THE UNIVERSITY of York

Discussion Papers in Economics

No. 2001/15

The Dynamics of Health in British Households: Simulation-Based Inference in Panel Probit Models

by

Paul Contoyannis, Andrew M Jones and Nigel Rice

Department of Economics and Related Studies
University of York
Heslington
York, YO10 5DD

The dynamics of health in British households: Simulation-based inference in panel probit models *

Paul Contoyannis, Andrew M. Jones †
Department of Economics
and Related Studies,
University of York,
Heslington,
York YO10 5DD,
United Kingdom.

and Nigel Rice
Centre for Health Economics
University of York,
Heslington,
York YO10 5DD,
United Kingdom.

October 1, 2001

Abstract

The study of income dynamics, such as the analysis of transitions into and out of poverty, has a long history. The empirical analysis of health dynamics is less well established. This paper considers the determinants of a binary indicator for the existence of functional limitations using seven waves (1991-1997) of the British Household Panel Survey(BHPS). Our analysis has two focal points : 1) the relative contributions of state dependence, heterogeneity and serial correlation in explaining the dynamics of health and, 2) the investigation of the effects of exogenous variables, with a particular focus on educational attainment and long-run and short-run variations in income. To investigate these issues we apply static and dynamic panel probit models with flexible error structures. To estimate the probit models we use maximum simulated likelihood(MSL) with antithetic acceleration and implement a recently proposed test for the existence of asymptotic bias. The dynamic models show strong positive state dependence, with the effect for men around 150% of the effect for women.

JEL codes I1 C1

Keywords: Health dynamics, Health inequalities, Simulation-based inference, Binary choice panel data models

*Data from the *British Household Panel Survey*(BHPS) were supplied by the ESRC Data Archive. Neither the original collectors of the data nor the Archive bear any responsibility for the analysis or interpretations presented here. Funding was provided by the ESRC award no: R000238169-'Simulation-based econometric approaches to investigating the interaction of lifestyle and health'. The authors would like to thank Roberto Leon Gonzalez for valuable programming advice, and participants at the iHEA Third International conference, York, 22-25 July 2001 for their comments.

†Tel: +44-1904-433790; Fax: +44-1904-433759; Email: amj1@york.ac.uk

1 Introduction

The study of income dynamics, such as the analysis of transitions into and out of poverty, has a long history (see Jenkins(1998) for a review). The empirical analysis of health dynamics is less well established. This paper considers the determinants of a binary indicator for the existence of functional limitations using seven waves (1991/92-1997/98) of the British Household Panel Survey(BHPS).

The main objective of this paper is to analyse the dynamics of individual health. This is of interest because of the persistent inequalities in health outcomes revealed by the BHPS data. Specifically, suppose the occurence of illness was completely random, with the data generated by a binomial distribution with a constant probability of illness in every period, $p = 0.125$, (approximately the sample mean of the binary variable indicating functional limitations). In this case, 40% of men would be expected to be healthy in every period, while the predicted probability of an individual being ill in every period would be almost zero(4.8 e-7). In contrast the observed sample proportions translate to around 73% and 4% respectively. Similar deviations between observed sample proportions and those predicted by a binomial model are found for women. Letting $p = 0.15$(the sample mean for women), around 32% of women would be expected to be healthy in every period, while a negligible proportion(1.7 e-6) would be ill in every period. In contrast, the observed proportions translate to 65% and 4 % respectively. This paper aims to decompose this observed persistence into components due to state dependence,serial correlation and unobserved heterogeneity.

A second objective of the paper is to consider the relationship between health and income. In particular we investigate whether and how health responds differently to long and short-run changes in household income. This is of particular interest in the context of the recent focus on the different impacts of transitory and permanent poverty and deprivation on health (e.g Benzeval*et al.* (2000)). From this point on we refer to these variations as 'permanent' and transitory'. If transitory income variations are found to be unimportant relative to permanent effects in explaining health outcomes, the focus of (health) policy interventions can ignore these variations, and if the permanent effect is sufficiently large, focus resources on those households who suffer persistent deprivation. Previous analyses of this issue using the BHPS (e.g Benzeval *et al.*(2000)) have employed simple empirical models and measures of income which have not fully exploited the panel dimension of the data. The empirical models used here allow for persistent unobservable effects and

correlations in the transitory error components and make full use of the outcome information contained in the dataset.

Previous literature concerning health dynamics has considered the relationship between health and schooling (e.g. Grossman(2000)). A third objective of this paper is to analyse whether the dynamics of health vary with levels of education. This is of particular relevance when considering interventions to alter health paths. To investigate this issue we estimate the empirical models after splitting the data by both gender and the highest academic qualification attained at the beginning of the survey. By conditioning on previous health outcomes we are also able to reduce fears of bias due to reverse causality.

There are a number of innovations in our empirical approach. Firstly, we consider two approaches to dealing with the problem of initial conditions in models with unobserved effects and lagged dependent variables. This problem is due to the generic feature that the starting point of a survey is not the beginning of a process, and that individuals inherit different unobserved and time invariant characteristics which affect outcomes in every period. These phenomena lead to endogeneity bias in dynamic models with covariance structures that are not diagonal. Here we consider the approaches of Wooldridge(2000) who considers random effects models which can be implemented with standard software, and Heckman(1981), which is relatively difficult to implement, but can more easily accommodate more flexible error structures.

Secondly, we allow for more flexible error structures than previously considered in dynamic empirical models of health outcomes. To estimate models with more complex error structures, we use maximum simulated likelihood(MSL) with antithetic acceleration and implement a recently proposed test for the existence of asymptotic bias due to simulation.

The structure of the paper is as follows. In section 2 we review some of the previous literature on health dynamics. In section 3 we introduce the empirical models and estimation strategy. Section 4 introduces the BHPS data and describes the samples we use for estimation. Section 5 outlines our results, while a short conclusion is provided in section 6.

2 Context

The starting point for an economic analyis of health dynamics is the household production model of Grossman(1972).[1] In its discrete time version, health evolves according to a difference equation where current health is equal to the undepreciated component of health in the previous period plus investment in health in the previous period. The theoretical model assumes that investment in health is produced by combining market goods and time. Given an intertemporal utility function and budget and time constraints, an optimality criterion is found for each period. When this criterion is satisfied in every period, the implied health path gives the optimal path for desired health. Differences in health paths are wholly deterministic and are due to differences in the parameters that define the optimality criterion. For example, increases in education are assumed to increase the efficiency of investment in health, so that more health is demanded by those that are more highly educated due to a decrease in its shadow price. Similarly, an increase in the wage rate, will, in general, lead to an increase in the demand for health by increasing the marginal rate of return on health capital. Ageing is assumed to lead to an increase in the rate of depreciation which leads to a reduction in the marginal rate of return for a given amount of investment in health and a reduction in the level of desired health.

Grossman derived a reduced form version of the demand for health equation as a function of age, wages, education, the prices of inputs in the investment function and the depreciation rate in the initial period. However, this empirical function relies on instantaneous adjustment to the desired health stock. Under a partial adjustment mechanism, due, for example, to adjustment costs, current health will also depend on previous health, and this model can be estimated using longitudinal data. This formulation was used by van Doorslaer(1987), Wagstaff(1993) and Salas(2000), and can be derived by using the identity outlined above in combination with a model for gross investment.

3 Models and Estimation Strategy

We include previous values in our dynamic empirical models and suggest that these may be viewed as approximating partial adjustment mechanisms. Our models should also be viewed as reduced form specifications as they do not include objects of choice such as medical care or other health inputs such as lifestyle.

[1] This model, with extensions and empirical applications, are reviewed in Grossman(2000) and it is not discussed in detail here.

The most general (reduced form) model that we estimate can be written as:

$$h_{it} = 1(h^*_{it} > 0) = 1(X'_{it}\beta + \gamma h_{it-1} + u_{it} > 0) \quad (i = 1, ..., N; t = 1, ...T) \qquad (1)$$

where 1(.) is a binary indicator function equal to one if the argument is true and zero otherwise. X_{it} is a set of observed variables which may affect the health indicator h_{it} but which are uncorrelated with the error term. In the dynamic models γ is a parameter to be estimated, while the static models restrict $\gamma = 0$. For most of the paper we focus on models which restrict the covariance matrix:

$$h_{it} = 1(X'_{it}\beta + \gamma h_{it-1} + \alpha_i + \epsilon_{it} > 0) \quad (i = 1, ..., N; t = 1, ..., T) \qquad (2)$$

Here α_i is an individual specific and time-invariant random component, assumed to come from a normal distribution with mean zero and variance σ^2_α. ϵ_{it} is a time and individual specific error term uncorrelated with X_{it} and α_i and across individuals, but which may be serially correlated. In particular we consider that the error process may be of the AR(1) form: $\epsilon_{it} = \rho\epsilon_{it-1} + \eta_{it}$. η_{it} is also assumed to be independently and identically normally distributed with mean zero and variance σ^2_η.

While our goal is to estimate the relative impacts of heterogeneity, σ^2_α, state dependence, γ, and autoregressive error structures, ρ, we begin by estimating models which do not include the lagged variables and then introduce state dependence.

3.1 Static Models

We use a number of alternative estimation strategies.[2] Firstly, and as a baseline against which to compare alternative estimators, we estimate a pooled probit model by maximum likelihood(ML). For the likelihood on which this estimator is based to be correct, it is necessary to assume that the sampled data points are completely independent: $E(u_{it}, u_{is}) = 0 \; \forall \; t \neq s$. This allows for neither heterogeneity nor serial correlation in ϵ_{it}. However, while the likelihood may be incorrect, it has been shown that the pooled probit (pseudo)-ML estimator for β is consistent irrespective of whether the assumed error structure is correct(Robinson(1982)).[3] In addition, this estimator has two attractive features: 1) It is computationally simple and fast, as it

[2] The total error variance in each period is constrained to equal one throughout.

[3] Guilkey and Murphy(1993) present Monte Carlo evidence which suggests that the pooled probit ML estimator performs well when the the error structure is misspecified provided that a robust estimator, consistent in the presence of heteroskedasticity and auto-correlation, is used for the covariance matrix of $\hat{\beta}$.

only requires (repeated) computation of the standard normal CDF to construct the likelihood. 2) It allows us to consistently test whether sub-samples of the data come from different populations. This is not true of the ML estimator for the random effects probit or more complex models. Unfortunately, the pooled probit estimator does not give consistent estimates of the other parameters of interest, ρ and σ_α^2, as they are both restricted to equal zero.

The random effects probit estimator is employed in the case where $\rho = 0$ and σ_α^2 is a parameter to be estimated. Given normality it is feasible to numerically integrate over the distribution of the time-invariant random effects to obtain a good approximation to the likelihood using Gauss-Hermite quadrature(see e.g Butler and Moffitt(1982) and Hyslop(1999)). To see the approach of Butler and Moffitt(1982) and to motivate the simulation-based approaches introduced later, let:

$$h_{it}^* = X_{it}'\beta + \alpha_i + \eta_{it}, \quad (i = 1, \ldots, N; t = 1, \ldots, T) \tag{3}$$

Then, the probability of observing the sequence $h_{i1}, \ldots, h_{iT}$ for a particular individual is:

$$Prob(h_{i1}, \ldots, h_{iT}) = \int_{a_{i1}}^{b_{i1}} \cdots \int_{a_{iT}}^{b_{iT}} f(u_{i1}, \ldots, u_{iT}) \mathrm{d}u_{iT}, \ldots \mathrm{d}u_{i1} \tag{4}$$

with $a_{it} = -X_{it}'\beta, b_{it} = \infty$ if $h_{it} = 1$ and $a_{it} = -\infty, b_{it} = -X_{it}'\beta$ if $h_{it} = 0$. Conditioning on the permanent component α_i allows us to write:

$$Prob(h_{i1}, \ldots, h_{iT}) = \int_{a_{i1}}^{b_{i1}} \cdots \int_{a_{iT}}^{b_{iT}} \int_{-\infty}^{\infty} f(u_i|\alpha_i) f(\alpha_i) \mathrm{d}\alpha_i \mathrm{d}u_{iT}, \ldots, \mathrm{d}u_{i1} \tag{5}$$

where $u_i = (u_{i1}, \ldots, u_{iT})$. This can also be written as:

$$Prob(h_{i1}, \ldots, h_{iT}) = \int_{a_{i1}-\alpha_i}^{b_{i1}-\alpha_i} \cdots \int_{a_{iT}-\alpha_i}^{b_{iT}-\alpha_i} \int_{-\infty}^{\infty} f(\eta_i|\alpha_i) f(\alpha_i) \mathrm{d}\alpha_i \mathrm{d}\eta_{iT}, \ldots, \mathrm{d}\eta_{i1} \tag{6}$$

where $\eta_i = (\eta_{i1}, \ldots, \eta_{iT})\prime$. Further, since the conditional density of $\eta_i|\alpha_i$ is multivariate normal, independent of α_i and has a diagonal covariance matrix:

$$Prob(h_{i1},\ldots,h_{iT}) = \int_{-\infty}^{\infty} \prod_{t=1}^{T} \left[\Phi\left(\frac{b_{it}-\alpha_i}{(1-\sigma_\alpha^2)^{1/2}} \right) - \Phi\left(\frac{a_{it}-\alpha_i}{(1-\sigma_\alpha^2)^{1/2}} \right) \right] f(\alpha_i) \mathrm{d}\alpha_i \quad (7)$$

This expression can easily be manipulated into the desired form for approximation by Gauss-Hermite quadrature. Unfortunately, and unlike the pooled probit estimator, the random effects estimator is not robust to misspecification of the covariance matrix (e.g $\rho \neq 0$), and is therefore not as useful as the pooled probit estimator in testing coefficient equality. Furthermore, given the constraints imposed on the model, serial correlation is ruled out. However, it is useful as a benchmark to assess the accuracy of the simulation-based approach.[4]

The problem for estimation of a general LDV model with more than 3 or 4 dimensions can be illustrated by examining the procedure of Butler and Moffitt(1982) outlined above. In essence, the conditioning used to obtain (7) as a uni-dimensional integral is no longer available: the conditional probability in (6) does not factorize if, for example, there is serial correlation in the error term. In this context, standard and computationally feasible numerical methods provide unsatisfactory approximations unless the dimensionality of the integral, T, is small.[5] Simulation-based estimation procedures have been developed to deal with situations where numerical approximations are expected to perform poorly.

Simulation-based estimation procedures replace functions(usually definite integrals) which are computationally intractable using numerical or analytical methods, by random approximations(simulators) for these functions. The simulators are generally sample averages obtained by drawing pseudo-random samples from an appropriate distribution and evaluating a known function at these sample points.[6] There

[4] An alternative estimator is based on generalized estimating equations(GEE)(see e.g. Pendergast et al. (1996)). The GEE approach is highly flexible allowing for general correlation structures and alternative representations of non-linearity. However, in non-linear models the random effects and GEE estimators identify two different parameters, with the GEE approach estimating 'population average' parameters while the random effects maximum likelihood estimator estimates 'cluster specific' parameters(see Pendergast et al. *op cit* and Neuhaus and Jewell(1993)). Nevertheless, a useful feature of the GEE approach is the ability to estimate models with unrestricted correlation matrices without computational difficulty. Results for this estimator are not presented here, but are available on request from the authors.

[5] Hajivassiliou and Ruud(1994) give numerous examples of the 'curse of dimensionality' in microeconometric models.

[6] Stern(1997) provides an accessible introduction and numerous references related to simulation-based estimation. Lerman and Manski(1981) provided the original work on simulation-based estimation in frequentist econometrics although Kloek and van Dijk(1978) used simulation techniques to estimate a Bayesian simultaneous equations model.

a number of generic approaches to the estimation of LDV models by simulation. In this application we use the method of maximum simulated likelihood(MSL). MSL has been used in the context of the binary panel probit model by Keane(1994) and Hyslop(1999) who also provide the results of Monte-Carlo experiments. These provide support for the use of this approach, given sufficient replications.

MSL is a conceptually simple extension of MLE: instead of forming the log-likelihood through analytical or numerical methods, the log-likelihood is simulated and then maximized to obtain MSL estimates of the model parameters.[7] Following Hyslop(1999), let the log-likelihood for the unknown parameters as a function of the data be:

$$l_N(\theta) = \sum_{i=1}^{N} ln(l(\theta; h_i, X_i)) \tag{8}$$

where $l(\theta; h_i, X_i)$ is equivalent to (4). Then let $\xi_i = \xi_{i1}, \ldots, \xi_{iR}$ be a sequence of primitive simulated values independent of the parameters of the model and the data.[8] We can then write:

$$\tilde{l}(\theta; h_i, X_i, \xi_i) = (1/R) \sum_{r=1}^{R} \tilde{l}(\theta; h_i, X_i, \xi_{ir}) \tag{9}$$

where $\tilde{l}(\theta; h_i, X_i, \xi_{ir})$ is an unbiased simulator for $l(\theta; h_i, X_i)$ and R is the number of simulation replications. The MSL estimator of θ is then defined as:

$$\widehat{\theta_{MSL}} = argmax(\theta) \sum_{i=1}^{N} ln\tilde{l}(\theta; h_i, X_i, \xi_i) \tag{10}$$

Implementation of MSL estimation requires a simulator for the probabilities that enter the log-likelihood function. There are many alternatives available for the simulation of multivariate normal rectangle probabilities(e.g. Hajivassiliou et al.(1996) and Vijverberg(1997)). Given the current Monte-Carlo evidence (e.g. Hajivassiliou et al.(1996) and Vijverberg(1997)), and the theoretical properties derived by Borsch-Supan and Hajivassiliou (1993), the leading simulator is the Smooth Recursive Sim-

[7] We prefer the acronym MSL rather than Simulated Maximum Likelihood(SML) as it describes the actual estimation procedure.

[8] These primitive values are independent pseudo-random draws from a $U[0, 1]$ distribution.

ulator (SRC), or Geweke-Hajivassiliou-Keane (GHK) simulator. This simulator is strictly bounded by zero and one, smooth in the parameters (conditional on a smooth method to obtain the inverse of the truncated normal CDF), unbiased, and consistent in the number of replications R. Monte-Carlo evidence also shows that it has low variance.[9]

The importance of these properties is clear when one considers the impact of simulation on the log-likelihood function. Given the logarithmic transformation, unbiasedness of a simulator is insufficent to obtain an unbiased simulator of the log-likelihood. Furthermore, the nonlinearity of the estimator of θ as a function of the log-likelihood means that unbiased simulation of the log-likelihood is not sufficient to obtain an unbiased estimator of θ. This is a familiar result for the theory of maximum likelihood estimation for nonlinear models. However, consistency can be obtained by reducing the error of the simulated sample log-likelihood to zero, as $R \to \infty$, at a sufficient rate with N. For a finite variance and unbiased probability simulator, as the number of replications grows the bias and variance of the approximation to the sample log-likelihood approach zero, but the variance must reduce at a sufficient rate to avoid asymptotic bias in the limiting distribution of $\widehat{\theta_{MSL}}$. Hajvassiliou and Ruud(1994) show that a sufficient rate for this is $R/\sqrt{N} \to \infty$ as $N \to \infty$. Furthermore, they show that this rate is also sufficient for MSL to be asymptotically efficient such that no correction is required for the covariance matrix relative to that obtained for maximum likelihood: 'Given enough simulations to overcome bias, there are enough simulations to make the asymptotic contribution of simulation to the limiting distribution of $\widehat{\beta_{MSL}}$ negligible.' (Hajivassiliou and Ruud(1994))[10]

One of the practical implications of the above results is that a low-variance simulator can reduce the number of replications required to obtain a given level of bias. In this application we implement a test proposed by Hajivassiliou(2000) for the sufficiency of the number of replications to reduce bias to a level which is dominated by the variance of the MSL estimator. This test is described in appendix A.

To further reduce the variance of the simulators we use antithetic acceleration.

[9] Good descriptions of the SRC simulator for the panel probit model are available in Hajivassiliou(1994), Hyslop(1999) and Inkmann(2000) and it is not outlined here.

[10] The main non-Bayesian competitors to MSL are the Method of Simulated Moments(MSM)(McFadden(1989)), and the Method of Simulated Scores(MSS)(Hajivassiliou and McFadden(1998)). Hyslop(1999) and Hajivassiliou(2000) discuss the relative merits of the MSL,MSM and MSS estimators noting that the principle advantage of the MSM and MSS is their ability to obtain consistent estimators with a finite number of replications. However, their computational disadvantages can be severe. These and other issues are discussed by Contoyannis(2000). The availability of a formal statistical test(Hajivassiliou,2000) for the sufficiency of the number of replications in the MSL context further diminishes the relative standing of the MSM and MSS estimators.

These simulators use the original set of uniform random draws along with their reflections to estimate the probability of the observed sequence for each individual:

$$\tilde{l}(\theta; h_i, X_i, \xi_i) = (1/2R) \sum_{r=1}^{2R} \tilde{l}(\theta; h_i, X_i, \xi_{ir}) \tag{11}$$

where $\xi_i = \xi_{i1}, \ldots, \xi_{i2R}$ and $\xi_j = -\xi_{j-R}$ for $j = R+1, \ldots, 2R$.[11] Hajivassiliou(2000) presents Monte-Carlo evidence suggesting that the antithetically accelerated simulator for multivariate normal rectangle probabilities is superior to the standard SRC simulator.

For the static models we also estimate (1) with an unrestricted correlation matrix where $u_i = (u_{i1}, ..., u_{iT})'$ is assumed to have a multivariate normal distribution with mean zero and covariance matrix Ω, with the only restriction on the matrix being that the variance terms are set equal to one for identification purposes.[12]

3.2 Dynamic Models

For the dynamic models we estimate models that allow for state dependence such that the lagged binary dependent variable affects the current probability of observing functional limitations. We consider two error structures. Firstly, a RE process with no serial correlation in the residual error term, and then a RE+AR(1) process. This

[11] Some authors have implemented either first order or second order bias corrections(e.g Munkin and Trivedi(1999)). We prefer to use the test statistic as it is informative of the magnitude of the bias and non-rejection of the null hypothesis indicates the sufficiency of R conditional on N large. While antithetic acceleration and the test we implement may equally well be applied to any simulation estimator for which the full data generating process is available(e.g. in conjunction with the bias-corrected MSL estimators) the computational requirements of the bias-corrected estimators are greater for a given number of replications. It is however possible that the bias-corrected estimators may reach a given level of bias relative to the standard error of the estimator with fewer replications and a lower computation time.

[12] In fact, with the β parameters assumed to be time invariant, it is only required that one variance term be restricted in the general case. However, the imposition of more structure, such as the variance of the random effect being specified as a constant proportion of the total error variance over time as we use here, leads to the variances being equal in every time period. For an analysis of the effects of incorrectly imposing the homoskedasticity assumption in a panel probit model see Inkmann(2000). Inkmann(2000) presents Monte Carlo evidence which suggests that a Generalized Method of Moments(GMM) estimator based on nonparametric estimation of the optimal instruments(Bertschek and Lechner(1998), outperforms the MSL estimator under misspecification in terms of false assumptions on the variance covariance matrix. However, the increase in performance is marginal and comes at the cost of a reduction in efficiency if the structure is as assumed. It is also difficult to envisage why the variance of the overall error term should vary over time in our context and we thus restrict the variances equal to one in every period. This also eases comparability with other results as all estimators of β have the same scaling. The GMM estimator above has not been developed for the dynamic case, while, as discussed in section 3.2, the MSL approach can obtain consistent estimators for models including lagged dependent and latent variables.

sequence allows us to observe whether and how the parameter estimates vary with restrictions on the error and probability processes.

State dependence, random effects and serial correlation

Heckman (1981) recognizes two assumptions that are typically invoked concerning a discrete time stochastic process with a binary outcome, and suggests alternative ways of dealing with the initial conditions problem when these are untenable. The first assumption is that the initial observations are exogenous variables. This is invalid when the error process is not serially independent and the first observation is not the true initial outcome of the process. In our case, the latter condition is violated, while the former is unlikely to be correct. Treating the lagged dependent variables as exogenous when these assumptions are incorrect leads to inconsistent estimators. The second assumption often invoked is that the process is in equilibrium such that the marginal probabilities have approached their limiting values and can be therefore be assumed time-invariant. This assumption is untenable when non-stationary variables such as age and time trends are included in the model as we do here. However, Heckman (1981) suggests two alternative ways of obtaining consistent estimators for small T and large N.

One approach is to construct the exact joint distribution of the outcomes including the initial observation, conditional on the exogenous variables and unobserved effects and then integrate over the distribution of the unobserved effects, as suggested in the previous section for static models with random effects. The unobserved effects are usually assumed normally distributed and uncorrelated with the exogenous variables. However, this assumption is easily relaxed. Unfortunately, this exact solution is not possible in general as it requires knowledge of the true initial conditions, and knowledge, or the ability to estimate the joint distribution of, the pre-sample observations of the exogenous variables.

The second suggested approach, and one which we employ, is to approximate the reduced form marginal probability of the initial observed outcome using a probit model while allowing the error terms in the initial period to be freely correlated with the error terms in all othr periods.[13] The regressors in this model are the exogenous variables in the first period with no restrictions imposed on the parameters of the first period index function and that of later periods. The approximate nature of this solution is clear from this lack of restrictions. The likelihood function to be

[13] The variance of the error term in the initial period is also restricted to equal one.

maximized is then constructed as the product of the first period marginal probability and the joint probability of the future values conditional on the first observation. Heckman (1981), find that this approximation works well for the RE case using exact ML, while Hyslop (1999) using MSL for the RE+AR(1) case, finds that the approximation works well *if* the bias due to simulation has been overcome. Here we implement this approximation for both the RE and RE+AR(1) cases using MSL. This is performed by using the SRC(GHK) simulator with antithetic acceleration to approximate the multivariate normal probabilities implied by the model. In this case however, the correlation structure is parameterised by $T-1$ more parameters while the parameters and variables for the first period index function differ from those for other periods.

Wooldridge(2000) has suggested an alternative approach to dealing with the initial conditions problem in non-linear, dynamic, random effects models. Rather than specifying or approximating the conditional distribution of the initial observation, Wooldridge(2000) suggests modelling the distribution of the unobserved effect conditional on the initial value and any exogenous explanatory variables. This conditional maximum likelihood (CML) approach results in a likelihood function based on the joint distribution of the observations, exclusive of, but conditional on, the initial observations and all periods of the exogenous variables. A benefit of the CML approach relative to the approximate method described above is that, in the dynamic random effects probit case, a parameterisation of the distribution of the unobserved effects can be found which leads to a likelihood function easily maximized using pre-programmed commands with standard software (e.g. STATA).[14]

We implement this approach using the suggestion of Wooldridge(2000) by parameterizing the distribution of the individual effects as normal:

$$\alpha_i = \alpha_0 + \alpha_1 y_{i0} + \bar{x}_i \alpha_2 + a_i, \tag{12}$$

where $\bar{x}_i$ is the average over the sample period of the observations on the exogenous variables and a_i is distributed $N(0, \sigma_\alpha^2)$ and independently of these variables and the initial condition. This approach leads to the density to be maximized having a random effects structure with the regressor vector at time t augmented to include

[14] While this parameterization is not restricted by the model for the outcomes, the CML approach does specify a complete model for the unobserved effects and may therefore be sensitive to misspecification. This feature is shared by the approximate approach we employ, as a model for the unobserved effects is required for construction of the unconditional joint density. In probit models with unobserved effects this is a requirement to obtain consistent estimators for fixed T.

y_{i0} and $\bar{x}_i$. Three features should be noted. Firstly, this specification implies that the identified effects of all time-invariant characteristics are composite effects of the relevant elements of β and α_2. Secondly, all time dummies must also be dropped from $\bar{x}_i$ to avoid perfectly collinearity with one another and with the constant term. Thirdly, the estimate of α_1 is also of interest as it is informative about the relationship between α_i and y_{i0}. We would expect this to be positive in general. We implement this approach for the random effects model, noting that while it provides a useful comparator for the simulation-based approach, we would not expect parameter estimates from this approach and that of Heckman(1981) to converge as the number of replications gets large. Unlike the static case, where the RE estimator using quadrature provides a benchmark for the accuracy of MSL, the underlying functions of the data that are maximized using the approaches of Heckman(1981) and Wooldridge(2000) are different.[15]

4 The Data

In estimating the models we exploit the panel data available in the British Household Panel Study(BHPS). This consists of seven waves(1991-1997) and includes rich information on occupational, socio-demographic and health variables. The BHPS is a longitudinal survey of private households in Great Britain(England, Wales, and Scotland), and was designed as an annual survey of each adult (16+) member of a nationally representative sample of more than 5,000 households, with a total of approximately 10,000 individual interviews. The first wave of the survey was conducted between 1st September 1990 and 30th April 1991. [16] The same individuals are re-interviewed in successive waves and, if they split off from their original households are also re-interviewed along with all adult members of their new households. In this analysis we use a balanced sample of respondents for whom information on all the required variables is reported at each wave.

After excluding missing values due to attrition and item non-response, we obtain a working sample of $N = 6,106$ individuals($42,742$ observations). Descriptive statistics and variable definitions for the sample are given in Table1.

[15] We do not consider using this approach for the RE+AR(1) case, as while it can be extended, the estimator will require simulation and further restrictions, and it does not therefore maintain its advantages over the approach of Heckman(1981).

[16] The initial selection of households for inclusion in the survey was performed using a two-stage stratified systematic sampling procedure designed to give each address an approximately equal probability of selection. For further details see Taylor(1998) .

Table 1: Variable definitions and sample means

		FULL SAMPLE $N = 6106$ $NT = 42742$
HLLTYES	1 if health limits daily activities compared to others of same age, 0 otherwise	.140
WIDOWED	1 if widowed, 0 otherwise	.082
NVRMAR	1 if never married, 0 otherwise	.146
DIVSEP	1 if divorced or separated , 0 otherwise	.069
OTHETH	1 if a member of ethnic group other than white, 0 otherwise	.027
DEGHDEG	1 if highest academic qualification is degree or higher degree, otherwise	.108
HNDALEV	1 if highest academic qualification is HND or Alevel, 0 otherwise	.224
OCSE	1 if highest academic qualification is Olevel or CSE, 0 otherwise	.289
HHSIZE	Number of people in household including respondent	2.802
NCH04	Number of children in household aged 0-4	.152
NCH511	Number of children in household aged 5-11	.268
NCH1218	Number of children in household aged 12-18	.184
MEANIN	Annual household income in pounds	21510.97
AGE	Age in years at 1st december of current wave	46.667

The health indicator (HLLTYES) is defined by a binary response to the question: 'Does your health in any way limit your daily activities compared to most people of your age?' HLLTYES should therefore be interpreted as an indicating a deviation from 'average' health at a given age.[17] Income is measured by the individual specific sample mean of annual household income(MEANIN) and by the ratio of current income to this sample mean(DEVINC). Both of these variables are transformed to natural logarithms to allow for concavity of the health-income relationship. Throughout the discussion these (transformed) variables are referred to as *permanent* and *transitory* income respectively.[18] Other variables included are marital status (WIDOWED,NVRMAR,DIVSEP) and the highest educational qualification attained by the end of the sample period in descending order of attainment (DEGHDEG,HNDALEV,OCSE).[19] We include an indicator of ethnic origin(OTHETH), the number of individuals living in the household including the respondent(HHSIZE), and the numbers of children living in the household at different ages (NCH04, NCH511, NCH1218). These variables may affect health directly, but the primary reason for their inclusion is to control for household composition effects. Thus, we estimate the effect of income conditional on these variables. This is an alternative to using an equivalence scale to standardize for household composition before estimation. Age is included as a fourth-order polynomial,$(AGE, AGE2 = AGE^2/100, AGE3 = AGE^3/10000, AGE4 = AGE^4/1000000)$, and a vector of time

[17] However, cohort effects and reporting bias, potentially due to misinterpretation of the question, may remain. In any case, we condition on a quartic function of age in the empirical analysis.

[18] We use the terms permanent and transitory income as a shorthand for the variables defined above. These should not be confused with the concepts of permanent income and deviations from permanent income as commonly used in the intertemporal consumption literature.

[19] Married or living as a couple(MARCOUP) is the excluded category for marital status. Similarly, NOQUAL(No academic qualifications) is excluded for the educational variable.

dummies are included to account for aggregate health shocks, time-varying reporting changes, and any effects of age which are not captured by the polynomial.

Tables 2 and 3 present sample means for men and women respectively. In order to obtain a parsimonious and informative description of the samples we select sub-samples of the data based on the sequences of outcomes.[20] The first column of each table presents the sample means for the full samples of men and women. The second column contains information on those who were 'healthy'(HLLTYES=0) for all seven periods, while the third column describes the data for those who were 'ill'(HLLTYES=1) for all seven waves. The remaining columns present data for those who made transitions in their health status over the sample period. The fourth column presents results for those who made a single move from illness(i.e 1000000,1100000,...,1111110), while the fifth column presents results for those who made a single move to illness(i.e 0111111,0011111,...,0000001). The sixth column contains information on those who made multiple moves(e.g. 1010000, 1101010). Columns 2-6 are thus based on sub-samples of the data which are mutually exclusive and exhaustive of the sample space.

Table 2: Variable means by sub-sample- MEN

	FULL $N = 2715$ $NT = 19005$	Ill 0 yrs $N = 1977$ $NT = 13839$	Ill 7 yrs $N = 103$ $NT = 721$	single move from illness $N = 66$ $NT = 462$	single move to illness $N = 181$ $NT = 1267$	multiple moves $N = 388$ $NT = 2716$
HLLTYES	.124	0	1	.320	.400	.361
WIDOWED	.033	.025	.085	.065	.036	.052
NVRMAR	.179	.190	.110	.184	.138	.166
DIVSEP	.053	.049	.093	.045	.061	.062
OTHETH	.029	.028	.019	.076	.033	.028
DEGHDEG	.128	.147	.049	.106	.055	.093
HNDALEV	.272	.298	.097	.197	.204	.227
OCSE	.141	.281	.165	.136	.204	.224
HHSIZE	2.85	2.94	2.30	2.58	2.83	2.61
NCH04	.146	.167	.021	.091	.115	.094
NCH511	.247	.266	.115	.188	.261	.191
NCH1218	.180	.192	.122	.099	.211	.131
MEANIN	22786.64	24693.34	13944.31	19524.60	18234.02	18097.31
AGE	46.118	43.54	58.96	51.41	52.07	52.18
No years ill %						
zero	72.82	100	-	-	-	-
one	9.47	-	-	59.09	38.12	38.40
two	4.46	-	-	12.12	16.57	21.39
three	2.62	-	-	4.55	10.50	12.63
four	2.50	-	-	4.55	11.05	11.60
five	1.99	-	-	9.09	9.39	7.99
six	2.36	-	-	10.61	14.36	7.99
seven	3.79	-	100	-	-	-

For men, average income is higher for the healthy than for the ill and for those that move from illness than for those that move to illness or who make multiple

[20] To describe the data for each outcome sequence would require(potentially) $2^7 = 128$ sub-samples.

Table 3: Variable means by sub-sample- WOMEN

	FULL $N = 3391$ $NT = 23737$	Ill 0 yrs $N = 2206$ $NT = 15442$	Ill 7 yrs $N = 141$ $NT = 987$	single move from illness $N = 104$ $NT = 728$	single move to illness $N = 254$ $NT = 1778$	multiple moves $N = 686$ $NT = 4802$
HLLTYES	.154	0	1	.338	.366	.367
WIDOWED	.121	.087	.236	.092	.208	.178
NVRMAR	.119	.120	.113	.155	.111	.114
DIVSEP	.083	.075	.103	.117	.093	.093
OTHETH	.026	.017	.057	.048	.039	.039
DEGHDEG	.093	.105	.014	.106	.075	.074
HNDALEV	.186	.200	.064	.298	.126	.172
OCSE	.312	.355	.149	.212	.217	.258
HHSIZE	2.76	2.89	2.20	2.73	2.52	2.55
NCH04	.157	.184	.061	.146	.092	.116
NCH511	.284	.320	.117	.243	.235	.228
NCH1218	.188	.203	.079	.173	.156	.178
MEANIN	20489.6	22495.50	13299.57	19557.36	16337.35	17195.73
AGE	47.11	44.40	60.27	44.10	53.67	51.14
No years ill %						
zero	65.05	100	-	-	-	-
one	12.36	-	-	51.92	42.91	37.32
two	5.78	-	-	18.27	16.14	19.83
three	4.10	-	-	5.77	12.20	14.87
four	3.24	-	-	3.85	9.84	11.81
five	2.65	-	-	5.77	8.27	9.18
six	2.65	-	-	14.42	10.63	7.00
seven	4.16	-	100	-	-	-

moves. A qualitatively similar, but reversed relationship exists between healthiness and age. Those that are always observed to be ill have a much lower level of academic attainment than those who are always observed to be healthy, and to a lesser degree, have lower qualifications than those who made a single move from illness or made multiple moves. Those who made a single move to illness also have lower educational qualifications than the last two groups. This may indicate that education interacts with past health, such that higher education increases the chances of recovering from illness. Empirically, this may translate into interactions between the lagged dependent variable and educational status in the dynamic models, or to differential evolution of exogenous variables or the error structure of the model. Those who are always ill live in smaller households, and have fewer young children. This does not appear to be solely an artefact of a negative correlation between age and fertility as those who make a single move to illness also have large numbers of young children in the household. Those who are ill in every period are more likely to be widowed or separated than those who are never ill, who are in turn more likely to have never been married or cohabited than any other category. This may be an artefact of a correlation between age and marital status. Non-whites are less likely to be ill in all seven periods and more likely, if initially ill, to make a 'permanent' move to healthiness. The distribution of years ill indicates significant persistence in observed illness. To compare this to the most extreme alternative, consider a

binomial distribution with constant probability of illness in every period, $p = 0.125$, (approximately the sample mean of h_i), independent across individuals. In this case, around 40% of men would be healthy in every period, while the probability of an individual being ill in every period would be almost zero(Prob=4.8 e-7). In contrast the sample proportions translate to around 73% and 4 % respectively.

The proportion of illness observations is around 25% higher for women than for men, in line with other data on the relative magnitudes of physician consultations and self-assessed general health. This indicates that our dichotomous measure is at least qualitatively in line with information from other sources. The patterns across income, age and levels of education are similar to those for men. Comparing the distribution of observed illness with that expected from independent and identical binomial distributions also indicates significant persistence. Letting $p = 0.15$, around 32% of women would be expected to be healthy in every period, while a negligible proportion(1.7 e-6) would be ill in every period. In contrast, the observed proportions translate to 65% and 4 % respectively. The decomposition of this observed persistence into components due to state dependence, serial correlation and unobserved heterogeneity is one of the main objectives of this paper.

Before continuing to a discussion of the empirical results, a number of considerations should be noted. Firstly, we do not include some standard measures of socio-economic and occupational status such as social class and occupational group. This is due to the likelihood of overparameterizing a model which is already difficult to identify due to the number of categorical variables we employ and the known difficulties in numerically identifying the parameters of the more general forms of the panel probit model. This is further justified by the high correlations of these variables with household income. However, we must be careful to note that the income effects are not conditional on these variables; they also include the effects of variables which are correlated with income and which may directly affect health. Given these considerations, we do not attempt to interpret the parameter estimates as pure causal effects. Rather, they are seen as composites of the effects of a change in income and an indirect effect due to variation in correlated variables such as socio-economic or occupational status(e.g a change from part-time to full-time work).

Furthermore, while the majority of the independent variables may be considered exogenous, the income effects may be further contaminated by the effects of correlation between the time-invariant individual effect and income. A number of authors(e.g. Mundlak(1978), Chamberlain(1984) have suggested parameterizing the

individual effect to obtain a correlated random effects model. The model we estimate may be derived from a number of structural models and correlated random effects specifications which are observationally equivalent. In particular, suppose the true structural model (where observed variables other than income are suppressed) is:

$$h_{it}^{*} = \beta ln(x_{it}/\bar{x}_i) + \alpha_i + \epsilon_{it}, \tag{13}$$

and the individual effect is parameterized as:

$$\alpha_i = \varphi ln(\bar{x}_i) + w_i. \tag{14}$$

By substitution, it can be seen that

$$h_{it}^{*} = \beta ln(x_{it}/\bar{x}_i) + \varphi ln(\bar{x}_i) + w_i + \epsilon_{it} \tag{15}$$

which is observationally equivalent to the specification we estimate.

Similarly, if a specification observationally equivalent to (15) was considered as the true structural model, then considering the individual effect to be parameterized as (14), the resulting observational model has the same form as (15). Other parameterizations of the individual effect in terms of $\sum_{s=1}^{T} \varphi_s ln x_{is} + w_i$ and a combination of this parameterization with (13) or (15) lead to models which are not observationally equivalent to our model. We estimated these models, and while Wald tests for the φ_s terms were generally not statistically significant, estimation was plagued by collinearity problems. Furthermore, previous work using the BHPS by Contoyannis and Rice(2000) has shown the correlation between an unobserved individual effect and self-assessed health to be time invariant in a model of earnings. This suggests that the individual effect in a model for health may be parameterized by a time invariant function of income such as (14) which leads to observational equivalence with our empirical model. In general, these considerations suggest that we should be wary of interpreting the estimates as structural effects.

5 Estimation Results

5.1 Static Models

To test whether the data could be pooled across men and women we estimated a pooled independent probit model with interaction terms for the explanatory variables. Pooling of the data is strongly rejected by a LR test: ($\chi^2(23) = 148.60, p = 0.0000$). It should be noted that this is a consistent test of parameter constancy as the pooled probit estimator is consistent for the coefficients given the correct specification of the latent conditional mean function and distributional form. Throughout the rest of the paper we present models estimated for men and women separately.

Secondly, we estimated independent probit models incorporating a robust estimator of the covariance matrix. Again, these estimators are consistent given correct specification of the latent conditional mean function and normality of the error terms. The results for men and women are presented in the first columns of Tables 13 and 14 in the appendix.

Thirdly, we implemented estimators of the Gaussian random effects model (equation (2) with $\gamma = \rho = 0$) using Gaussian quadrature with 24 evaluation points. This is useful as a benchmark to validate both the program and the accuracy of the simulation-based approach for likelihood evaluation and the construction of estimators. Thus Table 4 compares selected results using quadrature and MSL with $R = 150$. These are very close and indicate that the simulation-based approach delivers high accuracy for both likelihood evaluation and estimation in our context. Full results for the simulation estimators of the random effects model are in the second columns of Tables 13 and 14.

Fourthly, we estimated the static version of equation (2) allowing for serial correlation and random effects(RE+AR(1)) using MSL. The full results are presented in the third columns of Tables 13 and 14. Lastly we estimated the static version of equation (1) allowing for an unrestricted correlation structure. These results are in the fourth columns of Tables 13 and 14. Rather than describe in detail the results for all models here, we focus on comparisons of the main parameters of interest across the models.

In Table 5 alternative model selection criteria are displayed for the models estimated by MSL for $R = 150$. For both men and women, the unrestricted model is preferred on the grounds of the log-likelihood and the Akaike Information Criterion(AIC) defined as: $-2lnL + k$ where k is the number of estimated parameters in the model. While the unrestricted model is preferred when the penalty for in-

Table 4: Comparison of estimated permanent and transitory income coefficients and σ_α^2 using Gaussian quadrature and simulation for RE Probit models:($R = 150$)

	MEN		*WOMEN*	
	MSL	MLE	MSL	MLE
lnL	-4290.55	-4291.19	-6762.7	-6759.8
σ_α^2	.7883	.7836	.7425	.7471
Permanent Income	-0.510	-0.490	-0.307	-0.297
Transitory Income	-0.052	-0.049	-0.039	-0.038

Table 5: Alternative model selection criteria for models estimated using maximum simulated likelihood: ($R = 150$)

	lnL	*AIC*	*BIC*	*CAIC*
MEN:				
Unrestricted	-4188.07	8421.12	8731.92	8820.00
RE+AR1	-4214.83	8455.66	8635.23	8626.82
RE	-4290.55	8606.10	8778.76	8851.31
WOMEN:				
Unrestricted	-6662.12	13369.24	13690.04	13822.01
RE+AR1	-6683.35	13392.70	13578.05	13654.21
RE	-6762.67	13550.34	13728.56	13802.21

creasing k is additive in k, it is not preferred when the penalty for an increase for the number of parameters increases with the number of observations, as for the Bayesian Information Criterion(BIC): $-2lnL+(lnN)k$ or the Consistent AIC(CAIC) : $-2lnL+(1+lnN)k$. For both men and women, the RE+AR(1) model is preferred by the latter criteria. For nested models, the criteria can be seen as relating to the outcome from LR tests with progressively more stringent criteria required to reject the restricted model. For example, for women the LR statistic comparing the unrestricted with the RE+AR(1) model suggests a p-value of 0.0015. However, this is insufficient for the BIC and CAIC to prefer the unrestricted model; log-likelihood differences larger than implied by this p-value are required to prefer the general model, given the sample size and the number of extra parameters, relative to the RE+AR(1) model.[21] We therefore focus on the RE+AR(1) model as the results from this model are more concise, easier to interpret, and it is also preferred by the BIC and CAIC.

Table 6 shows that the magnitudes of the coefficients on permanent and transit-

[21] Cameron and Trivedi(1998) pp182-3 describe these alternative criteria.

Table 6: Permanent and transitory income effects under alternative models of the covariance structure:($R = 150$)-(standard errors in parentheses)

	INP(Robust)	*RE*	$RE + AR(1)$	*General*
MEN:				
Permanent	-0.573(0.0605)	-0.509(0.0488)	-0.509(.0486)	-0.511(.0488)
Transitory	-0.115(0.0296)	-0.052(0.0232)	-0.057(.0246)	-0.055(0.0241)
WOMEN:				
Permanent	-0.341(.0445)	-0.307(0.0375)	-0.313(0.0384)	-0.316(0.0379)
Transitory	-0.056(.0261)	-0.039(0.0217)	-0.035(0.0223)	-0.0341(0.0222)

ory income are largely invariant to the form of the correlation matrix. The relative magnitudes of the permanent to the transitory effects are consistently around 9 for both men and women. While we should be careful not to compare absolute parameter values across men and women as meaningful due to differential scaling, we can compare relative magnitudes directly. We can therefore infer that the estimated magnitudes of both the permanent and transitory income effects *relative to the latent error variance* seem to be about 5/3 for men relative to women. This condition should also be noted when comparing across other samples. With a similar distribution of exogenous characteristics across genders, this relationship will also hold for the ratio of the marginal effects at the respective sample means.

As noted in the descriptive analysis, it appears that the dynamics of illness may be influenced by educational status. We split the samples of men and women into further sub-samples based on the highest attained qualifications and estimated the preferred model(RE+AR(1)) for these sub-groups separately. These sub-group estimates are only available for the static models as the dynamic models were inestimable within education groups due to identification probems. Table 7 presents results for the estimates of the autocorrelation parameter and the proportion of variance attributable to the individual effect for the static models.

For the full sample of men, the inclusion of an autocorrelation term reduces the proportion of the total error variance due to the random effect by around 10%, suggesting that the estimate of unobserved heterogeneity is exaggerated when autocorrelation in the residual error component is ignored. Given that ρ is estimated to be positive, this result is expected. Across the subsamples of men there does not appear to be a discernible gradient in the estimates of ρ, while the variance of the individual effect is lower for those with intermediate levels of education. For women, the inclusion of the autocorrelation parameter has a similar effect in the full sample.

Table 7: Estimates of autocorrelation and proportion of variance parameters for RE and RE+AR(1) models by gender and educational group: $(R = 150)$

	σ^2_α		ρ
	RE	RE+AR(1)	RE+AR(1)
MEN:			
FULL SAMPLE	.7882	.6908	.5409
DEGHDEG	-	.6861	.4221
HNDALEV	-	.6102	.5147
OCSE	-	.7298	.4716
NOQUAL	-	.7028	.5781
WOMEN:			
FULL SAMPLE	.7425	.6811	.4009
DEGHDEG	-	.3880	.4215
HNDALEV	-	.5613	.4279
OCSE	-	.6913	.3702
NOQUAL	-	.7389	.3611

However, there is much more variation in the proportion of the variance explained by the individual effect, with the proportion much higher for those with no qualifications relative to those with a degree or higher degree: $(t = 3.39)$. This suggests more homogeneity amongst highly educated women in their unobserved propensities to suffer illness.[22] The autocorrelation parameters are, in general, lower for women suggesting that unobserved health shocks are not as persistent. However, the autocorrelation parameters are very similar for men and women with a degree or higher degree.

Table 8 presents estimates of permanent and transitory income effects for all samples based on RE+AR(1) models estimated using MSL with $R = 150$. It should be noted that the functional form of the model allows us to derive neat interpretations of the effects of income from the estimated coefficents. Letting τ be the transitory income effect and π the permanent income effect we can decompose the total conditional effect of income on latent health for individual i in period t as: $\tau ln(x_{it}) + (\pi - \tau) ln(\bar{x}_i)$. This decomposition allows us to examine whether various forms of income effect exist. There are five possible alternatives:

[22] This may also indicate the existence for a number of latent subgroups for the unqualified, splitting the population into those that are more likely(conditional on observed characteristics) to be be ill for all seven periods and those healthy for all seven periods. Investigation of this issue may be approached using a semi-parametric(finite mixture) approach, but is beyond the scope of this paper.

1.) $\tau \neq \pi \neq 0$, there exist both transitory and permanent income effects with the relative magnitudes given by the coefficients.

2.) $\tau = 0, \pi \neq 0$, existence of permanent effect only.

3.) $\tau \neq 0, \pi = 0$, existence of transitory effect only.

4.) $\tau = \pi \neq 0$, current income effect only.

5.) $\tau = 0, \pi = 0$, no income effects.

The parameter estimates(and standard errors) allow us to easily test which of these hypotheses are best supported by the data. For example, for the full sample of men, both coefficient estimates are individually statistically significant at the 5% level, while the hypothesis of equality is easily rejected, the test statistic is equal to 79.51, with a χ^2 critical value of 3.814. This implies the existence of both transitory and permanent income effects, with the permanent effect around 9 times the magnitude of the transitory effect. An almost identical result occurs for women, although the transitory effect is not statistically significant, indicating that only a permanent income effect may exist for women.

Table 8: Permanent and transitory income effects for RE+AR1 models by gender and educational category:($R = 150$)

	Permanent	*Transitory*	$\widehat{\beta_{PER}}/\widehat{\beta_{TRA}}$
MEN			
FULL	-0.509(0.049)	-0.057(0.025)	8.93
DEDHDEG	-0.277(0.192)	0.056(0.104)	-4.95
HNDALEV	-0.590(0.107)	-0.100(0.059)	5.90
OCSE	-0.505(0.096)	-0.040(0.058)	12.63
NOQUAL	-0.545(0.075)	-0.089(0.037)	6.12
WOMEN			
FULL	-0.313(0.038)	-0.035(0.022)	8.94
DEGHDEG	-0.406(0.149)	-0.004(0.090)	101.50
HNDALEV	-0.272(0.094)	0.031(0.069)	-8.77
OCSE	-0.392(0.071)	-0.040(0.043)	9.80
NOQUAL	-0.290(0.057)	-0.058(0.032)	5.00

For men only, the health of those with a degree or higher degree appears responsive to permanent differences in income, although neither coefficient is individually statistically significant, nor can the hypothesis of equality be rejected. The estimated permanent response is substantially smaller than for the other categories.

Other educational categories have similar permanent responses, although there is some variation in the transitory effects. However, the parameter estimate is statistically significant only for those with no qualifications.

For women with a degree or higher degree, as for men, health does not exhibit a transitory income response. However, unlike highly educated men, they exhibit a greater response to permanent income variation than other groups. For all groups, the effect of transitory income is not statistically significant, while for all groups we are able to reject the hypotheses of the non-existence of permanent effects and of the equality of coefficients at the 1% level.

Table 9: Estimates of permanent and transitory income effects, σ_α^2 and ρ by gender and educational group for RE+AR(1) model: $(R = 75/100/150)$

	Permanent			*Transitory*		
	R=75	R=100	R=150	R=75	R=100	R=150
MEN						
FULL	-0.515	-0.515	-0.510	-0.057	-0.059	-0.057
DEGHDEG	-0.268	-0.289	-0.277	0.053	0.051	0.056
HNDALEV	-0.582	-0.580	-0.590	-0.098	-0.097	-0.100
OCSE	-0.505	-0.514	-0.505	-0.036	-0.040	-0.040
NOQUAL	-0.5266	-0.5288	-0.5447	-0.0876	-0.0886	-0.0893
WOMEN						
FULL	-0.3080	-0.3159	-0.3127	-0.0319	-0.0343	-0.0348
DEGHDEG	-0.4215	-0.4124	-0.4060	-0.0080	-0.0077	-0.0040
HNDALEV	-0.2738	-0.2702	-0.2716	0.0318	0.0311	0.0311
OCSE	-0.3949	-0.3963	-0.3919	-0.0396	-0.0400	-0.0401
NOQUAL	-0.2921	-0.2956	-0.2905	-0.0589	-0.0595	-0.0579
		σ_α^2			ρ	
	R=75	R=100	R=150	R=75	R=100	R=150
MEN						
FULL	0.698	0.695	0.691	0.521	0.528	0.541
DEGHDEG	0.696	0.703	0.686	0.387	0.396	0.422
HNDALEV	.6057	0.5975	0.6102	.5043	0.5199	0.5147
OCSE	0.7217	0.7107	0.7298	0.4712	0.5004	0.4716
NOQUAL	0.7055	0.6920	0.7028	0.5721	0.5933	0.5781
WOMEN						
FULL	0.6803	0.6820	0.6811	0.3933	0.3916	0.4009
DEGHDEG	0.3810	0.3721	0.3880	0.4362	0.4360	0.4215
HNDALEV	0.5578	0.5538	0.5613	0.4246	0.4385	0.4279
OCSE	0.6780	0.6866	0.6913	0.3864	0.3786	0.3702
NOQUAL	0.7373	0.7335	0.7389	0.3688	0.3669	0.3611

Finally, we consider the effects of varying the number of replications in the MSL estimation of the RE+AR(1) model. This approach is standard as an informal approach to assessing whether a sufficient number of replications have been employed (e.g. Mealli and Pudney(1996)). Table 9 presents estimates of permanent and transitory income effects and the correlation matrix parameters for different levels of R. It can be seen from this table that R does not appear to be influential, with the possible exception of the autocorrelation parameter. $R = 150$ appears sufficient to ensure convergence of the estimates and indicates that this is a sufficient number of replications in our context. While this may appear excessive, we use a large

numbers of replications due to the Monte Carlo evidence of Hyslop(1999), which suggests that substantial simulation bias may exist for the estimates of the effects of state dependence and the autocorrelation parameter. This was observed by Hyslop (1999) for a large number, ($R > 100$) replications without antithetic acceleration, when there is both state dependence and serial correlation. Furthermore, the full samples of men and women that we use are substantially larger than the 1,000 used by Hyslop(1999) in his Monte Carlo analysis. As R must increase with N to ensure similar accuracy and for consistency of the estimators, this further justifies the use of larger values of R in our circumstances. We also use $R = 150$ for estimation of the dynamic models.

A more formal approach to the sufficiency of the number of replications is to apply the test for MSL bias suggested by Hajivassiliou(2000) and described in appendix A. The results are presented in Table 10.

As expected, column 1 shows that using only one replication is insufficient to obtain an asymptotically unbiased estimator. However, there is a positive relationship between the test statistics and sample size. This suggests that the statistics reflect relative magnitudes of bias: for smaller values of N, R can be reduced while maintaining the variance due to simulation. Scanning the columns, it is also apparent that, as the number of replications is increased, the average value of the test statistics gradually reduces. Between $R = 40$ and $R = 75$, the mean values of the statistics appear to be stabilising, with only the random variability due to each statistic being calculated from a fresh sample of the data generating process remaining(see Appendix A for details of the calculation of the test statistics). While the test statistic therefore appears informative, as Table 9 also suggests stability by $R = 75$, a full understanding of the properties of the test requires further investigation.

Table 10: Test statistics and p-values(.) for test for simulation bias for given numbers of replications: RE+AR(1) models: ($S = 10$)

	$R = 1$	$R = 10$	$R = 40$	$R = 75$	$R = 150$
MEN:					
FULL	701.11(0.0000)	78.34(0.0000)	30.35(.2533)	17.66(.8878	37.52(.0671)
DEGHDEG	72.09(0.0000)	27.04(.2543)	16.11(.8506)	34.93(.0528)	32.80(.0846)
HNDALEV	171.03(0.0000)	49.23(.0012)	24.78(.3618)	34.02(.0649)	17.86(.7651)
OCSE	273.66(0.0000)	48.12(.0016)	22.65(.4811)	29.97(.1503)	32.82(.0842)
NOQUAL	257.11(0.0000)	32.47(.0907)	58.09(0.0001)	38.33(.0235)	22.51(.4898
WOMEN:					
FULL	684.54(0.0000)	51.35(.0022)	20.10(.7868)	29.72(.2793)	21.28(.7275)
DEGHDEG	48.40(.0015)	26.20(.2913)	17.86(.7653)	22.41(.4955)	26.75(.2669)
HNDALEV	106.69(0.0000)	37.59(.0282)	21.64(.5420)	23.33(.4416)	22.76(.4751)
OCSE	265.65(0.0000)	35.06(.0513)	25.43(.3285)	17.91(.7626)	11.86(.9726)
NOQUAL	187.91(0.0000)	30.63(.1322)	22.71(.4775)	26.84(.2627)	33.01(.0809)

5.2 Dynamic results

We estimated the dynamic models allowing for state dependence and accounting for the initial conditions problem following the approach of Heckman(1981) using MSL with antithetic acceleration and $R = 150$. We implement this approach under both RE and RE+AR(1) error structures. We also use the approach of Wooldridge(2000) for the random effects model (as described in section 3.2). Tables 11 and 12 present selected parameter estimates for the full samples of men and women.

Table 11: Selected parameter estimates and t-statistics for models with state dependence-MEN(Full Sample)

	$MSL-RE$	$MSL-RE+AR(1)$	$CML-RE$
σ_α^2	.69(29.12)	.55(14.55)	.58(22.26)
ρ	-	-.35(-9.15)	-
y_{t-1}	.41(8.01)	.93(9.35)	.49(11.13)
y_{i0}	-	-	1.43(16.19)
Permanent	-.52(-10.64)	-.49(-10.57)	-.49(-8.40)
Transitory	-.05(-1.54)	-.05(-1.57)	-.04(-1.05)
deghdeg	-.21(-2.41)	-.20(-2.36)	-.14(-1.58)
hndalev	-.19(-2.89)	-.17(-2.76)	-.10(-1.40)
ocse	-.16(-2.38)	-.14(-2.29)	-.09(-1.38)

Table 12: Selected parameter estimates and t-statistics for models with state dependence-WOMEN(Full Sample)

	$MSL-RE$	$MSL-RE+AR(1)$	$CML-RE$
σ_α^2	.65(34.39)	.54(18.38)	.52(24.90)
ρ	-	-.29(-8.16)	-
y_{t-1}	.33(8.69)	.74(9.72)	.39(11.42)
y_{i0}	-	-	1.37(21.64)
Permanent	-.32(-8.49)	-.30(-8.40)	-.31(-6.98)
Transitory	-.05(-1.78)	-.04(-1.70)	-.03(-1.02)
deghdeg	-.19(-2.46)	-.18(-2.47)	-.06(-.79)
hndalev	-.13(-2.16)	-.12(-2.17)	-.08(-1.31)
ocse	-.21(-4.19)	-.20(-4.06)	-.11(-2.15)

The first column of Table 11 shows the results of estimating the model with RE and state dependence. Inclusion of the lagged dependent variable reduces the proportion of variance attributable to heterogeneity by about 10% relative to that shown in Table 7. The impact of the lagged dependent variable is substantial, and approximately equal to twice the effect of having no qualifications relative to having

a degree. The relative and absolute effects of permanent and transitory income are almost identical to the case with no state dependence. A gradient in the effects of education is discernible, but small.

The second column of Table 11 shows the effects of also allowing for serial correlation. The inclusion of the ρ parameter further reduces the proportion of variance due to the unobserved effect by around another 20 % and is itself now large and *negative*. This contrasts with the case without state dependence where ρ was estimated as 0.54. It is not obvious why this is so, but it may be that the model has a more general form of state dependence which is not captured by this first-order Markov model, but which is fitted by the serial correlation parameter. Considering only one lag of health outcomes in the model may then generate this result.[23] With the exception of the coefficient on the lagged dependent variable, which more than doubles in magnitude, the remainder of the coefficients change little. This is of interest as it suggests that the effects of income and education are remarkably robust to the dynamic specification of the model, at least in this context. Furthermore, we have now conditioned on previous health status, which may be considered a major contributor to current income in a model with feedback. We may now be more inclined to treat the permanent and transitory income parameters as structural effects.

The third column of Table 11 presents results using the CML estimator for the RE model suggested by Wooldridge(2000). In this case the proportion of variance accounted for by unobserved heterogeneity is smaller than that suggested using the approach of Heckman(1981), while the effect of the lagged dependent variable is slightly larger. As expected, a positive correlation is found between the initial outcome and the unobserved effect. The difference in the estimate of the proportion of the variance explained by the individual effect may be due to correlations between the effect and the explanatory variables which is not accounted for by the MSL estimator. However, this does not seem to affect the estimates of the impacts of permanent and transitory income, adding weight to the previous suggestion that these effects may be interpreted as structural. However, the effects of education are substantially diminished relative to the MSL estimates. The comparative effects for women are similar to those for men and, therefore, we do not discuss these results

[23] Coincidentally, very similar results for the effects of inclusion of state dependence on the parameters of the covariance matrix are found by Hyslop(1999) when considering the intertemporal labour force participation of married women. However, a concern here is the fragility of the identification of the two parameters. To assess this possibility we started the optimization routine from alternative starting values which included a positive value of ρ. This led to a local optimum but with a lower log-likelihood value than for the estimates reported above.

further.

6 Conclusion

The study of income dynamics, such as the analysis of transitions into and out of poverty, has a long history. The analysis of health dynamics is less well established. This paper considers the determinants of a binary indicator for the existence of functional limitations using seven waves(1991-1997) of the British Household Panel Survey(BHPS). Previous analyses of health using the BHPS (e.g Benzeval *et al.*(2000)) have used simple empirical models and measures of income which have not fully exploited the panel dimension of the data. Our models allow for persistence in the observed outcomes due to state dependence, unobservable individual effects (heterogeneity), and autocorrelation in the transitory error component. Allowing for persistence is important:comparison of the observed outcomes with a simple binomial model shows that persistence is substantial in our dataset.

We estimate static and dynamic panel probit models by Maximum Simulated Likelihood(MSL) using the GHK simulator with antithetic acceleration. We implement a test for the existence of asymptotic bias due to simulation suggested by Hajivassiliou(2000). This test is used to select the number of required replications for use in MSL estimation. We consider two approaches to dealing with the problem of initial conditions in models with unobserved effects and lagged dependent variables. We use the approach of Wooldridge(2000) for random effects models which can be implemented with standard software, and Heckman(1981), which is relatively difficult to implement, but can accommodate more flexible error structures.

Based on information criteria, our results suggest that a sufficient parameterization for the error process in these longitudinal models comprises a random effects structure with the addition of a first-order autocorrelated error component. This model was compared with an unrestricted and random effects correlation structures. For both men and women we find that, in the models which do not allow for state dependence, the addition of a serially correlated error component reduces the proportion of variance explained by the individual effect. The magnitude of this reduction -10%- is very similar to the effect of allowing for state dependence but without serial correlation in the error term. This suggests that the proportion of variance due to time-invariant unobservable factors, and hence the amount of outcome persistence due to these factors, is overestimated in models which do not allow for dynamics. However, allowing for both lagged outcomes and autocorrelated errors reduces the

proportion of variance due to time-invariant unobservables by a further 20% relative to including one form of dynamics. These effects do not differ by gender.

We find that the proportion of total variance due to time-invariant unobservable characteristics is generally slightly higher for men than for women, with the variation across education groups substantially higher for women than for men. This finding suggests that educational subgroups are more homogenous with respect to unobserved health for women than men. For models without state dependence, the effect of unobserved health shocks is less persistent for women than for men, suggesting that the trajectory of male health is determined by previous shocks to latent health status to a greater degree than for women.

The relationship between long-run changes in income and health is found to be around 10 times the effect of short-run changes in income, for the full samples, suggesting that permanent deprivation has a greater effect on an individuals health trajectory than short-run variations. These results are in line with previous findings that the relationship between permanent deprivation and health is substantially greater than for temporary deprivation(e.g Benzeval *et al.*(2000)). While the addition of a lagged outcome variable generally reduces the absolute values of the effects of exogenous regressors, it does not affect the short-run effect of income. In models with state dependence the effect of previous limitations is approximately twice that of having no qualifications relative to having a university education. State dependence explains more of the persistence of functional limitations for men than for women.

References

Benzeval, M., Taylor, J., and Judge, K. (2000). Evidence on the relationship between low income and poor health: Is the Government doing enough? *Fiscal Studies*, 21(3):375–399.

Bertschek, I. and Lechner, M. (1998). Convenient estimators for the panel probit model. *Journal of Econometrics*, 87(2):329–371.

Borsch-Supan, A. and Hajivassiliou, V. (1993). Smooth unbiased multivariate probability simulators for maximum likelihood estimation of limited dependent variable models. *Journal of Econometrics*, 58:347–368.

Butler, J. and Moffitt, R. (1982). A computationally efficient quadrature procedure for the one-factor multinomial probit model. *Econometrica*, 50(3):761–764.

Cameron, A. and Trivedi, P. (1998). *Regression analysis of count data.* Cambridge University Press.

Chamberlain, G. (1984). *Panel data*, chapter 22, pages 1247–1318. North-Holland, Amsterdam. in Handbook of Econometrics, volume 1, edited by Z. Griliches and M.D. Intriligator.

Contoyannis, P. (2000). An introduction to simulation-based estimation. *University of York: http://www.york.ac.uk/res/herc/docs/simulation.pdf* .

Contoyannis, P. and Rice, N. (2000). The impact of health on wages: evidence from the British Household Panel Survey. *Empirical Economics, forthcoming.*

Geweke, J., Keane, M., and Runkle, D. (1997). Statistical inference in the multinomial multiperiod probit model. *Journal of Econometrics*, 80:125–165.

Grossman, M. (1972). On the concept of health capital and the demand for health. *Journal of Political Economy*, 80(1):223–255.

Grossman, M. (2000). *The human capital model*, pages 347–408. Elsevier. chapter 7 in 'Handbook of Health Economics' edited by AJ Culyer and JP Newhouse.

Guilkey, D. and Murphy, J. (1993). Estimation and testing in the random effects probit model. *Journal of Econometrics*, 59(2):301–317.

Hajivassiliou, V. (1994). A simulation estimation analysis of the external debt crises of developing countries. *Journal of Applied Econometrics*, 9:109–132.

Hajivassiliou, V. (2000). *Some practical issues in maximum simulated likelihood*, chapter 3, pages 71–99. CUP. in Simulation-based inference in econometrics:Methods and Applications, edited by R Mariano, T Schuermann, and MJ Weeks.

Hajivassiliou, V. and McFadden, D. (1998). The method of simulated scores for the estimation of LDV models. *Econometrica*, 66(4):863–896.

Hajivassiliou, V., McFadden, D., and Ruud, P. (1996). Simulation of multivariate normal rectangle probabilities and their derivatives: Theoretical and computational results. *Journal of Econometrics*, 72:85–134.

Hajivassiliou, V. and Ruud, P. (1994). *Classical estimation methods using simulation*, pages 2383–2441. Amsterdam:North Holland. in 'Handbook of Econometrics Vol 4' edited by R. Engle and D. McFadden.

Heckman, J. (1981). *The incidental parameters problem and the problem of initial conditions in estimating a discrete time-discrete data stochastic process*, chapter 4, pages 179–195. MIT press: Cambridge,MA. in Stuctural analsysis of discrete data with econometric applications, edited by C.F. Manski and D. McFadden.

Hyslop, D. (1999). State dependence, serial correlation and heterogeneity in intertemporal labor force participation of married women. *Econometrica*, 67(6):1255–1294.

Jenkins, S. (1998). Modelling household income dynamics. Presidential address to the European Society for Population Economics Twelfth Annual Congess, Amsterdam, June 4-6 1998.

Keane, M. (1994). A computationally practical simulation estimator for panel data. *Econometrica*, 62(1):95–116.

Kloek, T. and van Dijk, H. (1978). Bayesian estimates of equation system parameters: an application of integration by Monte Carlo. *Econometrica*, 46(1):1–19.

McFadden, D. (1989). A method of simulated moments for estimation of discrete response models without numerical integration. *Econometrica*, 57(5):995–1026.

Mealli, F. and Pudney, S. (1996). Occupational pensions and job mobility in britain: estimation of a random-effects competing risks model. *Journal of Applied Econometrics*, 11(2):293–320.

Mundlak, Y. (1978). On the pooling of time series and cross-section data. *Econometrica*, 46(1):69–85.

Munkin, M. and Trivedi, P. (1999). Simulated maximum likelihood estimation of multivariate mixed-poisson regression models, with application. *Econometrics Journal*, 2:29–48.

Neuhaus, J. and Newell, N. (1993). A geometric approach to assess bias due to omitted covariates in generalized linear models. *Biometrics*, 80:807–816.

Pendergast, J., Gange, S., Newton, M., Lindstrom, M., Palta, M., and Fisher, M. (1996). A survey of methods for analysing clustered binary response data. *International Statistical Review*, 64:89–118.

Robinson, P. (1982). On the asymptotic properties of estimators of models containing limited dependent variables. *Econometrica*, 50(1):27–41.

Salas, C.-P. (2000). Econometric analysis of health status dynamics among the British elderly: Multivariate Markov chain models with unobserved heterogeneity. mimeo-University of Birmingham.

Stern, S. (1997). Simulation-based estimation. *Journal of Economic Literature*, 35(4):2006–2039.

Taylor, M., Brice, J., Buck, N., and Prentice, E. (1998). *British Household Panel Survey User Manual Volume A: Introduction, Technical Report and Appendices.* University of Essex, Colchester.

Van-Doorslaer, E. (1987). *Health, Knowledge and the the demand for medical care.* Assen, Maastricht.

Vijverberg, W. (1997). Monte Carlo evaluation of multivariate normal probabilities. *Journal of Econometrics*, 76:281–307.

Wagstaff, A. (1993). The demand for health: an empirical reformulation of the Grossman model. *Health Economics*, 2:189–198.

Wooldridge, J. (2000). The initial conditions problem in dynamic, nonlinear panel data models with unobserved heterogeneity. Mimeo, University of Michigan.

Appendix A: A test for simulation bias

This appendix describes the diagnostic test for the number of simulation replications and closely follows Hajivassiliou(2000). Hajivassiliou (2000) suggests a diagnostic test for simulation bias based on the fact that the expectation of the deterministic score function at the true parameter values is equal to zero. The sample analogue at the true values will also converge to zero as $N \to \infty$ in the absence of simulation ensuring that the MLE is consistent. However, in the presence of simulation, asymptotic bias due to an insufficent value of R, for a large value of N, would be indicated by the expectation taking a 'large' non-zero value.

So, under the null hypothesis that the MSL estimator is consistent:

$$E_{Y,X}[g(\theta; y, x, \omega)|\omega, \theta = \theta_0] = 0 \tag{16}$$

where $g(\theta; .)$ is the score of the (simulated) likelihood, ω is the set of draws used to construct g, and θ_0 is the true value of θ,and

$$Var_{Y,X}[g(\theta; y, x, \omega)|\omega, \theta = \theta_0] = E_{Y,X}(g[(\theta_0; y, x, \omega)]g[(\theta_0; y, x, \omega)]'|\omega) \tag{17}$$

A basis for a test of consistency is to check (16). Hajivassiliou(2000) recommends constructing a test statistic based on taking S additional simulations from the individual specific data generating process defined by an initial MSL estimate and the regressors and constructing a 'sample' analogue of the expectation of the score function based on these NS 'observations'. So, let $y(\hat{\theta})$ denote a simulation of the data generating process for y at $\hat{\theta}$ conditional on x, where this additional simulation is independent of ω, and let E_S denote the empirical expectation of functions of $y(\hat{\theta})$ over S replications of $y(\hat{\theta})$. Then

$$m \equiv E_S(g[\hat{\theta}; y(\hat{\theta}), x, \omega]) \tag{18}$$

and

$$V \equiv Var_S(g[\hat{\theta}; y(\hat{\theta}), x, \omega]) \tag{19}$$

are unbiased simulators of $E_Y(g[\hat{\theta}; y, x, \omega]|x, \omega)$ and $Var_Y(g[\hat{\theta}; y, x, \omega]|x, \omega)$ respectively. Then under the hypothesis that the MSL estimator is consistent

$$[E_N(V)]^{-1/2}\sqrt{SN}E_N(m) \underset{d}{\to} N(0, I) \quad as N \to \infty \tag{20}$$

Thus a specification test can be based on the Wald statistic:

$$W = SN.[E_N(m)]'[E_N(V)]^{-1}[E_N(m)] \tag{21}$$

Under the null of consistency, W has an asymptotic distribution that is central χ^2 with K degrees of freedom, where K is the dimension of θ, the number of estimated parameters. As the test statistic takes account of the variance of the underlying MSL estimator, interpretation of the result is conditional on this variance such that a statistically insignificant value is taken as evidence that the variation in the estimator is large relative to the simulation bias, while a statistically significant value may be interpreted as evidence of bias relative to the variance of the MSL estimator. Thus, a statistically insignificant value and a small variance of the MSL estimator can be interpreted as evidence that there is negligible bias in the MSL estimator itself. If the value of the test statistic is judged as non-negligible, a value of R to reduce the bias to an acceptable level may be found by performing the above calculations with different values of R using different simulations. It should be noted that we may reject the null due to insufficient N whatever the value of R chosen. Conversely, it may appear that failure to reject the null is evidence that R and N are jointly sufficient, although this is potentially misleading given that the distribution of the test statistic is valid only for large N. The test is valid conditional on a large value of N, such that the limiting distribution of the test statistic is well approximated. The test and estimators were programmed in GAUSS, with the estimators based on adaptations of the program used by Keane(1994) and Geweke et al. (1997).

Appendix B: Full Results

Table 13: Static Model Parameter estimates- MEN-(standard errors in parentheses)

	INP(Robust)	RE(MSL)	RE+AR(1)	General
WIDOWED	-.165(.149)	-.293(.084)	-.270(.090)	-.279(.091)
NVRMAR	.113(.087)	.085(.058)	.076(.060)	.067(.060)
DIVSEP	-.056(.112)	-.063(.069)	-.088(.074)	-.083(.073)
OTHETH	.129(.138)	.175(.133)	.170(.133)	.164(.133)
DEGHDEG	-.183(.097)	-.226(.090)	-.217(.089)	-.222(.090)
HNDALEV	-.217(.071)	-.215(.068)	-.216(.067)	-.215(.068)
OCSE	.156(.072)	-.182(.067)	-.183(.067)	-.183(.067)
HHSIZE	.086(.032)	.022(.021)	.022(.022)	.020(.022)
NCH04	-.141(.067)	-.040(.044)	-.037(.047)	-.032(.046)
NCH511	-.120(.051)	-.059(.035)	-.054(.037)	-.053(.038)
NCH1218	-.050(.051)	-.005(.034)	-.013(.037)	-.003(.037)
DEVINC	-.115(.030)	-.052(.023)	-.057(.025)	-.054(.024)
MEANIN	-.573(.061)	-.510(.049)	-.510(.049)	-.511(.049)
AGE	-.353(.098)	-.392(.078)	-.408(.086)	-.400(.086)
AGE2	1.272(.314)	1.314(.241)	1.371(.268)	1.351(.269)
AGE3	-1.751(.419)	-1.740(.315)	-1.820(.350)	-1.803(.351)
AGE4	.825(.198)	.803(.146)	.842(.163)	.837(.164)
yr9192	.068(.035)	.068(.035)	.077(.030)	.079(.034)
yr9293	.120(.036)	.119(.035)	.127(.034)	.137(.036)
yr9394	.149(.036)	.145(.035)	.151(.036)	.171(.036)
yr9495	.149(.036)	.156(.035)	.158(.038)	.175(.038)
yr9596	.164(.038)	.229(.035)	.233(.038)	.245(.039)
yr9697	.291(.042)	.287(.035)	.287(.038)	.294(.040)
CONSTANT	6.935(1.281)	7.328(1.019)	7.493(1.102)	7.384(1.112)
Ln L	-6263.52	-4290.55	-4214.83	-4188.07
σ_α^2	-	.788(.012)	.691(.025)	-
ρ	-	-	.541(.040)	-

Table 14: Static Model Parameter estimates- WOMEN-(Standard errors in parentheses)

	INP(Robust)	RE(MSL)	RE+AR(1)	General
WIDOWED	-.132(.070)	-.110(.052)	-.116(.055)	-.111(.055)
NVRMAR	-.096(.076)	-.109(.052)	.106(.054)	-.098(.054)
DIVSEP	.054(.072)	-.005(.049)	-.005(.050)	-.001(.050)
OTHETH	.546(.111)	.574(.113)	.585(.112)	.586(.111)
DEGHDEG	-.286(.085)	-.226(.081)	-.222(.080)	-.224(.080)
HNDALEV	-.129(.064)	-.135(.060)	-.128(.060)	-.129(.060)
OCSE	-.220(.056)	-.255(.052)	-.249(.052)	-.244(.052)
HHSIZE	.075(.027)	.033(.017)	.032(.018)	.033(.018)
NCH04	-.150(.052)	-.068(.033)	-.064(.035)	-.063(.035)
NCH511	-.097(.042)	-.056(.028)	-.054(.030)	-.054(.030)
NCH1218	-.089(.045)	-.030(.028)	-.029(.030)	-.031(.031)
DEVINC	-.056(.026)	-.039(.022)	-.035(.022)	-.034(.022)
MEANIN	-.341(.044)	-.307(.038)	-.313(.038)	-.316(.038)
AGE	-.264(.086)	-.307(.063)	-.350(.070)	-.348(.072)
AGE2	.927(.274)	.985(.196)	1.129(.217)	1.126(.224)
AGE3	-1.287(.366)	-1.302(.254)	-1.497(.282)	-1.496(.291)
AGE4	.627(.173)	.620(.118)	.711(.131)	.711(.135)
yr9192	.054(.028)	.055(.030)	.061(.027)	.060(.028)
yr9293	.059(.029)	.062(.030)	.069(.029)	.072(.029)
yr9394	.102(.030)	.112(.030)	.117(.031)	.123(.030)
yr9495	.119(.030)	.128(.030)	.133(.031)	.141(.031)
yr9596	.170(.032)	.180(.030)	.183(.031)	.187(.032)
yr9697	.241(.033)	.255(.030)	.255(.031)	.259(.033)
CONSTANT	4.373(1.042)	5.024(.826)	5.482(.889)	5.473(.912)
Ln L	-9340.24	-6762.67	-6683.35	-6662.12
σ_α^2	-	.743(.011)	.681(.016)	-
ρ	-	-	.401(.030)	-

Table 15: Dynamic Model Parameter estimates- MEN- (Standard errors in parentheses)

	CML-RE	MSL-RE	SD-RE+AR(1)
WIDOWED	-.424(.181)	-.334(.099)	-.340(.100)
NVRMAR	.121(.130)	.055(.066)	.054(.065)
DIVSEP	-.123(.133)	-.099(.081)	-.093(.083)
OTHETH	-.091(.141)	.105(.134)	.081(.124)
DEGHDEG	-.139(.091)	-.215(.089)	-.198(.084)
HNDALEV	-.093(.066)	-.193(.067)	-.172(.062)
OCSE	-.089(.064)	-.158(.066)	-.142(.062)
HHSIZE	-.036(.037)	.013(.025)	.020(.026)
NCH04	.050(.070)	-.002(.053)	-.002(.055)
NCH511	-.043(.061)	-.049(.042)	-.050(.043)
NCH1218	.088(.060)	.047(.043)	.052(.045)
DEVINC	-.038(.037)	-.047(.031)	-.054(.034)
MEANIN	-.485(.057)	-.524(.049)	-.494(.047)
AGE	-.719(.379)	-.523(.089)	-.495(.088)
AGE2	1.727(.524)	1.738(.279)	1.656(.274)
AGE3	-2.305(.699)	-2.305(.366)	-2.208(.358)
AGE4	1.091(.329)	1.068(.170)	1.028(.167)
yr9192	-	-	-
yr9293	.239(.344)	.047(.039)	.042(.049)
yr9394	.451(.684)	.065(.041)	.048(.046)
yr9495	.644(1.025)	.070(.041)	.043(.048)
yr9596	.936(1.366)	.154(.041)	.138(.047)
yr9697	1.181(1.708)	.201(.040)	.178(.046)
y_{t-1}	.476(.042)	.410(.051)	.931(.099)
y_0	1.431(.088)	-	-
CONSTANT	5.147(1.627)	8.836(1.136)	8.089(1.111)
Ln L	-3418.01	-4183.34	-4165.40
σ_α^2	.579(.026)	.693(.024)	.552(.038)
ρ	-	-	-.349(.038)

Table 16: Dynamic Model Parameter estimates- WOMEN-(Standard errors in parentheses)

	CML-RE	MSL-RE	SD-RE+AR(1)
WIDOWED	-.093(.103)	-.120(.057)	-.114(.056)
NVRMAR	-.176(.107)	-.124(.057)	-.121(.056)
DIVSEP	-.059(.091)	-.005(.054)	.009(.054)
OTHETH	.428(.106)	.570(.109)	.532(.102)
DEGHDEG	-.063(.080)	-.191(.078)	-.182(.073)
HNDALEV	-.078(.060)	-.128(.059)	-.121(.056)
OCSE	-.108(.050)	-.216(.051)	-.197(.049)
HHSIZE	-.008(.030)	.028(.020)	.028(.021)
NCH04	-.022(.055)	-.054(.040)	-.060(.041)
NCH511	-.060(.050)	-.048(.034)	-.040(.034)
NCH1218	-.019(.051)	-.026(.035)	-.031(.036)
DEVINC	-.033(.032)	-.048(.027)	-.049(.029)
MEANIN	-.314(.045)	-.322(.038)	-.305(.036)
AGE	.047(.435)	-.378(.077)	-.362(.076)
AGE2	-.250(.435)	1.223(.239)	1.176(.236)
AGE3	.384(.576)	-1.618(.308)	-1.559(.304)
AGE4	-.149(.268)	.764(.142)	.736(.140)
yr9192	-	-	-
yr9293	-.016(.417)	.003(.032)	-.001(.038)
yr9394	.032(.832)	.054(.033)	.055(.036)
yr9495	.032(1.248)	.063(.033)	.056(.037)
yr9596	.084(1.663)	.117(.033)	.113(.036)
yr9697	.158(2.078)	.192(.033)	.186(.036)
y_{t-1}	.392(.034)	.326(.038)	.743(.076)
y_0	1.372(.063)	-	-
CONSTANT	2.907(1.470)	5.811(.969)	5.325(.949)
Ln L	-5461.89	-6670.42	-6655.04
σ_α^2	.579(.026)	.649(.019)	.536(.029)
ρ	-	-	-.291(.036)