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A Bayesian Nonparametric Approach to Longitudinal Mediation Analysis

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Does Long-Term Care Provision Reduce Health Care Utilization? A Bayesian Nonparametric Approach to Longitudinal Mediation Analysis

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Abstract

The paper addresses causal relationship between long term care and health care utilization of the elderly. The expansion of long-term care (LTC) may improve health system efficiency by reducing hospitalisations, and pave the way for the implementation of health and social care coordination plans. We draw upon the longitudinal evidence from Survey of Health, Ageing and Retirement in Europe (SHARE) to derive causal estimates of the effects of receiving different types of LTC on health care utilization. We analyze the causal problem with health indicators as mediators. To solve for multiple reverse causality we utilize cross-lagged panel models, a form of longitudinal mediation analysis. While being a highly under-researched methodology, the latter is based on strong parametric assumptions. To solve for this we use a novel Bayesian nonparametric (BNP) estimator for longitudinal mediation, based on dependent Dirichlet processes for longitudinal data introduced by Quintana et al. (2016). The results of both parametric and nonparametric modelling confirm positive effects of LTC provision on reducing health care utilization with both direct and indirect significant effects in most criteria. The article provides a novel methodological possibility, not used so far in the analysis of the relationship between long term care and health care, and an application of a novel BNP estimator, first one for longitudinal mediation analysis and one of first nonparametric estimators for structural equation models.

Keywords: long term care provision, health care utilization, longitudinal mediation analysis, cross-lagged panel models, dependent Dirichlet process

JEL: C33, C14, C11, I18, I15, I11
1. Introduction and literature review

The ageing of countries’ populations, and in particular the growing number of the very old that is occurring in most industrialised countries, is increasing the need for long-term care (LTC) services. LTC is defined as "a range of services required by persons with a reduced degree of functional capacity, physical or cognitive, and who are consequently dependent for an extended period of time on help with basic activities of daily living" (Colombo et al., 2011). Unlike other personal services, the development of both private and public LTC insurance has been limited, which has contributed to the escalation of public and household LTC expenditures. At the same time, the transformation of family structures, the distancing of children from their parents (Costa-Font, 2010) and higher female labour market participation rates are responsible for a decline in the supply of informal caregiving (Pezzin and Steinberg Schone, 1999).

The combination of population ageing and social change suggests that in the coming years, there will be a greater demand for formal LTC (e.g. personal care, community care and institutional care provided in people’s homes or nursing homes and assisted living facilities) funded by government programmes, private LTC insurance or individuals’ out-of-pocket payments. However, such a shift in the type of LTC has important economic implications insofar as the cost of LTC in Europe, and Organisation for Economic Co-operation and Development (OECD) countries generally, has traditionally been borne by families themselves or by the public purse to a great extent when fiscal conditions have been favourable. Spending on LTC in OECD countries averaged 1.5% of gross domestic product (GDP) in 2008, but if current trends continue, it is predicted to more than double by 2050 (Colombo and Mercier, 2012). This poses an important policy dilemma and raises questions about the financing of LTC, especially when a large share of such expenditures currently is publicly funded. To protect against the risk of needing costly LTC, various financial mechanisms are available to varying degrees in different countries. One set of mechanisms is of the ex-ante type – that is, measures are taken before the onset of dependence. These comprise insurance (social or private), prevention (reducing either the probability of needing LTC or its future cost) and precautionary savings. Another set of financing mechanisms is of the ex post type – that is, measures are taken after the onset of dependence. These include the subsidisation of formal and informal LTC, family support and the use of housing equity for financing LTC (e.g., ‘reverse mortgages’). Although population ageing exerts pressure on governments, it is difficult to conceive of an expansion of existing public programmes covering LTC in times of austerity.

Health care systems face the challenge of responding to the rising costs of health care treatments (Breyer et al., 2010). Part of such rise in health care demand is deemed to result from an inefficient use of health services (especially hospital care) by individuals who would need long-term care (LTC) instead. This is typically the case when LTC services are not affordable, and/or not adequately-coordinated with health care services. Indeed, a shortage of either suitable and/or affordable LTC due to limited insurance or public subsidy, or inadequate integration, is suggested to result in inefficient and costlier hospital care utilisation (Mur-Veeman and Govers, 2011; Hofmarcher et al., 2007; Bodenheimer, 2008). However, limited research has so far focused on the identification of such an effect.

The effect of the introduction of social care programmes on hospitalisations has shown mixed results so far. Hospital readmissions, rate of hospital-delayed discharges and emergency readmission rates are found to decline after the introduction of a home visits programme (Hermiz et al., 2002; Weaver and Weaver, 2014; Sand et al. 2006), but other studies find no
Another set of studies draws on quasi-experimental data. Rapp et al. (2015) measure the impact of financial assistance for non-medical provision on the probability of requiring emergency care among patients with Alzheimer’s disease. They conclude that the beneficiaries of LTC subsidies have a significantly lower rate of emergency care than non-beneficiaries. Holmás et al. (2008) found that a system of penalties for a non-smooth transfer process from hospital to LTC services involved hospital stays that were approximately 2.3 days shorter. However, the elimination of the penalties lead to hospital stays that are three days longer. Costa-Font and colleagues (2016) seek to fill some of the gaps in the literature, and as in previous studies (Geil et al., 1997; van Houtven and Norton, 2004; Card et al., 2004; Nielsen, 2016) draw upon individual data to study hospital admissions.

Finally, some literature related to our study examines the effect of improvements in integration and care coordination on health care use. Health and social care coordination is found to improve individual’s quality of life (Hofmarcher et al., 2007), but without a cost increase (Singh and Ham 2005). However, the effects on hospital admission are not always consistent across different programmes.

In the article, we want to add to the literature by solving the reverse causality between long term care provision and health care utilization in a manner which would be more feasible and not needing exogenous change and natural or quasi-natural experiments. To this end, we use a novel empirical approach to this problem, longitudinal mediation analysis, which controls for reverse causality with a longitudinal approach. We use panel data of Survey of Health, Ageing and Retirement in Europe (SHARE) for Slovenia in Waves 4-7 to estimate the effects of different types of long term care provision on different outcome variables in health care utilization. We conceptualize the problem in light of mediation analysis with health status as mediator (using three different health indicators). As longitudinal mediation suffers from statistical problems based on its parametric assumptions, we experiment with a new, Bayesian nonparametric estimator using dependent Dirichlet processes for longitudinal data. Our application confirms the expected negative effect of long term care provision on health care utilization in almost all used criteria.

The article is structured in the following manner. In section 2 we present the method and derivation of the new estimator. In section 3 we describe the dataset and used variables. In section 4 we present results of all modelling. We conclude with the discussion of the findings, limitations and relevance of the article in section 5.

1 This paper uses data from SHARE Waves 4, 5, 6 and 7 (10.6103/SHARE.w4.710, 10.6103/SHARE.w5.710, 10.6103/SHARE.w6.710, 10.6103/SHARE.w7.710), see Börsch-Supan et al. (2013) for methodological details. The SHARE data collection has been funded by the European Commission through FP5 (QLK6-CT-2001-00360), FP6 (SHARE-I3: RII-CT-2006-062193, COMPARE: CIT-CT-2005-028857, SHARELIFE: CIT-CT-2006-028812), FP7 (SHARE-PREP: GA N°211909, SHARE-LEAP: GA N°227822, SHARE M4: GA N°261982, DASISH: GA N°283646) and Horizon 2020 (SHARE-DEV3: GA N°676536, SHARE-COHESION: GA N°870628, SERISS: GA N°654221, SSHOC: GA N°823782) and by DG Employment, Social Affairs & Inclusion. Additional funding from the German Ministry of Education and Research, the Max Planck Society for the Advancement of Science, the U.S. National Institute on Aging (U01_AG09740-13S2, P01_AG005842, P01_AG08291, P30_AG12815, R21_AG025169, Y1_AG-4553-01, IAG_BSR06-11, OGHA_04-064, HHSN271201300071C) and from various national funding sources is gratefully acknowledged (see www.share-project.org).
2. Longitudinal mediation analysis and derivation of the estimator

Mediation analysis is a statistical approach used to understand how a predictor (generically, \(X\)) produces an indirect effect on an outcome (\(Y\)) through an intervening variable (mediator, \(M\)). For example, diet programme might be hypothesised to reduce food intake, which, in turn, is hypothesised to reduce the participant’s body mass index. This analysis, therefore, aims to uncover causal pathways along which changes are transmitted from causes to effects. There are two essential ingredients of modern mediation analysis. First, the indirect effect is not merely a modelling artefact formed by suggestive combinations of parameters but an intrinsic property of reality that has tangible policy implications. Second, the policy decisions concern the enabling and disabling of processes (hiring vs. education) rather than lowering or raising values of specific variables. These two considerations lead to the analysis of natural direct and indirect effects (Pearl, 2014: 459).

We define the direct and indirect effect following Chen and Hung (2016). Figure 1 shows the typical mediation model; path coefficient \(c\) is termed as the direct effect of the independent variable \((X)\) on the dependent variable \((Y)\), also known as the effect of independent variable \((X)\) on dependent variable \((Y)\) controlling for the mediator variable \((M)\), or the residual effect. Path coefficient \(a\) is the effect of independent variable \((X)\) on mediator variable \((M)\), also known as the first stage effect. Path coefficient \(b\) is the effect of the mediator variable \((M)\) on the dependent variable \((Y)\), also known as the second stage effect. The multiplication of the first stage effect and second stage effect \(ab\) is known as the indirect effect. If the direct effect of independent variable \((X)\) on the dependent variable \((Y)\) after the addition of the mediator variable \((M)\) is insignificant (namely, path coefficient \(c\) is significant), it is known as the full mediation.

**Figure 1:** Basic mediator model.

![Basic mediator model](image)

Source: Chen and Hung, 2016.

For the estimation of mediating effects, the simple and most commonly used algorithm of Baron and Kenny (1986) has been advanced using longitudinal mediation analysis. Baron-Kenny algorithm proposes a four step approach in which several regression analyses are conducted and significance of the coefficients is examined at each step (\(Y\) is the response, in our case hospitalizations; \(X\) is the predictor, in our case LTC; and \(M\) is the mediator variable, in our case health status). The detailed scheme of the approach is provided in Figure 2.

**Figure 2:** Basic diagram of Baron and Kenny's approach
There are a number of fundamental problems with the application of traditional mediation models to cross-sectional data (Gollob and Reichardt, 1987). Firstly, the causal relationships implied by the paths in the mediation model take time to unfold. The use of cross-sectional data implies that the effects are instantaneous. Secondly, it is well known that conclusions based on a causal model that omits a key predictor can be seriously in error, yet a model based on cross-sectional data leaves out several key predictors—namely the variables measured at previous times. When previous levels of the variables are not controlled for, the paths in the mediation model may be over- or underestimated relative to their true values. Third, effects unfold over time, and we would not expect the magnitude of a causal effect to remain the same for all possible intervals.

Selig and Preacher (2009) consider three mediation models for longitudinal data: a cross-lagged panel model (CLPM), a latent growth curve model, and a latent difference score model. In our analysis, we focus on the first one. The CLPM is a multivariate extension of the univariate simplex model, one of the most commonly used structural models for the analysis of longitudinal data (Jöreskog, 1970, 1979). The CLPM allows time for causes to have their effects, supports stronger inference about the direction of causation in comparison to models using cross-sectional data, and reduces the probable parameter bias that arises when using cross-sectional data. Extensive overviews of the use of this model for mediation analyses were given by Cole and Maxwell (2003), MacKinnon (2008) and Bernal Turnes and Ernst (2016). Figure 3 depicts such a model.

**Figure 3:** A cross-lagged panel mediation model

In Figure 3, three constructs – X, M and Y – are each measured at four times. The CLPM can be used with more or fewer waves of measurement, but at least three are needed to achieve a fully longitudinal mediation model. The constructs X, M and Y are often latent variables with multiple indicators, although the model can be used with observed variables. Using latent variables has the advantage of addressing the problem of measurement error, thus disattenuating relationships among the constructs. The CLPM for X, M and Y can be expressed by the following three equations,

\[
X_{[t]} = \beta_{X,[t-1]}X_{[t-1]} + \zeta_{X,[t]} \quad (1)
\]

\[
M_{[t]} = \beta_{M,[t-1]}M_{[t-1]} + \beta_{X,[t-1]}X_{[t-1]} + \zeta_{M,[t]} \quad (2)
\]

\[
Y_{[t]} = \beta_{Y,[t-1]}Y_{[t-1]} + \beta_{M,[t-1]}M_{[t-1]} + \beta_{X,[t-2]}X_{[t-2]} + \zeta_{Y,[t]} \quad (3)
\]

where \(X_{[t]}\) is the value of X at time \(t\), \(\beta_{X,[t-1]}\) expresses the relationship between the construct X at time \(t\) and the same construct measured at the previous time \(t-1\), and \(\zeta_{X,[t]}\) is a random disturbance that is different for each time. Similar interpretations can be given to corresponding terms in the equations for \(M_{[t]}\) and \(Y_{[t]}\). The mediated, i.e. indirect effect of \(X\) on \(Y\) can therefore be expressed in terms of the product of \(\beta_{X,[t-1]}\) and \(\beta_{M,[t-1]}\).

What is most important for our analysis, the scheme of the model, depicted in Figure 3, allows to overcome the multiple reverse causality present in a cross-sectional form of mediation model by utilizing a longitudinal empirical strategy which is able to break the contemporaneous reverse causal relationships.

Furthermore, the models in (1)-(3) are estimated under strong parametric assumptions, which can impose statistical problems (see Bernal Turnes and Ernst, 2016, which refer to Judd & Kenny, 1981; Gollob & Reichardt, 1987; Sobel, 1990; Kraemer et al., 2002; Cole & Maxwell, 2003; Selig & Preacher, 2009). It is, therefore, recommended to use semi- or nonparametric approaches (for example, Bernal Turnes and Ernst, 2016 suggest bootstrapping).

For final verification purposes, it is therefore recommendable using a different modelling approach. We decided to use Bayesian nonparametric modelling, which is subject to many discussions and research in mathematical statistics in recent years. A Bayesian nonparametric model is a Bayesian model on an infinite-dimensional parameter space (Orbanz and Teh, 2011). The parameter space is typically chosen as the set of all possible solutions for a given learning problem. A Bayesian nonparametric model uses only a finite subset of the available parameter dimensions to explain a finite sample of observations, with the set of dimensions chosen depending on the sample, such that the effective complexity of the model (as measured by the number of dimensions used) adapts to the data. Classical adaptive problems, such as nonparametric estimation and model selection, can thus be formulated as Bayesian inference problems. Popular examples of Bayesian nonparametric models include Gaussian process regression, in which the correlation structure is refined with growing sample size, and Dirichlet process mixture models for clustering.

There are a large number of methods for mediation analysis in the literature proposed from a frequentist perspective (Pearl, 2001; MacKinnon et al., 2002; Robins, 2003; Preacher and Hayes, 2004; Petersen et al., 2006; VanderWeele, 2009; Imai et al. 2010, Albert and Nelson, 2011; Tchetgen Tchetgen et al. 2012; Valeri and VanderWeele, 2013), with fewer approaches using Bayesian inference. The latter include Yuan and MacKinnon (2009), Elliott et al. (2010),
Schwartz et al. (2011), Daniels et al. (2012) and Mattei et al. (2013). Elliott et al. (2010) develop an approach for estimating nonparametric bounds of principal strata causal effects of a dichotomous mediator and a dichotomous outcome by using prior distributions over a possible range of values. Schwartz et al. (2011) use a Bayesian nonparametric model, a Dirichlet process mixture model, to construct the distribution of principal strata of continuous mediators. Their model identifies the strata of continuous mediators and explores the latent structure of the data automatically. Mattei et al. (2013) develop a Bayesian principal stratification inference method for multiple outcomes not based on structural assumptions but based on flexible distributional assumptions. Kim et al. (2017; 2018; 2019) propose a Bayesian non-parametric (BNP) framework for estimating causal effects of mediation, the natural direct and indirect effects, in two parts: Part one is a flexible model (using BNP) for the observed data distribution, specifying a Dirichlet process mixture of multivariate normals as a prior on the joint distribution of the outcome, mediator and covariates; Part two is a set of uncheckable assumptions with sensitivity parameters.

In our analysis, we extend the existing Bayesian nonparametric model of causal mediation by Kim et al. (2019) with Dirichlet dependent processes appropriate for longitudinal data. In their basic model, Kim and colleagues model causal effects of mediation in combination of a Bayesian dynamic model with Dirichlet Processes Priors. Their basic Bayesian dynamic model specification lists as follows. For each exposure level at time \( t \), \( Z(t) \), they assume that conditional on direct preceding observations and the vector of state parameters \( \theta(t) \), \( Y(t) \) (or \( M(t) \) or \( W(t) \)) is independent of all future and past observations at time \( s \) and \( \theta(s) \) for all \( s \neq t \):

**Observation model:**

\[
Y(t) \sim p_0(Y(t)|Z(t), M(t), W(t), Y(t-1), \theta(t)) \quad (4)
\]

\[
M(t) \sim p_0(M(t)|Z(t), M(t-1), W(t), \theta(t)) \quad (5)
\]

\[
W(t) \sim p_0(W(t)|Z(t), M(t-1), W(t-1), \theta(t)) \quad (6)
\]

**Evolution model:**

\[
(\theta(t)|\theta(t-1) \sim p_e(\theta(t)|\theta(t-1)) \quad (7)
\]

where the vector of state parameters \( \theta(t) \) evolves via the evolution model in (7).

They place Dirichlet process priors on the distributions of coefficient parameters and variance parameters of the models (4)-(7). For instance, at each time \( t \) and exposure level \( z \):

\[
\begin{aligned}
(\alpha_{t,i}^z, \sigma_{M,t,i}^z) & \sim F_t^z \\
F_t^z & \sim DP(\lambda_t^z, \mathcal{F}_t^z) \\
\mathcal{F}_t^z & = \prod_{h=1}^{card(\alpha_{t,i}^z)} \mathcal{N} \left( \mathcal{A}_{t,h}^z, \frac{1}{\tau_{t,h}^z} \right) \times \text{Inv.Gamma}(a, b)
\end{aligned}
\]

(8) \hspace{1cm} (9) \hspace{1cm} (10)

where \( \alpha_{t,i}^z = \{\alpha_{t0,i}^z, \alpha_{t1,i}^z, \alpha_{t2,i}^z\} \) and \( DP \) denotes the Dirichlet process with two parameters, a mass parameter \( (\lambda_t^z) \) and a base measure \( (\mathcal{F}_t^z) \). Here, \( \mathcal{A}_{t,h}^z \) and \( \tau_{t,h}^z \) denote the mean and the precision parameters of the \( h \)-th base distribution (i.e. the base distribution for the \( h \)-th element of \( \alpha_{t,i}^z \)).

Models for dependent data, including random and/or fixed functions in time, various forms of random effects, latent stochastic processes, combination of functions and robust methods that accommodate without modeling covariance structure, have been applied to Bayesian
nonparametric modelling in several works (e.g. Laird and Ware, 1982; Zeger and Diggle, 1994; Shi, Weiss and Taylor, 1996; Zhang and Davdian, 2001; Li, Lin and Müller, 2010; He, Zhu and Fung, 2002). Quintana et al. (2016) presented a novel statistical model that generalizes standard mixed models for longitudinal data that include flexible mean functions as well as combined compound symmetry (CS) and autoregressive (AR) covariance structures. They allow for AR structure by considering a broader class of models that incorporates a Dirichlet Process Mixture (DPM) over the covariance parameters of the GP. Yang and Dunson (2010) propose a broad class of semiparametric Bayesian SEMs, to include identifiability restrictions on the latent variable distributions, rely on centered Dirichlet process (CDP) and CDP mixture (CDPM) models.

We define a Dirichlet process (DP) as follows. Given a measurable set \( S \), a base probability distribution \( H \) and a positive real number \( \alpha \), Dirichlet process \( DP(H, \alpha) \) is a stochastic process whose sample path (infinite sequence of random variates drawn from the process) is a probability distribution over \( S \), such that the following holds: for any measurable finite partition of \( S \), denoted \( \{B_i\}_{i=1}^n \), if \( X \sim DP(H, \alpha) \) then \( (X(B_1), \ldots, X(B_n)) \sim \text{Dir}(\alpha H(B_1), \ldots, \alpha H(B_n)) \).

DP random measure is defined as \( G \sim DP(\text{Dir}(\alpha H), M) \), where:

\[
G(\cdot) = \sum_{h=1}^{\infty} w_h \delta_{m_h}(\cdot) \\
\quad \text{for } w_h = v_h \prod_{g<h} (1 - v_g) \text{ with } v_h \sim \text{Beta}(1, M)
\]

Dirichlet dependent process is defined using the same construction for each \( G_x \),

\[
G_x(\cdot) = \sum_{h=1}^{\infty} w_h \delta_{m_{x,h}}(\cdot)
\]

\( m_h = \{m_{x,h}: x \in X\} \) are independent realizations from a stochastic process on \( X \).

The generative model can be written as:

\[
\theta_{t,i} | D_t \sim D_t \quad \text{for } i = 1, \ldots, n_t, \ t = 0, \ldots, T \tag{14}
\]

\[
X_{t,i} | \theta_{t,i} \sim F(\theta_{t,i}) \quad \text{for } i = 1, \ldots, n_t, \ t = 0, \ldots, T \tag{15}
\]

In light of this, our BNP estimator for Baron-Kenny-based cross-lagged panel model is defined as follows.

Assume that observations are made on individual \( i \) at times \( \{t_{i1}, \ldots, t_{in_i}\} \), namely \( y_i = \{y_{ij}: j = 1, \ldots, n_i\}^\prime \). At time \( t_{ij} \) we allow for a vector of possibly time dependent covariates \( x_{ij}^\prime = (1, x_{i1}(t_{ij}), \ldots, x_{ip}(t_{ij})) \), and assume that \( E(y_{ij}) = x_{ij}^\prime \beta \). Define the \( n_i \times (p + 1) \) design matrix \( X_i = (x_{i1}, x_{i2}, \ldots, x_{in_i})^\prime \), leading to an assumed mean vector \( E(y_i) = \mu_i = X_i \beta \). The allow for a corresponding \( n_i \times q \) design matrix \( Z_i \) with \( q \leq p \) and with the column space of \( Z_i \) restricted to be contained in the column space of \( X_i \).
Our model is based on a generalization of the linear mixed model (Diggle, 1988) that allows for AR structure:

\begin{align*}
y_i &= \mu_i + Z_i b_i + w_i + \varepsilon_i \quad (17) \\
b_i | \xi &\sim N_r(0, D(\xi)) \quad (18) \\
w_i | \phi &\sim N_{n_i}(0, H_i(\phi)) \quad (19)
\end{align*}

where \( H_i(\phi) \) is \( n_i \times n_i \) and has a structural form, and where \( \varepsilon_i \sim N(0, \sigma^2 I_{n_i}) \). The vectors \( \xi \) and \( \phi \) contain variance-covariance parameters for \( b_i \) and \( w_i \), respectively.

The vectors \( w_i \) are generated by mean zero Gaussian stochastic processes, \( \{ w_i(t) : t > 0 \} \). If \( \text{Cov}(w_i(t + s), w_i(t)) = \sigma^2(s) \), with \( \sigma(s) = \rho^2 \), the resulting stationary process is an Ornstein-Uhlenbeck process which gives an exponential covariance function and induces AR structure.

Consider \( w_i \sim \text{GP} \) for the \( i \)th subject, with covariance matrix \( H_i(\phi) = \sigma^2 \tilde{H}_i(\phi) \), where \( \phi = (\sigma^2, \rho) \) and \( \{ \tilde{H}_i(\rho) \}_{k=1}^N = \rho^{t_{i(k)} - t_{i(k-1)}} \) with \( (t_{i1}, \ldots, t_{in_i}) \) the times at which observations \( y_i = \{ y_{ij} : j = 1, \ldots, n_i \} \) for the \( i \)th subject are made.

We model \( \phi | G \sim G \) with \( G \sim \text{DP}(G_0, M) \) so that

\begin{equation}
f(\omega_i | G) = \int N(\omega_i | 0, \sigma^2 \tilde{H}_i(\rho))dG(\phi) = \sum_{k=1}^{\infty} \omega_k N_{n_i}(\omega_i | 0, \tilde{\sigma}^2_{\omega k} \tilde{H}_i(\tilde{\rho}_k)) \\
\quad (\tilde{\sigma}^2_{\omega k}, \tilde{\rho}_k \sim \text{iid} \, G_0) \quad (20)
\end{equation}

Basic specification of the model then follows as combination of Kim et al. (2019) and above approach:

\begin{align*}
(Y_{obs, i}^T, M_{obs, i}^T, X_{\xi}^T) &\sim N_q(\mu_{\xi, i}, \Sigma_{\xi, i}), i = 1, \ldots, n_z \quad (22) \\
(\mu_{\xi, i}, \Sigma_{\xi, i}) &\sim G_{\xi, i}, i = 1, \ldots, n_z \quad (23) \\
G_{\xi, i} &\sim \text{DDP}(\alpha_{\xi}, \theta_{\xi}) \quad (24)
\end{align*}

\begin{align*}
\theta_{\xi, i} | D_i &\sim \text{D}_t \quad \text{for } i = 1, \ldots, n_z, t = 0, \ldots, Z \\
X_{\xi, i} | \theta_{\xi, i} &\sim \tilde{F}(\theta_{\xi, i}) \quad \text{for } i = 1, \ldots, n_z, t = 0, \ldots, Z \\
\phi_1 &\sim G_0, \quad \phi_k | (\phi_1, \ldots, \phi_{k-1}) \sim \frac{MG_0 + \sum_{l=1}^{k-1} \delta_{\phi_l}}{M + k - 1} \quad (25)
\end{align*}

where the base distribution \( \Theta_{\xi} \) is taken to be the conjugate normal-inverse-Wishart distribution (NIW):

\begin{equation}
N(\mu_{\xi}, m_{\xi}, \kappa_0^{-1} \Sigma_{\xi}) \mathcal{W}^{-1}(\Sigma_{\xi}, v_{\xi}, \Psi_{\xi}) \quad (28)
\end{equation}

and \( G_{\xi, i} \) are MacEachern type dependent Dirichlet processes.

The inverse Wishart is parameterized such that

\begin{equation}
\mathbb{E} [\Sigma_{\xi}] = \frac{\Psi_{\xi}^{-1}}{v_{\xi} - q - 1} \quad (29)
\end{equation}
We specify a Gamma prior $G(1,1)$ on the mass parameter $\alpha_z$.

For posterior computation we use two different algorithms (Neal, 2000):

a) Metropolis Hastings:

- Let the state of the Markov chain consist of $\theta = (\theta_1, ..., \theta_n)$. Repeatedly sample as follows:
- For $i = 1, ..., n$, repeat the following update of $\theta_i$ $R$ times: Draw a candidate, $\theta_i^*$, from the following distribution:
  \[
  \frac{1}{n - 1 + \alpha} \delta(\theta_j) + \frac{\alpha}{n - 1 + \alpha} G_0
  \]  
  (31)
- Compute the acceptance probability
  \[
  a(\theta_i^*, \theta_i) = \min \left[ 1, \frac{F(y_i, \theta_i^*)}{F(y_i, \theta_i)} \right]
  \]  
  (32)
- Set the new value of $\theta_i$ to $\theta_i^*$ with this probability; otherwise let the new value of $\theta_i$ be the same as the old value.

b) Partial Gibbs:

- Let the state of the Markov chain consist of $c = (c_1, ..., c_n)$ and $\phi = (\phi_c: c \in \{c_1, ..., c_n\})$. Repeatedly sample as follows:
- For $i = 1, ..., n$, update $c_i$ as follows: If $c_i$ is not a singleton (i.e., $c_i = c_j$ for some $j \neq i$), let $c_i^*$ be a newly created component, with $\phi_{c_i^*}$ drawn from $G_0$. Set the new $c_i$ to this $c_i^*$ with probability
  \[
  a(c_i^*, c_i) = \min \left[ 1, \frac{\alpha}{n - 1} \frac{F(y_i, \phi_{c_i^*})}{F(y_i, \phi_{c_i})} \right]
  \]  
  (33)
- Otherwise, when $c_i$ is a singleton, draw $c_i^*$ from $c_{-i}$, choosing $c_i^* = c$ with probability $n_{-i,c}/(n - 1)$. Set the new $c_i$ to this $c_i^*$ with probability
  \[
  a(c_i^*, c_i) = \min \left[ 1, \frac{n - 1}{\alpha} \frac{F(y_i, \phi_{c_i^*})}{F(y_i, \phi_{c_i})} \right]
  \]  
  (34)
- If the new $c_i$ is not set to $c_i^*$, it is the same as the old $c_i$.
- For $i = 1, ..., n$: If $c_i$ is a singleton (i.e., $c_i \neq c_j$ for all $j \neq i$), do nothing. Otherwise, choose a new value for $c_i$ from $\{c_i, ..., c_n\}$ using the following probabilities: $P(c_i = c|c_{-i}, y_i, \phi, c_i \in \{c_i, ..., c_n\}) = b^{n-1-c}F(y_i, \phi_c)$, where $b$ is the appropriate normalizing constant.
- For all $c \in \{c_i, ..., c_n\}$: Draw a new value from $\phi_c|y_i$ such that $c_i = c$, or perform someother update to $\phi_c$ that leaves this distribution invariant.

Simulation-based evidence (presented in Srakar and Bartolj, 2019) shows that the estimator attains the information rate (Alaa and van der Schaar, 2018), being defined as the asymptotic equivalence class of the expected value of the Kullback-Leibler divergence between the estimated and true distributions as a function of the number of samples. It also shows the estimator is asymptotically consistent.

3. Dataset and variables
The Survey of Health, Ageing and Retirement in Europe (SHARE) is a multidisciplinary and cross-national panel database of micro data on health, socio-economic status and social and family networks of about 140,000 individuals aged 50 or older (around 380,000 interviews). SHARE covers 27 European countries and Israel.

SHARE was started in 2004 to study paths of ageing of people aged 50 years and older in several European countries (and Israel). SHARE is an ex-ante harmonised cross-country survey. The questionnaire has been designed by a core team consisting of international experts of health, employment, social networks, and so on. It is largely based on the US Health and Retirement Study and the English Longitudinal Study of Ageing with multidisciplinarity, cross nationality and longitudinality being the main principles of including questions.

SHARE Wave 4 was the first time this survey was conducted in Slovenia, therefore no previous information on the country was available for analysis. To map the initial situation of older Slovenians, we use Slovenian data from the SHARE Waves 4-7. The Slovenian SHARE survey uses a randomised sample stratified by age, sex, origin (native born or foreign born) and regional distribution as of 1 January 2010. The sample is representative of the 50+ aged population of the country and provides a sufficient number of cases for subgroups to be analysed. The sample size and the response rate (Bergmann et al., 2019) were relatively high compared with other countries participating in this wave. Until July 2011, SHARE was reviewed and approved by the Ethics Committee of the University of Mannheim. Since then, the Ethics Council of the Max Planck Society for the Advancement of Science (MPG) is responsible for ethical reviews and the approval of the study.

The data used in this analysis are based on the main survey respondents and their partners aged 50 and above who were interviewed in at least three waves among waves 4-7 of the SHARE study in Slovenia. With regard to the other variables, the analysis included missing values and ‘don’t know’ responses. The final sample for our analysis after excluding the non-responses was 1,354 people.

Our basic mediation model in contemporaneous terms is presented in Figure 1. Clearly, all three included types of variables are expected to have causal relationships to each other and vice versa.

To resolve multiple reverse causal relationships, we transform the analysis into longitudinal mediation problem (cross-lagged panel model) where, for example, health care utilization in time \( t \) depends on health status in time \( t - 1 \), which depends on long term care provision in time \( t - 2 \).

Figure 4: Contemporaneous causal structure of our model

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2 This paper uses data from SHARE Waves 4, 5, 6 and 7 (10.6103/SHARE.w4.700, 10.6103/SHARE.w5.700, 10.6103/SHARE.w6.700, 10.6103/SHARE.w7.700), see Börsch-Supan et al. (2013) for methodological details. The SHARE data collection has been funded by the European Commission through FPS (QLK6-CT-2001-00360), FP6 (SHARE-I3: RII-CT-2006-062193, COMPARE: CIT5-CT-2005-028857, SHARELIFE: CIT4-CT-2006-028812), FP7 (SHARE-PREP: GA N°211909, SHARE-LEAP: GA N°227822, SHARE M4: GA N°261982) and Horizon 2020 (SHARE-DEV3: GA N°676536, SERISS: GA N°654221) and by DG Employment, Social Affairs & Inclusion. Additional funding from the German Ministry of Education and Research, the Max Planck Society for the Advancement of Science, the U.S. National Institute on Aging (U01_AG09740-1352, P01_AG05842, P01_AG08291, P30_AG12815, R21_AG025169, Y1-AG-4553-01, IAG_BSR06-11, OGHAS_04-064, HHSN271201300071C) and from various national funding sources is gratefully acknowledged(see www.share-project.org).
As main variables we use three types: dependent variables, describing health care utilization; mediators, describing health status; and independent/source variables, describing long-term care provision. All of the main variables used in the modelling are depicted in Table 1.

**Table 1: Main variables, used in the analysis, by three types (dependent, mediating, independent/source)**

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Description</th>
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<tr>
<td>No. of hospitalisations</td>
<td>Number of hospitalisations in a hospital overnight during the last twelve months</td>
</tr>
<tr>
<td>Probability of hospitalisation</td>
<td>Response to the following question: »During the last twelve months, have you been in a hospital overnight? Please consider stays in medical, surgical, psychiatric or in any other specialised wards.«</td>
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<tr>
<td>Length of hospitalization</td>
<td>Length of hospitalization in days</td>
</tr>
<tr>
<td>No. of taken medications</td>
<td>Number of taken medications as a sum of answers to the following question: »Do you currently take drugs at least once a week for problems mentioned?«</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Mediator</th>
<th>Description</th>
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<tr>
<td>ChronDis</td>
<td>Count variable, counting number of chronic diseases</td>
</tr>
<tr>
<td>Depression</td>
<td>Count variables, having the value of the score on the Euro-Depression scale</td>
</tr>
<tr>
<td>Self-rated health</td>
<td>Ordinal variable ranked according to a five-point scale: (1) ‘excellent’, (2) ‘very good’, (3) ‘good’, (4) ‘fair’ and (5) ‘poor’</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Source – Type of care</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Informal care outside of</td>
<td>Binary variable, having the value of 1 if the respondent is receiving</td>
</tr>
</tbody>
</table>

3 The drugs include the following: 1. Drugs for high blood cholesterol; 2. Drugs for high blood pressure; 3. Drugs for coronary or cerebrovascular diseases; 4. Drugs for other heart diseases; 6. Drugs for diabetes; 7. Drugs for joint pain or for joint inflammation; 8. Drugs for other pain (e.g. headache, back pain, etc.); 9. Drugs for sleep problems; 10. Drugs for anxiety or depression; 11. Drugs for osteoporosis; 13. Drugs for stomach burns; 14. Drugs for chronic bronchitis; 15. Drugs for suppressing inflammation (only glucocorticoids or steroids); 97. Other drugs, not yet mentioned.
4. Results

In Tables 2, 3 and 4 we present full results of the analysis for three different mediating health indicators: number of chronic diseases, EURO-Depression Score and self-rated health\(^4\). Standard errors for basic longitudinal mediation models have been bootstrapped following Pan et al. (2018).

Table 2 shows the results when using as mediator number of chronic diseases. Clear and negative effects on health care utilization can be seen in almost all variables included and when observing both direct and indirect effects. The coefficients denote the coefficient $\beta_{X[t-2]}$ from equation (3) for direct effect and the mediated, i.e. indirect effect expressed in terms of the product of $\beta_{X[t-1]}$ and $\beta_{M[t-1]}$ from equations (2) and (3). The total effect is the sum of both effects. Only coefficients which are statistically significant to 5% level are shown, the insignificant ones are left blank.

The effects when analyzed through the mediating effect of number of chronic diseases in Table 2 show consistency in terms of the effect of informal care provision – several coefficients in the left part of the table (parametric longitudinal mediation analysis/LMA) are negative and statistically significant. Very similar is the situation for formal care provision.

Slightly different are results for the hospitalization variables and number of medications. For the latter, the effects are clearly negative when significant. In total, the summed effects of all care types are negative and significant for all four health care utilization variables, and stronger when summed as separate variables for informal and formal care.

Finally, the results of Bayesian nonparametrics do not show significant differences, with main difference being that several coefficients, also for the informal care part, become insignificant. Also, the total effects when analyzed for a pooled care variable are mainly lower in size than in the parametric model.

Table 2: Total results, mediator: number of chronic diseases, left: results of parametric LMA analysis, right: results of Bayesian nonparametric estimation

<table>
<thead>
<tr>
<th>Mediator: Nr. of chronic diseases</th>
<th>LMA</th>
<th>ProbHosp</th>
<th>NeHosp</th>
<th>LghHosp</th>
<th>NeMedic</th>
<th>Mediator: Nr. of chronic diseases</th>
<th>BNP</th>
<th>ProbHosp</th>
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<td>-0.1091</td>
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<td></td>
<td>Total</td>
<td>-0.0057</td>
<td>-0.0164</td>
<td>-0.1244</td>
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<tr>
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</table>

\(^4\) For some of the empirical results we acknowledge the help of the student Vasilije Simeunović.
Almost every day, almost every care within and outside household are calculated almost every month, 3, 1 (Less often), 0 (Not receiving at all); 2, \((\text{results of Bayesian nonparametric estimation})\) as consequence of long term care provision (as attested in the literature) as well as a reason to stimulate health care utilization.

The same holds for the Bayesian nonparametric part of the model. Also, the intensity of receiving informal care, which remain negative whenever they are significant. The same holds for the Bayesian nonparametric part of the model. Also, the intensity of receiving informal care, which remain negative whenever they are significant.

Table 3 provides the analysis for a different mediator, namely depression score. Results change in terms of significance for the variable of informal care, which remain negative whenever they are significant. The same holds for the Bayesian nonparametric part of the model. Also, the results of Bayesian nonparametrics confirm some particularities when mental health is observed as consequence of long term care provision (as attested in the literature) as well as a reason to stimulate health care utilization.

Table 3: Total results, mediator: EURO-Depression Score, left: results of parametric LMA analysis, right: results of Bayesian nonparametric estimation.
Almost every day (2), Almost every month (1), Less often (0), Not receiving at all (0).

Results of Bayesian nonparametric estimation which are less reflected in health care utilization.

Care intensity having short term psychological effects on self-rated health, measured on the five point scale five.

Also, the indirect effect of the intensity of care through mediating effect of number of mediators.

Table 4 provides the results for our final mediator, self-rated health, measured on the five point scale five-point scale where higher value denotes worse self-rated health. Interestingly, the effects here become more significant for number of medications in both parametric and nonparametric models. Also, the indirect effect of the intensity of care through mediating effect of self-rated health almost vanishes in both parametric and nonparametric models, which could be explained by care intensity having short term psychological effects on self-rated health which are less reflected in health care utilization.

Table 4: Total results, mediator: self-rated health, left: results of parametric LMA analysis, right: results of Bayesian nonparametric estimation

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Note: Only statistically significant results (to the level of 5%) are shown and included. Abbreviations denote: InfCareWin – probability of receiving informal care within household; InfCareOut – probability of receiving informal care outside household; InfCareIntens – intensity of receiving informal care outside household, on the scale 4 (Almost every day), 3 (Almost every week), 2 (Almost every month), 1 (Less often), 0 (Not receiving at all); InfCareTot – probability of receiving any type of informal care, within or outside household; FormCare – probability of receiving any type of formal care; FormHomeC – probability of receiving help with personal care in own home; FormHelp – probability of receiving help with domestic tasks in own home; FormMeals – probability of receiving help with meals-on-wheels; Form+InfWO – total effects when informal care within and outside household are calculated separately; Form+InfCT – total effects when informal care within and outside household are calculated as pooled variable InfCareTot.

Source: Own calculations.
Note: Only statistically significant results (to the level of 5%) are shown and included. Abbreviations denote: InfCareWin – probability of receiving informal care within household; InfCareOut – probability of receiving informal care outside household; InfCareIntens – intensity of receiving informal care outside household, on the scale 4 (Almost every day), 3 (Almost every week), 2 (Almost every month), 1 (Less often), 0 (Not receiving at all); InfCareTot – probability of receiving any type of informal care, within or outside household; FormCare – probability of receiving any type of formal care; FormHomeC – probability of receiving help with personal care in own home; FormHelp – probability of receiving help with domestic tasks in own home; FormMeals – probability of receiving help with meals-on-wheels; Form+InfWO – total effects when informal care within and outside household are calculated separately; Form+InfCT – total effects when informal care within and outside household are calculated as pooled variable InfCareTot.

Source: Own calculations.

Figure 5 shows partial dependence plots for our Bayesian nonparametric modelling (they are shown only for the outcome variable of number of hospitalizations). The visualization follows a common approach to estimate marginal effects from nonparametric models, developed in Friedman (2001) and labelled as partial dependence functions/plots. In descriptive terms, partial dependence is an approximation to the target function which maps independent on dependent variables and minimizes the expected value of some specified loss function $L(y, F(x))$ over the joint distribution of all $(y, x)$ values.

The picture depicted in Tables 2-4 becomes even more clear and pronounced. Direct effects, shown in fourth and eighth graphs from the left of each row are clearly negative, furthermore, clearly negative are also the products of functions in second and third, respectively sixth and seventh graph in each row, which represent the indirect effects. This observation is confirmed for each part of the figure, which shows only the results for informal care, left part for informal care outside household, and the right one for informal care within household.
**Figure 5:** Partial dependence plots for results of Bayesian nonparametric modelling, dependent variable: number of hospitalisations.

Note: From top to bottom – three mediators: top, number of chronic diseases; middle, EURO-Depression score; bottom, self-rated health. From left to right, effect of: for informal care outside household – first lag of mediator to the original level of mediator; first lag of informal care outside household to the original level of mediator; first lag of mediator to dependent variable; second lag of informal care outside household to dependent variable; for informal care within household – first lag of mediator to the original level of mediator; first lag of informal care within household to the original level of mediator; first lag of mediator to dependent variable; second lag of informal care within household to dependent variable. 
Source: Own calculations.

5. **Discussion and conclusion**

The article presents several novelties. In the first place, we provide a new approach to analyze relationship between long term care provision and health care utilization, being able to solve the complex causal scheme, clearly mediated through the effects on health. The approach relies on longitudinal mediation analysis, deriving from the general framework of structural equation modelling, but in this case not being linked to latent variables. The approach resolves the causal scheme through longitudinal modelling, being able to include all cross-relationships in the
model structure.

Due to method being prone to distributional/parametric problems, we presented a new approach and estimator, based on Bayesian nonparametric approach using dependent Dirichlet processes for longitudinal data. Our estimator allows more flexibility than previous estimation in the line of Kim et al. (2019), although it needs to be tested in further empirical and theoretical work.

Finally, our main claim that the relationship between long term care provision and health care utilization is negative has been fully confirmed. We provided a rich analysis, using several source (long term care provision), mediating (health indicators) and outcome (health utilization) variables. In almost all cases we were able to confirm significant and negative, both direct and indirect effects of long term care provision to health care utilization. Indeed, as our analysis is based only on Slovenian SHARE data, it allows rich possibilities of extensions to datasets of other countries and also non-SHARE based analyses. It conveniently resolves what appears to be the main pressing issue in the literature: the reverse causality between two types of care provision, without the need of any external and exogenous variables, natural or quasi-natural experiments and the like. In this light, it appears to have large application possibilities and should deserve more attention in future.

Extensions of the article appear numerous. On the one hand, other types of estimators, surely to be developed for longitudinal mediation analysis in future, could be applicable here. In a previous article (Srakar and Bartolj, 2019) we propose a nonparametric estimator based on dynamic panel modelling and sieve-consistent estimation (following Su and Lu, 2013). As structural equation modelling in general lacks nonparametric considerations (pointed to in a recent article of Kelava et al., 2017), this approach should deserve more attention in statistical and econometric theory in future (based also on the current and longer standing theoretical interest in Bayesian nonparametric modelling).

The extensions to applications and policy analyses seem clear. As our estimates are causal, they could be multiplied by costs of each health care utilization feature (e.g. cost of hospitalization) to derive exact estimates, supporting future reforms in any country under study. Indeed, feasibility of our approach in empirical terms (as noted it does not require an exogenous change to resolve reverse causality) should provide ground for more applications. Also, the modelling could be extended in terms of variables, a clear possibility is using different individual diseases instead of a general variable for number of chronic diseases. In this case, one would get the effect mediated by each individual disease change due to long term care provision. This consideration should be provided more research in future.
References


