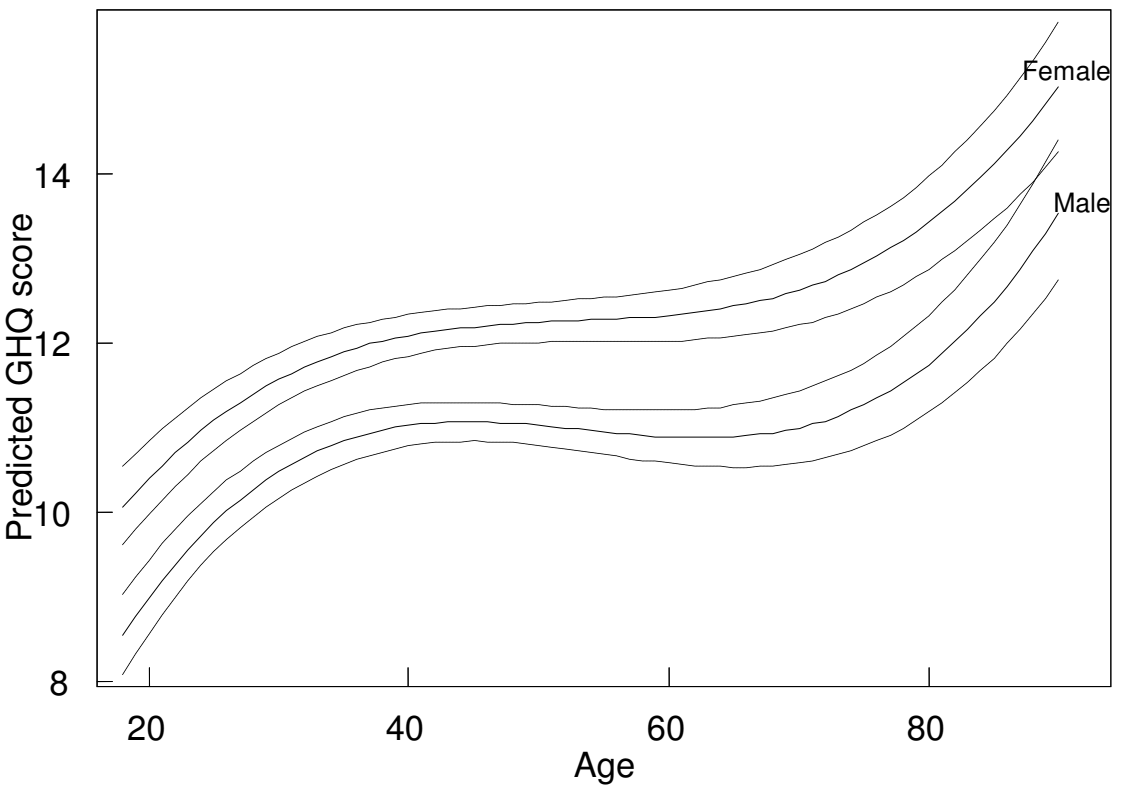

Figure-3: (a) age and cohort (fixed) effects combined into a single graph (each line represents a different cohort, with birth years labelled; (b) age effect on GHQ, split by gender and conditional on cohort and other covariates, (c) cohort effect (combining fixed and random part estimates) on GHQ, split by gender and conditional on age and other covariates; (d) period effects on GHQ, based on the period-level residuals. Dotted lines are 95% confidence intervals.
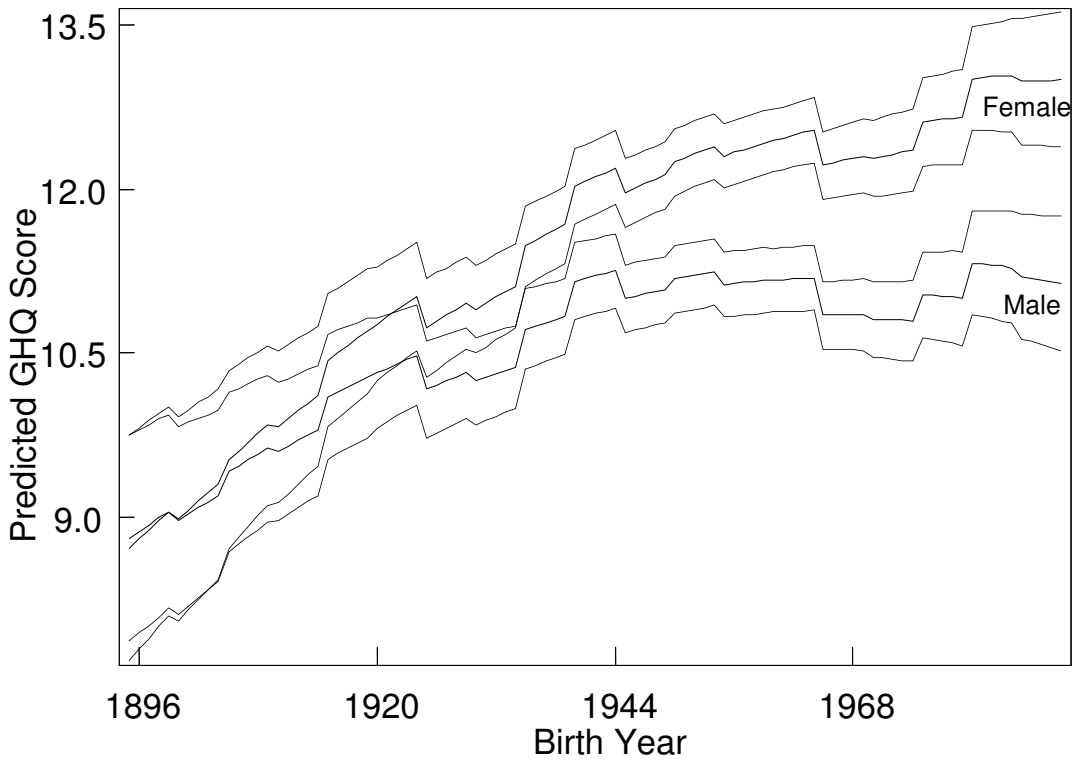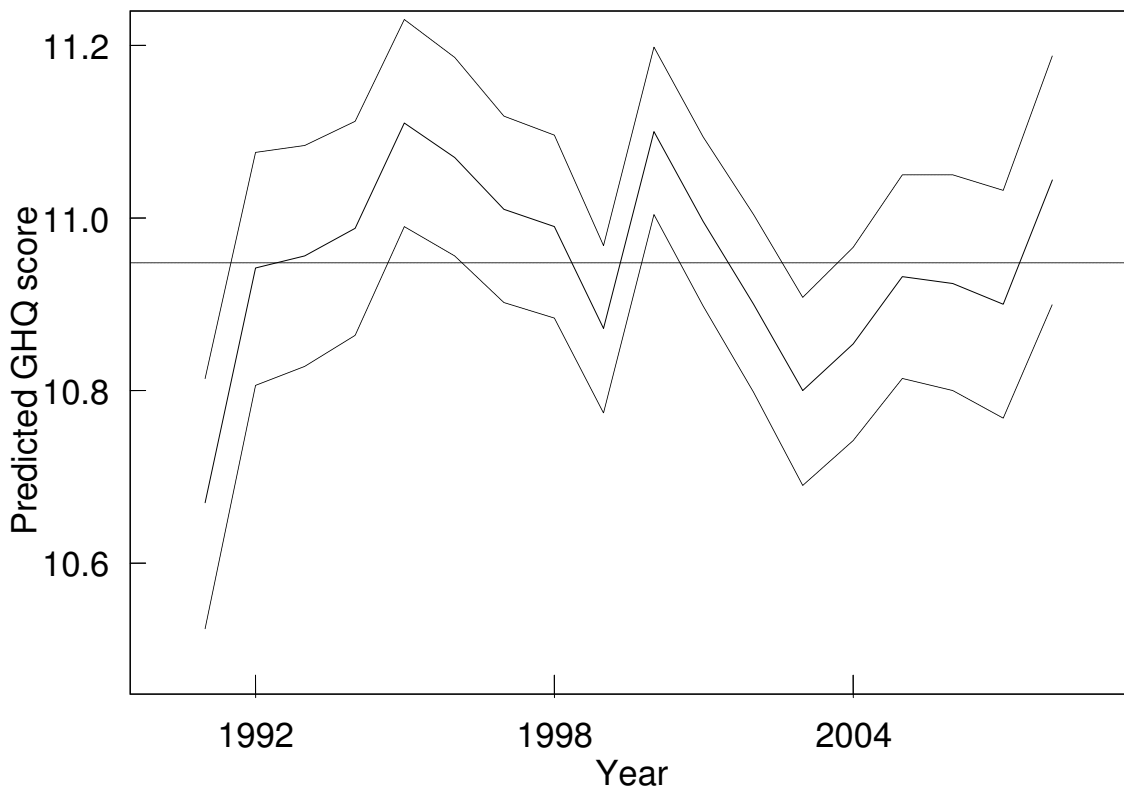
3a

3b

3c

3d

Figure-4: Conditional age effect on GHQ score, with individual-level coverage bounds. These show the extents to which individuals vary in their life-course (conditional on covariates) and should not be confused with confidence intervals in previous graphs (which show the uncertainty in the parameter estimates rather than variation in the sample.
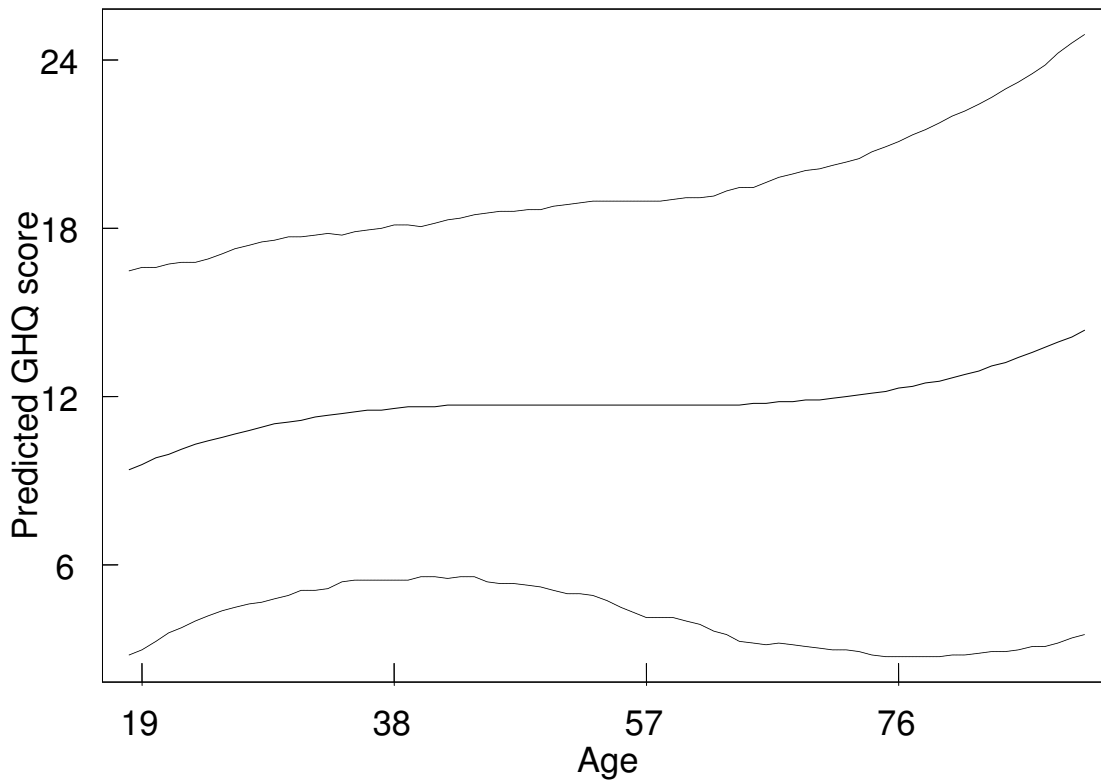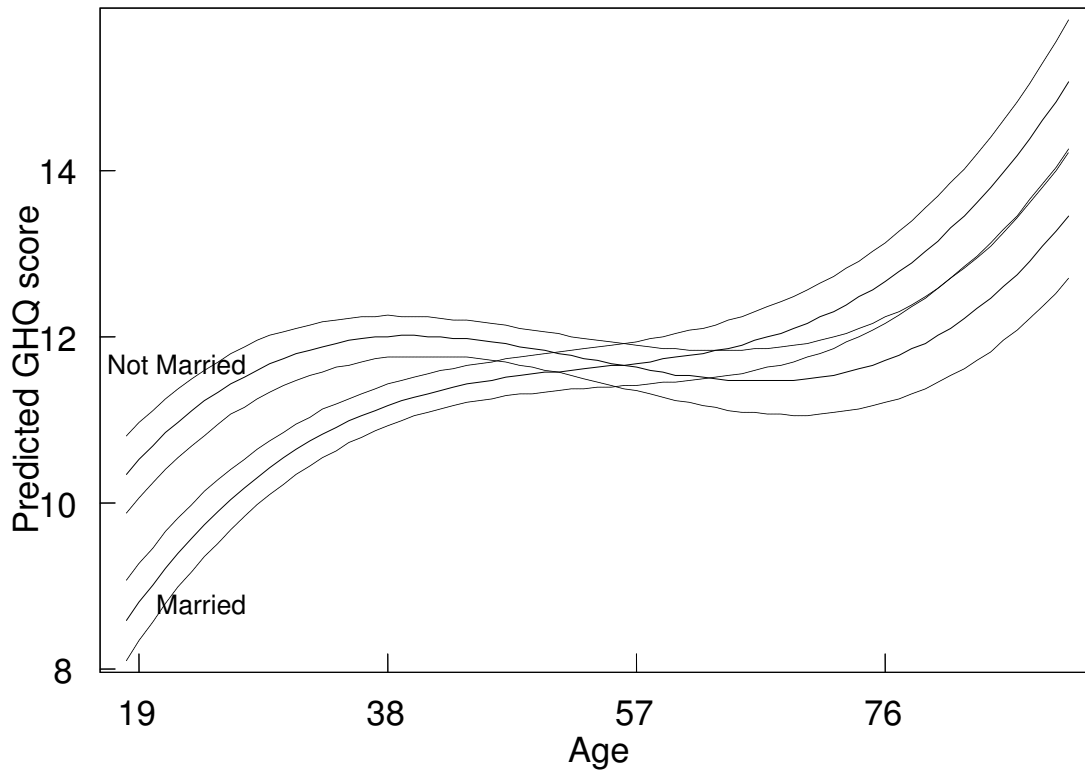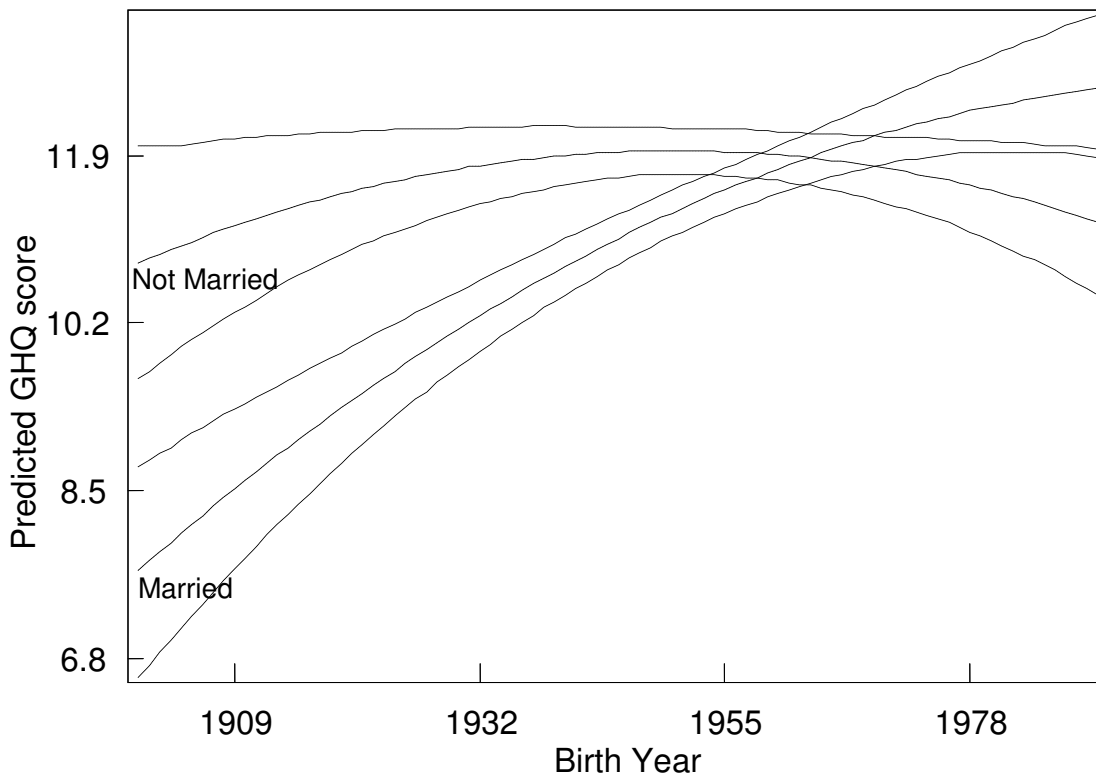

Figure-5: Interactions between covariates, and age/cohort. (a) The age effect for individuals who are married and not married, conditional on cohort and other covariates; (b) the cohort effect for individuals who are married and not married, conditional on age and other covariates; (c) the age effect for individuals with only primary education and more than primary education, conditional on cohort and other covariates; (d) the cohort effect for individuals who smoke and do not smoke, conditional on age and other covariates. Dotted lines are 95% confidence intervals.
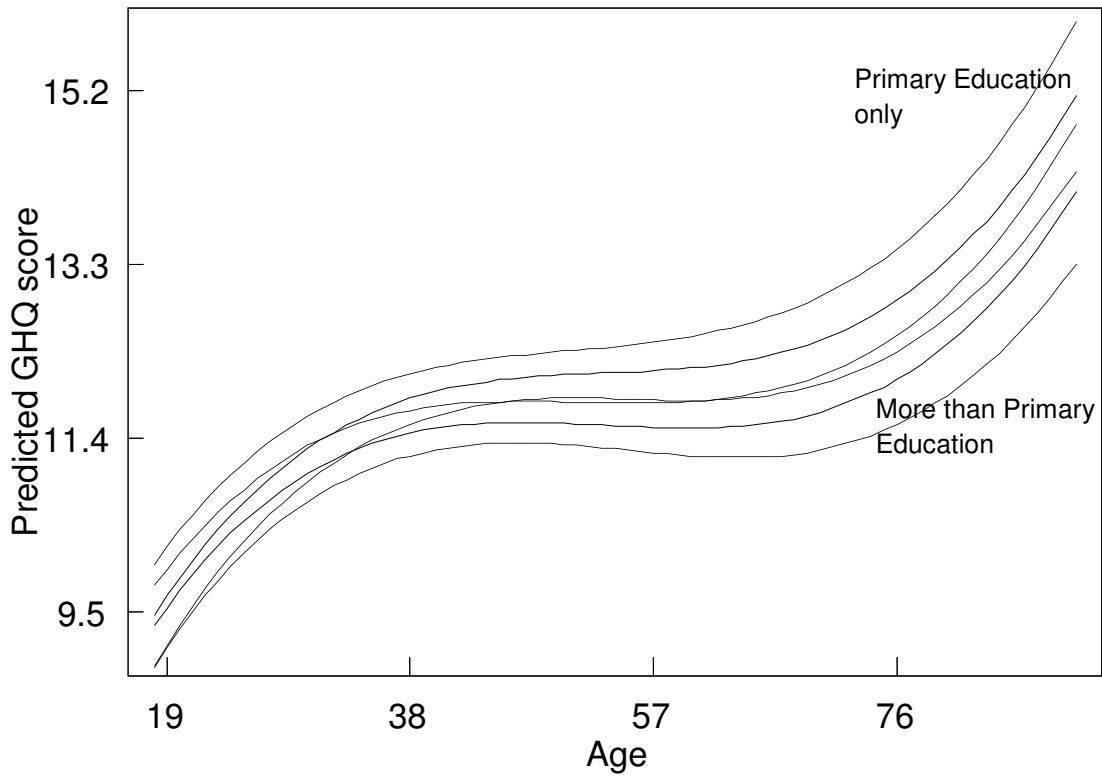
5a

5b

5c

5d

Appendix: Algebraic specifications of the seven models in table 2:

Model 1:

$$y_i = \beta_0 + \beta_{1i}Age_i + \beta_2 Age_i^2 + \beta_3 Age_i^3 + \beta_4 Female_i + \beta_5 Cohort_i + \beta_6 Cohort_i^2 + \beta_7(Female_i$$
$$* Age_i) + \beta_8(Female_i * Age_i^2) + \beta_9(Female_i * Age_i^3) + \beta_{10}(Female_i * Cohort_i)$$
$$+ \beta_{11}(Age_i * Cohort_i) + [u_{Individual} + e_i]$$
$$u_{Individual} \sim N(0, \sigma_{u(1)}^2), \qquad e_i \sim N(0, \sigma_e^2)$$

Model 2:

$$y_i = \beta_0 + \beta_{1i}Age_i + \beta_2 Age_i^2 + \beta_3 Age_i^3 + \beta_4 Female_i + \beta_5 Cohort_i + \beta_6 Cohort_i^2 + \beta_7(Female_i$$
$$* Age_i) + \beta_8(Female_i * Age_i^2) + \beta_9(Female_i * Age_i^3) + \beta_{10}(Female_i * Cohort_i)$$
$$+ \beta_{11}(Age_i * Cohort_i) + [u_{LAD} + u_{Household} + u_{Period} + u_{Cohort} + u_{Individual}$$
$$+ e_i]$$
$$u_{LAD} \sim N(0, \sigma_{u(5)}^2), \qquad u_{Household} \sim N(0, \sigma_{u(4)}^2), \qquad u_{Cohort} \sim N(0, \sigma_{u(3)}^2), \qquad u_{Period} \sim N(0, \sigma_{u(2)}^2),$$
$$u_{Individual} \sim N(0, \sigma_{u(1)}^2), \qquad e_i \sim N(0, \sigma_e^2)$$

Model 3:

$$y_i = \beta_0 + \beta_{1i}Age_i + \beta_2 Age_i^2 + \beta_3 Age_i^3 + \beta_4 Female_i + \beta_5 Cohort_i + \beta_6 Cohort_i^2 + \beta_7(Female_i$$
$$* Age_i) + \beta_8(Female_i * Age_i^2) + \beta_9(Female_i * Age_i^3) + \beta_{10}(Female_i * Cohort_i)$$
$$+ \beta_{11}(Age_i * Cohort_i) + \beta_{12}Class_i + \beta_{13}Urban_i + \beta_{14}NonWhite_i$$
$$+ \beta_{15}PrimaryEd_i + \beta_{16}NCigarettes_i + \beta_{17}Married_i + \beta_{18}Income_i + [u_{LAD}$$
$$+ u_{Household} + u_{Period} + u_{Cohort} + u_{Individual} + e_i]$$
$$u_{LAD} \sim N(0, \sigma_{u(5)}^2), \qquad u_{Household} \sim N(0, \sigma_{u(4)}^2), \qquad u_{Cohort} \sim N(0, \sigma_{u(3)}^2), \qquad u_{Period} \sim N(0, \sigma_{u(2)}^2),$$
$$u_{Individual} \sim N(0, \sigma_{u(1)}^2), \qquad e_i \sim N(0, \sigma_e^2)$$

Model 4:

$$y_i = \beta_0 + \beta_{1i}Age_i + \beta_2 Age_i^2 + \beta_3 Age_i^3 + \beta_4 Female_i + \beta_5 Cohort_i + \beta_6 Cohort_i^2 + \beta_7(Female_i$$
$$* Age_i) + \beta_8(Female_i * Age_i^2) + \beta_9(Female_i * Age_i^3) + \beta_{10}(Female_i * Cohort_i)$$
$$+ \beta_{11}(Age_i * Cohort_i) + \beta_{12}Class_i + \beta_{13}Urban_i + \beta_{14}NonWhite_i$$
$$+ \beta_{15}PrimaryEd_i + \beta_{16}NCigarettes_i + \beta_{17}Married_i + \beta_{18}Income_i + [u_{LAD}$$
$$+ u_{Household} + u_{Period} + u_{Cohort(0)} + (u_{Cohort(1)} * Female_i) + u_{Individual(0)}$$
$$+ (u_{Individual(1)} * Age_i) + e_i]$$

$$u_{LAD} \sim N(0, \sigma_{u(5)}^2), \qquad u_{Household} \sim N(0, \sigma_{u(4)}^2), \qquad \begin{bmatrix} u_{Cohort(0)} \\ u_{Cohort(1)} \end{bmatrix} \sim N\left(0, \begin{bmatrix} \sigma_{u3I}^2 & \\ \sigma_{u3Iu3S} & \sigma_{u3S}^2 \end{bmatrix}\right),$$

$$u_{Period} \sim N(0, \sigma_{u(2)}^2), \qquad \begin{bmatrix} u_{Individual(0)} \\ u_{Individual(1)} \end{bmatrix} \sim N\left(0, \begin{bmatrix} \sigma_{u1I}^2 & \\ \sigma_{u1Iu1S} & \sigma_{u1S}^2 \end{bmatrix}\right),$$

$$e_i \sim N(0, \sigma_e^2)$$

Model 5:

$$y_i = \beta_0 + \beta_{1i}Age_i + \beta_2 Age_i^2 + \beta_3 Age_i^3 + \beta_4 Female_i + \beta_5 Cohort_i + \beta_6 Cohort_i^2 + \beta_7(Female_i$$
$$* Age_i) + \beta_8(Female_i * Age_i^2) + \beta_9(Female_i * Age_i^3) + \beta_{10}(Female_i * Cohort_i)$$
$$+ \beta_{12}Class_i + \beta_{13}Urban_i + \beta_{14}NonWhite_i + \beta_{15}PrimaryEd_i$$
$$+ \beta_{16}NCigarettes_i + \beta_{17}Married_i + \beta_{18}Income_i + [u_{LAD} + u_{Household} + u_{Period}$$
$$+ u_{Cohort} + u_{Individual(0)} + (u_{Individual(1)} * Age_i) + e_i]$$

$$u_{LAD} \sim N(0, \sigma_{u(5)}^2), \qquad u_{Household} \sim N(0, \sigma_{u(4)}^2), \qquad u_{Cohort} \sim N(0, \sigma_{u(3)}^2), \qquad u_{Period} \sim N(0, \sigma_{u(2)}^2),$$

$$\begin{bmatrix} u_{Individual(0)} \\ u_{Individual(1)} \end{bmatrix} \sim N\left(0, \begin{bmatrix} \sigma_{u1I}^2 & \\ \sigma_{u1Iu1S} & \sigma_{u1S}^2 \end{bmatrix}\right), \qquad e_i \sim N(0, \sigma_e^2)$$

<u>Model 6:</u>

$$y_i = \beta_0 + \beta_{1i}Age_i + \beta_2 Age_i^2 + \beta_3 Age_i^3 + \beta_4 Female_i + \beta_5 Cohort_i + \beta_6 Cohort_i^2 + \beta_7(Female_i$$

$$* Age_i) + \beta_8(Female_i * Age_i^2) + \beta_9(Female_i * Age_i^3) + \beta_{10}(Female_i * Cohort_i)$$

$$+ \beta_{12}Class_i + \beta_{13}Urban_i + \beta_{14}NonWhite_i + \beta_{15}PrimaryEd_i$$

$$+ \beta_{16}NCigarettes_i + \beta_{17}Married_i + \beta_{18}Income_i + \beta_{19}(Age_i * Urban_i)$$

$$+ \beta_{20}(Cohort_i * Urban_i) + \beta_{21}(Age_i * Married_i) + \beta_{22}(Cohort_i * Married_i)$$

$$+ \beta_{23}(Age_i * Income_i) + \beta_{24}(Cohort_i * Income_i) + \beta_{25}(Age_i * PrimaryEd_i)$$

$$+ \beta_{26}(Cohort_i * PrimaryEd_i) + \beta_{27}(Age_i * NCigarettes_i) + \beta_{28}(Cohort_i$$

$$* NCigarettes_i) + [u_{LAD} + u_{Household} + u_{Period} + u_{Cohort} + u_{Individual(0)}$$

$$+ (u_{Individual(1)} * Age_i) + e_i]$$

$$u_{LAD} \sim N(0, \sigma_{u(5)}^2), \qquad u_{Household} \sim N(0, \sigma_{u(4)}^2), \qquad u_{Cohort} \sim N(0, \sigma_{u(3)}^2), \qquad u_{Period} \sim N(0, \sigma_{u(2)}^2),$$

$$\begin{bmatrix} u_{Individual(0)} \\ u_{Individual(1)} \end{bmatrix} \sim N\left(0, \begin{bmatrix} \sigma_{u1I}^2 & \\ \sigma_{u1Iu1S} & \sigma_{u1S}^2 \end{bmatrix}\right), \qquad e_i \sim N(0, \sigma_e^2)$$

<u>Model 7:</u>

$$y_i = \beta_0 + \beta_{1i}Age_i + \beta_2 Age_i^2 + \beta_3 Age_i^3 + \beta_4 Female_i + \beta_5 Cohort_i + \beta_6 Cohort_i^2 + \beta_7(Female_i$$

$$* Age_i) + \beta_8(Female_i * Age_i^2) + \beta_9(Female_i * Age_i^3) + \beta_{10}(Female_i * Cohort_i)$$

$$+ \beta_{12}Class_i + \beta_{13}Urban_i + \beta_{14}NonWhite_i + \beta_{15B}\overline{PrimaryEd_\iota}$$

$$+ \beta_{16W}(NCigarettes_i - \overline{NCigarettes_\iota}) + \beta_{17W}(Married_i - \overline{Married_\iota})$$

$$+ \beta_{18W}(Income_i - \overline{Income_\iota}) + \beta_{16B}\overline{NCigarettes_\iota} + \beta_{17B}\overline{Married_\iota}$$

$$+ \beta_{18B}\overline{Income_\iota} + \beta_{19}(Age_i * Urban_i) + \beta_{20}(Cohort_i * Urban_i) + \beta_{21W}(Age_i$$

$$* (Married_i - \overline{Married_\iota})) + \beta_{22W}(Cohort_i * (Married_i - \overline{Married_\iota}))$$

$$+ \beta_{21B}(Age_i * \overline{Married_\iota}) + \beta_{22B}(Cohort_i * \overline{Married_\iota}) + \beta_{23W}(Age_i * (Income_i$$

$$- \overline{Income_\iota})) + \beta_{24W}(Cohort_i * (Income_i - \overline{Income_\iota})) + \beta_{25B}(Age_i$$

$$* \overline{PrimaryEd_\iota}) + \beta_{26B}(Cohort_i * \overline{PrimaryEd_\iota}) + \beta_{27W}(Age_i * (NCigarettes_i$$

$$- \overline{NCigarettes_\iota})) + \beta_{28W}(Cohort_i * (NCigarettes_i - \overline{NCigarettes_\iota}))$$

$$+ \beta_{27B}(Age_i * \overline{NCigarettes_\iota}) + \beta_{28B}(Cohort_i * \overline{NCigarettes_\iota}) + [u_{LAD}$$

$$+ u_{Household} + u_{Period} + u_{Cohort} + u_{Individual(0)} + (u_{Individual(1)} * Age_i) + e_i]$$

$$u_{LAD}\sim N(0, \sigma_{u(5)}^2), \quad u_{Household}\sim N(0, \sigma_{u(4)}^2), \quad u_{Cohort}\sim N(0, \sigma_{u(3)}^2), \quad u_{Period}\sim N(0, \sigma_{u(2)}^2),$$

$$\begin{bmatrix} u_{Individual(0)} \\ u_{Individual(1)} \end{bmatrix} \sim N\left(0, \begin{bmatrix} \sigma_{u1I}^2 & \\ \sigma_{u1Iu1S} & \sigma_{u1S}^2 \end{bmatrix}\right), \quad e_i \sim N(0, \sigma_e^2)$$

In model 7, a line over the variable means the mean of the individual, such that $\overline{NCigarettes_\iota}$ is the individual mean of $NCigarettes_i$, etc. For the betas in model 7, a B in the subscript means a between effect is estimated, and a W represents a within effect. In all models, the random part is within square brackets at the end of the equation.