VOLUME 7 | ISSUE 1 | **JANUARY 2016**

ISSN 1757-9597

Longitudinal and Life Course Studies: International Journal

Published by

SLLS Society for Longitudinal and Life Course Studies

Inside this issue

- Childhood economic hardship and chronic disease in mid-life
- Trends in time-of-day energy and macro-nutrient distributions from age 10 to 13
- Relationships between early childhood body mass index and height
- New method for handling missing data in the Australian Longitudinal Study of Youth
- Overview of the Swiss Household Panel Study - *Study Profile*
- Origins of health inequalities: the case for Allostatic Load - *Debate*

Co-sponsors:

VU university medical center

ISER INSTITUTE FOR SOCIAL & ECONOMIC RESEARCH

SRC SURVEY RESEARCH CENTER UNIVERSITY OF MICHIGAN

LLCS EDITORIAL BOARD

SUBSCRIPTIONS

For immediate access to current issue and preceding 2015 issues (Volume 6):

Annual Individual rate: **£20 (discounted rate £55 for 3 years)**.

Annual Library **rate: £200** to register any number of library readers.

Annual Society for Longitudinal and Life Course Studies (SLLS) Membership: Individual **£65,** ***Student*** **£26,** ***Corporate*** **£300**.

SLLS members have automatic free access to the journal among many other benefits. For further information about SLLS and details of how to become a member, go to http://www.slls.org.uk/#!services/ch6q

All issues are accessible free of charge 12 months after publication.

Print on demand

An attractive printed version of each Issue of the journal, including all back numbers, is available at a price of £10 (plus postage and packaging). If you would like to purchase a full set or individual copies, visit the SLLS Bookshop at http://www.slls.org.uk/#!journal-bookshop/czkc Depending on distance, you will receive the order within two weeks of submitting your order.

Please note

The reselling of personal registration subscriptions and hard copies of the journal is prohibited.

INTRODUCTION

1 – 2 **Editorial**
John Bynner

PAPERS

3 – 24 **Heterogeneity in chronic disease outcomes among women and men in midlife: examining the role of stability and change in childhood economic hardship**
cole Etherington, Andrea Willson, Kim Shuey

25 – 40 **Temporal trends in energy and macronutrient distribution in meals eaten by children from the Avon Longitudinal Study of Parents and Children**
Suzana Almoosawi, Victoria Cribb, Pauline Emmett, Alison Lennox

41 – 52 **Bi-directional relationships between body mass index and height from three to seven years of age: an analysis of children in the United Kingdom Millennium Cohort Study**
Laia Bécares, Yvonne Kelly, Scott Montgomery, Amanda Sacker

53 – 63 **Handling attrition and non-response in longitudinal data with an application to a study of Australian youth**
Joy Cumming, Harvey Goldstein

STUDY PROFILE

64 – 78 **The Swiss Household Panel Study: Observing social change since 1999**
Robin Tillmann, Marieke Voorpostel, Erika Antal, Ursina Kuhn, Florence Lebert, Valérie-Anne Ryser, Oliver Lipps, Boris Wernli

COMMENT AND DEBATE

79 – 103 **Origins of heath inequalities: the case for Allostatic Load**
Cyrille Delpierre, Cristina Barbosa-Solis, Jerome Torrisani, Muriel Darnaudery, Melanie Bartley, David Blane, Michelle Kelly-Irving, Tony Roberston, Per E. Gustafsson, Linn Getz, Margret Olafia Tomasdottir

LLCS Journal can be accessed online at: www.llcsjournal.org

Published by
The Society for Longitudinal and Life Course Studies
Institute of Education, 20 Bedford Way, London, WC1H 0AL

info@slls.org.uk http://www.slls.org.uk

SLLS Society for Longitudinal and Life Course Studies

SAVE THE DATE!

SLLS International Conference

'Education and the Life Course: Determinants and Consequences of Unequal Educational Opportunities'

Bamberg, Germany
5 - 8 October 2016

(Pre-conference workshop on 5.10.16, with main conference starting on 6.10.16)

A call for papers and symposia to be announced in February 2016.

The fee for conference registration will be discounted for SLLS members so please consider joining if you have not already done so! Please visit the membership page of the website for full details of how to join online: http://www.slls.org.uk/#!services/ch6q

Registration to open in July 2016.

Further information will be posted on the SLLS website as it becomes available.

www.slls.org.uk

Editorial

John Bynner Executive Editor

This issue sees the LLCS journal passing the 2,000 mark for readership. Citations of journal articles have also been steadily increasing with more than 20 citations of an article now commonplace – a good reflection of the impact the journal is having on longitudinal and life course studies literature.

A steady flow of papers across the scientific areas the journal covers is the lifeblood on which its continuing success depends. And a key source for that is the Society for Longitudinal and Life Course Studies (SLLS) annual conference.

This year's highly successful Dublin conference in October continued the trend of expansion with 330 people participating and just over 300 papers and posters presented. Next year's conference, scheduled for October 8-12 in Bamberg, Germany is likely to continue the upward trend as will the following year's event, likely to be in Copenhagen. Conference presentations are the first step towards publication, which we obviously hope will increasingly be in LLCS.

A large part of a journal's reputation is the rigour and quality of the review process through which the published papers have progressed. LLCS has benefitted from the high standards set by our excellent panel of reviewers. This is a good opportunity to thank them for the quality of the inputs they bring to this time-consuming and demanding task.

This issue continues the trend of high-quality papers with the main emphasis this time on an area that has dropped a little in submissions recently – health and the life course. The issue starts with three excellent papers concerned in various ways with the foundations of health risk factors in childhood. The first uses US Panel Study of Income Dynamics (PSID) data to analyse the effects of childhood disadvantage on chronic disease outcomes in mid-life. The second uses diary data to investigate the longitudinal trends in time-of-day energy and macronutrient intake at ages 10 and 13 in the Bristol-based ALSPAC study. The final paper of the trio is devoted to bidirectional relationships between child body mass and height through ages three, five and seven in the UK-wide Millennium Cohort Study.

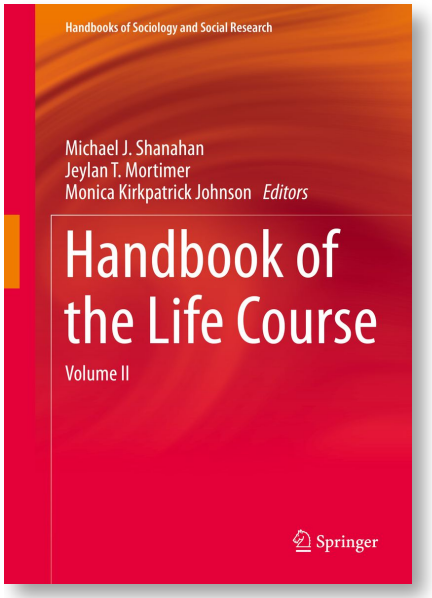
The issue moves next to methodology, with the presentation of a new imputation method for missing data, as applied in the Australian Longitudinal Study of Youth. The mode then shifts again in a *Study Profile* of the highly productive Swiss Household Panel study established in 1991. We then return to health through our new journal section, *Comment and Debate*.

Attention turns this time to the origins of health inequalities. The case is made, and elaborated upon by expert commentators, for attributing such phenomena to adverse social conditions in development. These impact on, and become 'embodied' in, the biological systems through which health is maintained – 'allostatic load'. In summary, *how the social becomes the biological*.

Taken together these papers demonstrate the great value of longitudinal data in understanding life course processes and outcomes, addressing problems and pointing to solutions that could not be identified in any other way. As argued in the recently published *Handbook of the Life Course Volume 2** the insights gained from interdisciplinary population-wide longitudinal studies underwrite the substantial post-millennial public investment in them. Further back, in the prescient words of Howard Newby, ex-UK Economic and Social Research Council director: "As we move increasingly towards scientific collaboration – not least in the social sciences – large scale longitudinal cohort studies will come to represent the social science equivalent of the large scale facilities and laboratories enjoyed by our colleagues in the natural sciences." Longitudinal and life course study may not have reached the stage of the Hadron Collider quite yet but working towards the model it offers for the future institutionalisation of life course science could not be more pressing.

Finally, I would like to extend our sincere thanks to former LLCS Health Sciences editor and founding Section Editor Mike Wadsworth, who copy edited the *Allostatic Load* debate published in this issue.

*Shanahan, M., Mortimer, J.T. & Johnson, M. K., Springer (Eds), New York, 1916; ** Preface to Bynner, J (Ed) *The use of longitudinal cohort studies in the policy process*, Anglo German Foundation & ESRC, 1993.



M.J. **Shanahan**, J.T. **Mortimer**, M. **Kirkpatrick Johnson** (Eds.)

Handbook of the Life Course

Volume II

Series: Handbooks of Sociology and Social Research

- **Takes stock of a fast-growing, complex and multidisciplinary field**
- **Emphasizes qualitative novelty in life course research**
- **Clearly describes the opportunities and challenges of the field**

Building on the success of the 2003 Handbook of the Life Course, this second volume identifies future directions for life course research and policy. The introductory essay and the chapters that make up the five sections of this book show consensus on strategic "next steps" in life course studies. These next steps are explored in detail in each section: Section I, on life course theory, provides fresh perspectives on well-established topics, including cohorts, life stages, and legal and regulatory contexts. It challenges life course scholars to move beyond common individualistic paradigms. Section II highlights changes in major institutional and organizational contexts of the life course. It draws on conceptual advances and recent empirical findings to identify promising avenues for research that illuminate the interplay between structure and agency. It examines trends in family, school, and workplace, as well as contexts that deserve heightened attention, including the military, the criminal justice system, and natural and man-made disaster. The remaining three sections consider advances and suggest strategic opportunities in the study of health and development throughout the life course; methodological innovations, including qualitative and three-generational longitudinal research designs, causal analysis, growth curves, and the study of place; and building bridges between life course research and public policy.

1st ed. 2016, X, 720 p. 54 illus., 20 illus. in color.

Heterogeneity in chronic disease outcomes among women and men in midlife: examining the role of stability and change in childhood economic hardship

Cole Etherington Western University, Canada
netherin@uwo.ca
Andrea Willson Western University, Canada
Kim Shuey Western University, Canada

(Received April 2015 Revised September 2015) dx.doi.org/10.14301/llcs.v7i1.352

Abstract

In this study, we advance existing research on the long-term effects of childhood disadvantage on health in adulthood by examining how the timing and duration of childhood economic hardship differentiates between those at low and high risk of chronic disease onset in midlife for women and men, across four different health outcomes. The study uses prospective data on childhood and adulthood from the US Panel Study of Income Dynamics and discrete time hazard models are estimated using logistic regression. Results indicate that, in general, childhood economic context results in an increased risk of multiple diseases for women but not for men. Specifically, women who experienced long-term economic hardship in childhood, or began life in poverty but moved out of poverty in childhood, were more likely to experience the onset of diabetes, arthritis, and cardiovascular diseases in midlife, net of other factors, such as adult resources. The impact of childhood economic hardship on disease onset also varied by age for women but not for men, and this relationship was also dependent on the health outcome examined. This study draws attention to the importance of conceptualising and measuring childhood disadvantage as dynamic, and reveals that the process of cumulative disadvantage may be different for women and men.

Keywords

Cumulative disadvantage, gender and health, life course, childhood economic hardship

Introduction

Improving gender equity in health has been recognised by the World Health Organization as "one of the most direct and potent ways to reduce health inequities overall and ensure effective use of health resources" (Sen & Ostlin 2007: viii.). Gender differences in health are well documented in the United States (US) (see Read & Gorman, 2010 for a review), with women experiencing greater morbidity than men despite living longer. Yet attempts to explain and reduce disparities in health faced by women and men have typically focused on the relationship between health and resources in adulthood, with limited attention to the early origins

of disease and health over the life course.

Through its emphasis on human development and aging as lifelong processes (Elder, Johnson, & Crosnoe, 2003), the life course perspective has directed attention to the 'long arm' of childhood disadvantage, or how early life conditions impact health and other outcomes in adulthood. Research increasingly has recognised the importance of childhood origins in shaping health disparities (Diprete & Eirich, 2006; Murray et al., 2011), yet little work has examined how childhood context may differentially affect men and women. Existing research treats gender as a control variable rather than a focal point (e.g. Bowen, 2010), often attempting to 'explain away' gender differences rather than examining how social factors may operate differently for women and men and lead to divergent health trajectories and heterogeneity within groups (Evans-Campbell, Lincoln & Takeuchi, 2010). Further, childhood disadvantage has been treated as static, rather than as a dynamic process involving stability and change over time (e.g. Lemelin et al., 2009; Pudrovska & Anikputa, 2013; Walsemann, Ailshire, Bell & Frongillo, 2012), and its effects rarely compared across health outcomes.

The current study advances research on childhood disadvantage, gender, and health by conceptualising and measuring childhood economic context as a dynamic process that may affect disease onset in midlife differently for women and men. Specifically, we take into consideration stability and change in the experience of childhood poverty and its impact on health in midlife. Using the US Panel Study of Income Dynamics (PSID), these relationships are examined across four chronic disease outcomes that are among the most prominent causes of morbidity and mortality in the United States.

Background

Diabetes, hypertension, arthritis, stroke, heart attack and heart disease are among the most common causes of morbidity and mortality in the US (Centers for Disease Control and Prevention, 2014; Gluckman & Hanson, 2005; Heron 2007). In general, men tend to experience more life-threatening chronic diseases at younger ages, while women have higher rates of chronic debilitating conditions (Bird & Rieker, 2008). Across all age groups, heart disease is more prevalent among men than women, although it remains the leading cause of death for both genders (National Center for Health Statistics, 2009). Partly due to heart attack occurring at later ages for women, nearly half of all fatal heart attacks each year in the US occur in women. For men up to age 75, the incidence of stroke is higher than in women, but this trend reverses in adults 85 years and older (Petrea et al., 2009). Women also have a higher lifetime risk of stroke (Petrea et al., 2009). Gender differences do not appear with regard to the overall prevalence of hypertension (33.6% of men and 32.3% of women), but prevalence is higher for men under 25 (Doumas, Papademetriou, Faselis & Kokkinos, 2013). Finally, women experience higher rates of arthritis than men (26% vs. 19%) while a slightly higher percentage of men have diabetes than women (14% vs. 11%) (Centers for Disease Control and Prevention, 2014).

Social explanations of differences in men and women's health outcomes have centred on differential access to protective resources, including income and education, as well as exposure to factors that negatively affect health, such as behavioural risk factors, in adulthood (Bird & Rieker, 2008). Life course research in both the United States and many European countries, however, has consistently linked each of these chronic disease outcomes to childhood socioeconomic circumstances (Blackwell, Hayward, & Crimmins, 2001; Danese, Pariante, Caspi, Taylor & Poulton, 2007; Drakopoulos, Lakioti, & Theodossiou, 2011; Hamil-Luker & O'Rand, 2007; Johnson & Schoeni, 2011; Kivimaki et al., 2006; Luo & Waite, 2005; Maty, Lynch, Raghunathan & Kaplan, 2008; Mckenzie, Carter, Blakely & Ivoer, 2011; Mensah & Hobcraft, 2008). Cumulative dis/advantage is a key framework used to conceptualise this link, referring to a process through which initial disadvantage or advantage is compounded or amplified over time to produce heterogeneity in life course outcomes, such as health (O'Rand, 1996). In other words, the relationship between socioeconomic resources and health begins in early life and is magnified over time. Widening health disparities between advantaged and disadvantaged groups with age suggest that processes of cumulative dis/advantage operate across the life course (Brown, O'Rand, & Adkins, 2012;

Dupre, 2007; Lynch, 2003; Shuey & Elder, 2008; Shuey & Willson, 2014; Willson et al., 2007).

Early life inequalities in socioeconomic environment are thought to initiate processes of cumulative advantage and disadvantage which lead to divergent trajectories of health across the life course (Corna, 2013). Research also indicates that the timing, duration, and sequencing of childhood exposure to economic hardship are critical for many adulthood outcomes, including health (Shuey & Willson, 2014; Wagmiller, Lennon, Kuang, Alberti & Aber, 2006). Existing models of cumulative dis/advantage, however, differentially emphasise the importance of each temporal complexity (see Shuey & Willson, 2014, for a review). Such approaches also do not take into account heterogeneity in childhood circumstances, ignoring the way in which socioeconomic circumstances can improve or deteriorate throughout childhood, as well as issues of timing related to the onset of disadvantage. Instability in resources in childhood and throughout the life course often occurs, challenging notions of disadvantage that view poverty as a long-term and irreversible state (McDonough & Berglund, 2003; McDonough, Sacker, & Wiggins, 2005; Western, Bloome, Sosnaud & Tach, 2012). Little attention has been given to patterns of change in childhood circumstances. Existing research has also relied heavily on retrospective data and static measures of childhood socioeconomic status (SES). Measures of childhood SES used in previous studies have included: parents' education (e.g. Bowen, 2010; Lemelin et al., 2009; Walsemann et al., 2012), parents' occupation (e.g. Gustafsson & Hammarstrom, 2012; Hallqvist, Lynch, Bartley, Lang & Blane, 2004; Lidfelt, Li, Hu, Manson, & Kawachi, 2007; Maty et al., 2008; Pudrovska & Anikputa, 2013), family income at a single point in childhood (e.g. Fothergill, Ensminger, Green, Robertson & Juon, 2009), or some combination of factors, such as receipt of welfare, parental divorce, and father's education (e.g. Montez & Hayward, 2014; Schafer, Markus & Ferraro, 2012).

While these studies have made key contributions to our understanding of life course processes of health, they are not able to addresses the effects of dynamic and differing experiences of economic hardship. For example, long-term exposure to childhood disadvantage appears to have the strongest negative effect on adult achievement outcomes and is harmful to health in adulthood (e.g., Shuey & Willson, 2014; Wagmiller et al., 2006). However, research also suggests that transitions into or out of sustained poverty in childhood have distinct effects on health. For example, deteriorating health in mid-life is more likely among those who transition into sustained economic hardship in childhood, while those whose families move out of poverty during childhood have health trajectories similar to those who never faced economic hardship (Shuey & Willson, 2014). Accordingly, the timing and duration of experiences of disadvantage in childhood are important to understanding life course trajectories of health. Yet studies tend to draw conclusions about long-term processes based on single snapshots in time (e.g. Pudrovska & Anikputa, 2013).

Although research demonstrates processes of cumulative advantage and disadvantage begin early in life, it should not be assumed that they operate similarly across groups (George, 2005). Little empirical attention has been given to whether cumulative processes of inequality that begin in childhood may differ for men and women. Such differences are likely given gender differences in biological disease processes, responses to stressors and social conditions, and access to resources (Taylor et al. 2000; Zunzunegui, Alvarado, Béland & Vissandjee, 2008). For example, women earn less than men even after controlling for education, work experience, and marital status (Hogan & Perrucci, 2007), dominate temporary and part-time jobs (Fuller & Vosko, 2008; Prokos, Padavic, & Schmidt, 2009), and are more likely to experience discontinuity in their employment histories due to their role as primary caregiver (Moen, Robison, & Fields, 1994). Research that has incorporated gender into the study of childhood disadvantage and adult health suggests that childhood socioeconomic disadvantage predicts psychological distress, depressive symptoms, body mass index (BMI), cardiovascular disease, metabolic syndrome, diabetes and risk of heart attack for women significantly more than for men (Fitzmaurice & Buka, 2002; Gilman, Kawachi, Pudrovska & Anishkin, 2013; Gustafsson & Hammarstrom, 2012; Hamil-Luker & O'Rand 2007; Lemelin et al., 2009; Lipowicz, Kozieł, Hulanicka & Kowalisko, 2007; Maty et al., 2008; Walsemann et al., 2012). Pudrovska and

Anikputa (2013) find evidence of an indirect relationship between early life SES and health through the operation of health behaviours for women only, though it should be noted that early life conditions are only measured at a single point in time. It is likely that cumulative processes differ by gender; however, research has not adequately problematised heterogeneity among women and men to understand how the timing and duration of childhood economic hardship generate health inequality within these groups.

Finally, in examining these processes, it is important to consider multiple measures of health rather than single or monolithic measures for two reasons. First, "different health conditions vary in their etiologies" (Brown et al., 2012, p. 360). Therefore, combining multiple health concerns into an all-encompassing measure risks overlooking the differential accumulation of risk factors that lead to different conditions. Second, the direction and magnitude of gender differences in health vary depending on the condition examined (Denton, Prus, & Walters, 2004). It is therefore useful to examine multiple health conditions in order to understand similarities and differences in the processes leading to each and to capture important variations by gender. Whereas past studies on cumulative disadvantage, gender and health have considered a single or limited number of health outcomes, the present analysis examines multiple chronic diseases and compares how dynamic experiences of childhood economic hardship are related to each for men and women.

Research questions

Based on the above considerations, we ask whether trajectories of childhood economic hardship are associated with chronic disease outcomes in midlife for both women and men. In other words, we examine patterns of stability and change in childhood circumstances and their association with processes of cumulative health disadvantage for both women and men. Previous literature suggests childhood disadvantage may be more detrimental for women's health outcomes, so it is possible that the relationship between trajectories of childhood economic hardship and some health outcomes will differ for men and women. We therefore investigate whether the observed relationships between childhood economic hardship and disease onset vary by the health outcome under investigation.

Methods

Data

This study uses the US Panel Study of Income Dynamics (PSID), an ongoing survey that began in 1968 with a nationally representative sample of 4,802 families (Panel Study of Income Dynamics, 2013). Information was collected on all household members, primarily from the household head, annually until 1997 when interviewing became biennial. In married families, the 'head' is the husband unless he is incapable of being interviewed and 'wife' is the female in a married or cohabitating couple (McGonagle, Schoeni, Sastry & Freedman, 2013). The 'head' can also refer to a single female. Annual response rates have ranged from 95 to 98 percent (McGonagle et al., 2013). Interviews are conducted via telephone. The latest wave of available data used in this analysis was collected in 2011. Children of PSID families who leave their parents' homes also become PSID family units, and sample representativeness has been maintained (McGonagle et al., 2013). The multi-generational design of data collection enables adult children to be linked to their parents. Data on childhood socioeconomic environment provided prospectively by parents at the time the child was in the parental home avoids recall bias in childhood conditions. An oversample of families from low-income neighborhoods was included in the original sample design of the PSID, which enables the differentiation of various experiences of childhood economic hardship. Finally, the PSID contains rich information on various health outcomes, health behaviours, and other important covariates such as income, employment, and marital status. The PSID is one of the few large survey data sets worldwide that has followed multiple generations of families for such a long period of their lives, and as such provides a unique opportunity to prospectively examine the long-term health effects of childhood economic hardship.

Analytic sample

This study focuses on individuals who were newborn to eight years old in 1968. This age range is

particularly useful for this analysis as, during the observation period, these individuals enter a stage of the life course in which many health problems begin to emerge. Latent classes of childhood economic hardship experience were estimated for the full sample of these respondents (N=4,167) using data collected from PSID families from 1968-1977 (see Shuey & Willson, 2014). The sample used in multivariate analyses includes the subsample of individuals who remained in the study in adulthood and were a PSID 'head' or a 'wife' at the start of the observation period in 1999 as these are the household members that the PSID collects detailed information on in each survey year (Number of individuals=1,229; 697 women, 532 men).

Missing data is a challenge in any longitudinal study. This paper uses survival analysis, which allows the use of unbalanced panels, meaning individuals who attrited from the PSID after the initial observation year (1999) are still included in the analysis. Additionally, one advantage of the PSID is that, unlike retrospective studies, which do not begin studying individuals until much older ages, many selection processes are observable. Multiple studies have extensively examined the effects of the attrition of this cohort of children from the PSID sample on intergenerational models (e.g., those using family income during the respondent's childhood) with covariates that predict adult health outcomes and demonstrate that the PSID maintains its representativeness over time without strong evidence of attrition bias, with the exception that the effect of higher education on sample attrition is stronger than that of health and that female subsamples demonstrate weaker effects of attrition than males (Fitzgerald, 2011; Halliday, Kimmitt, & Kimmitt, 2012; Meer, Miller, & Rosen, 2003). Previous research also has found that individuals who experienced childhood poverty are less likely to have remained in the PSID to have an observed health outcome in 1999 when health data began to be collected (see Shuey & Willson, 2014). Any selective attrition with respect to health will likely lead to an underestimate of the impact of childhood economic hardship. Taken together, this indicates that, while not significantly biased, results from this study are likely conservative estimates of the association of childhood economic hardship and adult health (Shuey & Willson, 2014).

Measures

Disease outcomes

Four disease outcomes are assessed in this study: high blood pressure, diabetes, arthritis, and a measure consisting of heart attack, heart disease, and stroke. Stroke, heart attack, and heart disease were grouped together due to relatively low prevalence levels in middle age in addition to all affecting the heart and circulatory system (Johnson & Schoeni 2011). The conditions are measured by responses to the question: "Has a doctor or health professional ever told you that you have had–?" Respondents were asked this question in each survey wave from 1999 to 2011. It is possible for individuals to have comorbidities, but each condition was examined separately and each measure included all those individuals who reported having been diagnosed with that particular health condition. It should be acknowledged that these measures are somewhat non-specific and the measure of arthritis does not distinguish between types of arthritis, which are experienced at different rates by men and women and have differing etiologies. Variation in the experience of arthritis could contribute to gender differences in association with childhood economic hardship, however we believe this is minimal given the similar rates of arthritis among the men and women in the sample. We further discuss the potential implications in the discussion section.

Childhood economic hardship

Children's histories of economic hardship were analysed over a 10-year period, from 1968 (when the children were 0-8 years old) to 1977. A child was considered to be living in poverty in a given year if the family's total annual income fell below 125% of the official US poverty threshold.[i] These indicators and repeated measures of latent class analysis were used to identify subgroups of individuals with similarities in their experience of economic hardship in childhood (see Shuey & Willson, 2014, for a detailed discussion). Based on fit statistics from the latent class models, and the previous literature (Wagmiller et al., 2006), it was determined that there were four groups into which respondents could be classified: non-poor, moving into poverty, moving out of poverty, and

long-term poverty. Those who moved into poverty began with a relatively low risk of experiencing poverty, which increased as they reached and transitioned into adolescence, while those who moved out of poverty had a relatively high risk of poverty in early childhood that dropped steadily as they approached late childhood. The long-term poor had a very high probability of exposure to poverty during the entire period of observation (1968-1977), and the non-poor had a very low probability of experiencing poverty during this period.

Other covariates

Both adult resources and health behaviours are associated with childhood disadvantage and adult health (e.g. Hayward & Gorman, 2004; Pudrovska & Anishkin, 2013). All covariates belonging to these categories were included as time varying, with the exception of education (coded as less than high school, high school, some post-secondary, and post-secondary). Employment status and marital status were dichotomised (1=employed; 1=married). Total household income was lagged by one year, adjusted for inflation, and logged for each year of observation. Based on considerations from previous literature (Hamil-Luker & O'Rand, 2007; Kagotho, 2009), frequency of heavy physical activity was coded as 1=never engages in physical activity. Drinking and smoking were also dichotomised, with 1=drinks one or more drinks per day and 1=current smoker (see Kagotho, 2009). Race/ethnicity, which is strongly associated with both childhood poverty and adult health (Lynch, 2008; National Poverty Center, 2013), was also included as a covariate and coded as non-Hispanic black (1) and non-Hispanic white (0). Other racial/ethnic groups were not included due to an inadequate number of observations.

Analytic strategy

Survival analysis was used to determine how experiences of economic hardship in childhood affect the risk of disease onset in midlife. Multivariate analyses were clustered by person ID. The unit of analysis for all analyses was person-years (*N*=15,624). Analyses were weighted using the PSID longitudinal weight to adjust for oversampling of low-income families as well as for attrition (McGonagle et al., 2013). The population at risk for each disease outcome was defined as those individuals who had not experienced disease onset for the particular condition under investigation before age 40. Thus, the models predict the likelihood that an individual would develop a condition by the end of the observation period assuming they did not have it by age 40. This restriction was imposed for two reasons: to address left-censoring, and also, because the focus of this study was to examine disease onset in mid-life.

Multivariate discrete-time hazard models were estimated using logistic regression. These models were appropriate given the fairly large intervals at which the presence of each disease was measured (years) as well as the censoring of some data. Women and men were analysed as separate groups. Such an approach provides greater ease with which to assess the significance of covariate effects within each group (Phillips & Sweeney, 2005). In addition, it allows us to assess how childhood economic hardship produces heterogeneous health outcomes within women and men, similar to Hamil-Luker and O'Rand (2007). This is particularly important considering our limited understanding of the unique health experiences of men and women who face economic hardship in childhood.

In analyses of temporal dependence, the risk of experiencing the onset of a chronic condition was found to change over time for both women and men; therefore, age was included in the multivariate models as a categorical variable: 40-45 (0) and 46-52 (1). These categories were chosen as they are reflective of a division between early and late middle-age. The proportional hazards assumption was also evaluated for women and men. This assumption implies that predictor variables have uniform effects across time, or that there are no interactions between predictors and time (see Allison, 2010; Borucka, 2013). The proportional hazards assumption was violated for women with regard to the effect of poverty class on health, indicating that an interaction term between age and poverty class was necessary for models of women's health to allow for non-proportional hazards (Allison, 2010; Borucka, 2013). The assumption was not violated for men; therefore, models predicting men's outcomes did not include the aforementioned interaction terms. In other words, the effect of childhood economic hardship on disease onset changes over time for women but not men. This prevents testing for significant differences

across the two groups, as the model specification for women and men is different; however, the focus of this paper is to examine whether and how childhood economic hardship produces heterogeneous health outcomes among women and among men. Results will demonstrate how the timing and duration of childhood economic hardship impacts the health of women and men.

Results

Descriptive results

Weighted proportions and means by gender are presented in table 1. On average, over the observation period, the same percentage of women and men reported having diabetes (5%) and stroke, heart disease, or heart attack (3%). In any given year, about 8% of men reported arthritis compared to 12% of women. Over the observation period, men and women experienced similar rates of high blood pressure, at 17% and 16%, respectively. Rates of childhood economic hardship were roughly similar across women and men. High school graduates make up the largest proportion of education categories for both men and women (39% and 34%, respectively). More men (43%) than women (25%) reported drinking one or more drinks per day and more women (33%) than men (25%) reported never exercising. Smoking rates were similar at 25% for women and 23% for men.

Table 1. Descriptive statistics by gender (weighted), 1999-2011 PSID

Variable	Women	Men
Diabetes		
Yes	0.05	0.05
No	0.95	0.95
High blood pressure		
Yes	0.16	0.17
No	0.84	0.83
Arthritis		
Yes	0.12	0.08
No	0.88	0.82
Stroke,heart disease, heart attack		
Yes	0.03	0.03
No	0.97	0.97
Childhood poverty status		
Non-poor	0.75	0.75
Move into poverty	0.05	0.04
Long-term poor	0.10	0.08
Move out of poverty	0.09	0.13
Race/Ethnicity		
Non-Hispanic Black	0.27	0.20
Non-Hispanic White	0.73	0.80
Age	41.37	41.27
Adult education		
<High school	0.10	0.07
High school	0.34	0.39
Some post-secondary	0.29	0.22
Post-secondary	0.27	0.32
Employment status		
Employed	0.22	0.90
Not employed	0.78	0.10
Marital status		
Married	0.66	0.76
Not married	0.34	0.24
Income (median)	60851	78087
Smoking status		
Yes	0.25	0.23
Drinking frequency		
1+/Day	0.25	0.43
Physical activity		
Never engages	0.33	0.25
N	697	532
N(person-years)	8848	6776

Notes: Proportions for disease outcomes refer to the average proportion in each category over the observation period. Data were converted into person-year format required for survival analysis.

Multivariate analyses

In all models for women, the reference group is women in early midlife (aged 40-45) who did not experience economic hardship in childhood. The comparison group is women in late midlife (aged 46-52) who have experienced some form of poverty.

Diabetes (table 2)

In model 1, women in late midlife who experienced long-term poverty in childhood were approximately eight times more likely to experience the onset of diabetes by the end of the observation period compared to their younger counterparts who did not experience poverty (p=0.006). In addition, older women who moved out of poverty in late childhood were still 16 times more likely to experience the onset of diabetes (p=0.022). Conversely, younger women were less likely to experience diabetes even if they experienced long-term poverty as children (OR=0.20; p=0.026). With the introduction of adult resources, health behaviours and other covariates, women belonging to both of these poverty classes remained more likely to experience the onset of diabetes by the end of the observation period relative to younger women who did not experience poverty (OR=9.01, p=0.007; OR=12.97, p=0.032). Stated as probability, women who experienced long-term poverty had a 90% chance of developing diabetes by the end of the observation period and women who moved out of poverty had a 93% chance. For women, then, the effects of childhood economic hardship on diabetes onset in midlife vary by age, with the impact of long-term poverty and poverty in early childhood manifesting in late middle age. Conversely, childhood economic hardship was not a significant predictor of diabetes onset for men.

Table 2. Discrete-time logistic regression estimated effects of childhood economic hardship on the risk of onset of Diabetes within 12 years, by gender: 1999-2011 PSID

Independent variable	Model 1				Model 2[a]				Model 3[b]				Model 4[c]			
	Women		Men		Women		Men		Women		Men		Women		Men	
	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value
Childhood economic hardship (non-poor)																
Move into poverty	0.92	0.889	2.15	0.285	1.17	0.819	1.75	0.371	0.94	0.929	1.83	0.350	0.70	0.652	1.54	0.433
Long-term poverty	0.20	0.026	0.76	0.289	0.17	0.019	0.73	0.422	0.15	0.011	0.72	0.401	0.10	0.007	0.49	0.168
Move out of poverty	0.15	0.081	2.21	0.279	0.15	0.126	1.78	0.429	0.13	0.091	1.63	0.518	0.10	0.051	1.29	0.718
Age (40-45)																
46-52	2.04	0.096	7.42	0.000	2.44	0.045	8.90	0.000	2.51	0.076	9.01	0.000	2.57	0.062	9.82	0.000
Childhood economic hardship X age																
Move into poverty X 46-52	1.06	0.921			0.76	0.688			1.02	0.982			0.90	0.887		
Long-term poverty X 46-52	8.17	0.006			8.43	0.006			9.07	0.008			9.01	0.007		
Move out of poverty X 46-52	15.67	0.022			12.37	0.034			11.88	0.038			12.97	0.032		
Constant	0.29	0.002	0.12	0.000	19.75	0.190	0.00	0.002	6.27	0.465	0.00	0.002	6.77	0.437	0.00	0.002

Notes:
Number of observations (women) = 810. Number of observations (men) = 594.
[a] Model 2 controls for adult resources: education, income, employment status, and marital status.
[b] Model 3 controls for the variables specified in Model 2 and adds adult health behaviours: smoking, drinking, and physical activity.
[c] Model 4 controls for the variables specified in Models 2 and 3 and adds race/ethnicity.

Table 3. Discrete-time logistic regression estimated effects of childhood economic hardship on the risk of onset of high blood pressure within 12 years, by gender: 1999-2011 PSID

Independent variable	Model 1				Model 2[a]				Model 3[b]				Model 4[c]			
	Women		Men		Women		Men		Women		Men		Women		Men	
	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value
Childhood economic hardship (Non-poor)																
Move into poverty	1.13	0.758	1.52	0.280	1.22	0.635	1.65	0.200	1.40	0.412	1.63	0.235	1.50	0.321	1.66	0.228
Long-term poverty	0.65	0.108	0.97	0.861	0.72	0.259	0.98	0.937	0.78	0.396	1.01	0.953	0.85	0.589	0.95	0.838
Move out of poverty	0.59	0.192	0.95	0.808	0.68	0.340	1.03	0.873	0.70	0.392	1.03	0.891	0.76	0.504	0.96	0.897
Age (40-45)																
46-52	5.90	0.000	7.69	0.000	6.85	0.000	8.30	0.000	6.65	0.000	8.17	0.000	6.70	0.000	8.17	0.000
Childhood economic hardship X age																
Move into poverty X 46-52	0.62	0.289			0.53	0.170			0.55	0.191			0.55	0.194		
Long-term Poverty X 46-52	1.51	0.191			1.31	0.394			1.34	0.366			1.33	0.379		
Move out of poverty X 46-52	1.81	0.202			1.52	0.380			1.65	0.294			1.62	0.310		
Constant	0.17	0.000	0.12	0.000	0.20	0.082	0.17	0.049	0.26	0.147	0.17	0.062	0.30	0.225	0.15	0.055

Notes:
Number of observations (women) = 2592. Number of observations (men) = 2020.
[a] Model 2 controls for adult resources: education, income, employment status, and marital status.
[b] Model 3 controls for the variables specified in Model 2 and adds adult health behaviours: smoking, drinking, and physical activity.
[c] Model 4 controls for the variables specified in Models 2 and 3 and adds race/ethnicity.

High blood pressure (table 3)

Childhood economic hardship was not a significant predictor of the midlife onset of high blood pressure for women or men in any of the four models.

Arthritis (table 4)

In model 1, women in late middle-age who moved out of poverty were about six times more likely to experience arthritis compared to their younger peers who did not experience childhood poverty (p=0.006). This relationship persisted when adult resources, health behaviours, and race/ethnicity were taken into account (OR=7.75, p=0.011 in Model 4). In other words, these women had an 89% chance of developing arthritis. For men, however, childhood economic hardship was not a significant predictor of arthritis onset in midlife.

Stroke, heart disease, heart attack (table 5)

In model 1, women in late midlife who lived in long-term poverty as children were about six times more likely to experience stroke, heart disease, or a heart attack compared to their younger peers who did not experience childhood poverty (p=0.001). This relationship remained the same controlling for adult resources, health behaviours, and race/ethnicity (OR=6.02, p=0.002; Model 4). That is, these women had an 86% probability of developing stroke, heart disease, or heart attack. For men, having moved into poverty in childhood was associated with being three times more likely to experience stroke, heart disease, or a heart attack by the end of the observation period (p=0.004; model 1). With introduction of adult resources in model 2, this increased to four times more likely (p=0.001). When health behaviours were added in model 3, men who moved into poverty as children were nearly six times more likely to experience stroke, heart disease, or a heart attack compared to their non-poor peers (p=0.001). This remained true in model 4.

Table 4. Discrete-time logistic regression estimated effects of childhood economic hardship on the risk of onset of arthritis within 12 Years, by gender: 1999-2011 PSID

Independent variable	Model 1				Model 2[a]				Model 3[b]				Model 4[c]			
	Women		Men		Women		Men		Women		Men		Women		Men	
	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value
Childhood economic hardship (Non-poor)																
Move into poverty	0.35	0.085	0.82	0.535	0.30	0.043	0.64	0.233	0.29	0.034	0.67	0.340	0.31	0.053	0.66	0.297
Long-term poverty	0.55	0.066	0.77	0.268	0.49	0.033	0.65	0.193	0.48	0.030	0.65	0.219	0.54	0.105	0.47	0.108
Move out of poverty	0.19	0.005	1.62	0.203	0.18	0.008	1.39	0.435	0.17	0.008	1.47	0.357	0.19	0.011	1.07	0.901
Age (40-45)																
46-52	4.03	0.000	6.07	0.000	4.38	0.000	6.40	0.000	4.60	0.000	6.43	0.000	4.57	0.000	6.62	0.000
Childhood economic hardship X age																
Move into poverty X 46-52	2.56	0.167			2.57	0.162			2.49	0.174			2.58	0.151		
Long-term poverty X 46-52	1.63	0.209			1.46	0.329			1.45	0.333			1.48	0.307		
Move out of poverty X 46-52	6.18	0.006			5.55	0.011			5.53	0.012			5.75	0.011		
Constant	0.27	0.000	0.15	0.000	1.11	0.931	0.30	0.481	1.03	0.981	0.18	0.330	1.39	0.804	0.18	0.330

Notes:
Number of observations (women) = 1944. Number of observations (men) = 950
[a] Model 2 controls for adult resources: education, income, employment status, and marital status.
[b] Model 3 controls for the variables specified in Model 2 and adds adult health behaviours: smoking, drinking, and physical activity.
[c] Model 4 controls for the variables specified in Models 2 and 3 and adds race/ethnicity.

Table 5. Discrete-time logistic regression estimated effects of childhood economic hardship on the risk of onset of stroke, heart disease, heart attack within 12 Years, by Gender: 1999-2011 PSID

Independent variable	Model 1				Model 2[a]				Model 3[b]				Model 4[c]			
	Women		Men		Women		Men		Women		Men		Women		Men	
	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value	Odds ratio	p-value
Childhood economic hardship (Non-poor)																
Move into poverty	1.35	0.534	2.92	0.004	1.43	0.513	4.03	0.001	1.99	0.215	5.83	0.001	2.03	0.217	5.99	0.000
Long-term poverty	0.33	0.022	0.73	0.399	0.32	0.053	0.43	0.146	0.39	0.110	0.44	0.158	0.40	0.173	0.14	0.000
Move out of poverty	0.71	0.421	1.06	0.915	0.76	0.540	1.09	0.864	0.76	0.542	1.05	0.928	0.78	0.646	0.44	0.18
Age (40-45)																
46-52	2.13	0.015	4.52	0.000	2.35	0.008	6.21	0.000	2.42	0.012	5.95	0.000	2.40	0.013	8.91	0.000
Childhood economic hardship X age																
Move into poverty X 46-52	1.02	0.986			1.02	0.985			0.78	0.793			0.79	0.808		
Long-term poverty X 46-52	6.07	0.001			5.80	0.002			5.98	0.002			6.02	0.002		
Move out of poverty X 46-52	2.71	0.093			2.62	0.143			3.24	0.102			3.24	1.00		
Constant	0.34	0.000	0.23	0.000	3.08	0.532	1.43	0.883	5.92	0.279	25.08	0.285	6.13	0.302	15.91	0.353

Notes:
Number of observations (women) = 486. Number of observations (men) = 356.
[a] Model 2 controls for adult resources: education, income, employment status, and marital status.
[b] Model 3 controls for the variables specified in Model 2 and adds adult health behaviours: smoking, drinking, and physical activity.
[c] Model 4 controls for the variables specified in Models 2 and 3 and adds race/ethnicity.

Discussion

While studies of gender and health have typically focused on 'explaining away' the gender difference, we take a different approach to this commonly investigated phenomenon through examining how patterns of change and stability in childhood economic hardship initiate processes of disadvantage in health among women and men rather than between them. Although men and women in this study experienced chronic disease at similar rates, the cumulative processes leading to heterogeneity within each group were quite different. Consistent with previous US and European literature, in this analysis childhood economic hardship differentiated between women at low and high risk of chronic disease in midlife but not men (e.g. Gustafsson & Hammarstrom, 2012; Lemelin et al., 2009; Lipowicz et al., 2007; Walsemann et al., 2012). One exception was stroke, heart disease, and heart attack, where childhood economic hardship increased risk of onset for both men and women. This inconsistency with previous research could be the result of a more nuanced and prospective measure of childhood economic hardship than was used in previous studies (e.g. Hamil-Luker & O'Rand, 2007).

Overall, our findings indicate that it is not just long-term poverty that matters for women's health outcomes, but poverty may have lasting effects even after leaving it. For example, long-term poverty in childhood significantly predicted women's risk of onset for diabetes and stroke, heart disease, and heart attack. In addition, women who began life in poverty but moved out of poverty in childhood also were more likely than those who were never in poverty to experience diabetes onset in late midlife, as well as arthritis. These findings reveal the importance of measuring childhood poverty as dynamic rather than capturing it at a single point in time or as a retrospective global measure. Research on the impact of SES in early life on later life health that relies on one measure of parents' SES (e.g. Beebe-Dimmer et al., 2004), and retrospective accounts of childhood SES, which increase the likelihood of recall bias may underestimate the effects of childhood context (e.g. Galobardes et al., 2004). Thus, while research in many Western countries finds disparities in adult health outcomes to be linked to childhood circumstances (e.g. Drakopoulos, Lakioti, & Theodossiou, 2011; Gustafsson & Hammarström, 2012; Johnson & Schoeni, 2011; Kivimaki et al., 2006; Mckenzie et al., 2011; Mensah & Hobcraft, 2008), our study provides support for a more nuanced and dynamic conceptualisation and measurement of childhood poverty with implications for how we understand processes of status and health.

These analyses demonstrate that the timing and duration of childhood economic hardship is associated with more negative health outcomes in later midlife for women than men. It should be noted that findings remained significant and odds ratios remained about the same even after adjustment for covariates. This may be indicative of a direct effect of childhood poverty, as hypothesised by other research (e.g. Diprete & Elrich, 2006). Nevertheless, future research should continue to examine the mechanisms through which childhood poverty affects adult health as this was beyond the scope of this paper. Potential pathways through which childhood disadvantage affects later life health include negative changes in physiology and metabolism in utero (DeBoo & Harding, 2006), disruptions to biological functioning (Miller, Chen & Parker, 2011), increased stress levels (Miech & Shanahan, 2000), and continuous exposure to health-compromising circumstances as a result of disadvantage (Willson, Shuey & Elder, 2007).

Socioeconomic conditions in childhood may be more detrimental to women's health outcomes, in particular, because they are less likely to experience social mobility over the life course than men (Walsemann et al., 2012). In other words, the impact of childhood disadvantage on health is eventually less important for men because of their greater resource attainment in adulthood. Childhood adversity is also associated with reduced accumulation of life course capital, and this relationship is stronger for women (Hamil-Luker & O'Rand, 2007). Additionally, qualitative research suggests a greater accumulation of adversity over the life course for women than men (deVries & Watt, 1996). Disadvantage experienced in childhood may therefore continue to accumulate for women over the life course based on the structuring of opportunities and life chances by gender (Hunt & Annandale, 1999). For example, through disadvantages in paid and unpaid labour,

discriminatory experiences, stress, and caregiving burdens, the impact of childhood economic hardship on health may be aggravated (Coltrane, 2000; Lundberg, 1996; Lundberg & Parr, 2000; Turner & Avison, 2003; Turner, Wheaton & Lloyd, 1995). While gender differences in health are consistent across many European countries and the US (Crimmins, Kim, & Sole-Auro, 2010), the relationship between socioeconomic resources, gender and health can very by welfare regime (Bambra et al., 2009). Due to the unique nature of the social welfare and health care systems in the US, it is possible that the association between childhood economic hardship and adult health found in this analysis may be more pronounced than would be found in countries where SES and health are not as strongly linked. Future research should continue to assess pathways through which heterogeneous trajectories of childhood economic hardship are associated with health among women.

As expected, the observed relationships between childhood economic hardship and health for both men and women also depended on the health outcome examined. For example, women who began childhood with a high risk of exposure to poverty but moved out of poverty as they reached adolescence were more likely to have arthritis in late midlife; in contrast, long-term poverty was most consequential for women's heart disease outcomes. For men, a move in to poverty in childhood predicted stroke, heart disease, and heart attack in midlife, but childhood economic hardship was not a significant predictor of men's other health outcomes. These findings are not surprising given that different health outcomes often have different etiologies (Brown et al., 2012), and also provide support for the dynamic measurement of childhood economic hardship in future research given the nuanced effects on health that emerge (Shuey & Willson, 2014).

This study has several limitations. First, as in any longitudinal analysis that covers a large span of time, there is the potential for unobserved heterogeneity resulting from panel attrition; although there is comprehensive evidence that suggests that the observed relationships were not seriously biased due to attrition, they were potentially weakened. Second, childhood disadvantage was conceptualised and measured as economic hardship based on household income. Future research may also consider such experiences as change in family structure. Based on available disease measures, we were also unable to account for different types of particular diseases, which is most relevant for the measure of arthritis (e.g. osteo vs. rheumatoid arthritis); however, analyses were stratified by gender and disease given known gender differences in patterns of disease. Research demonstrates that women more often experience acute and chronic conditions while men experience more life-threatening disease (Bird & Rieker, 2008). Not surprisingly, we find childhood disadvantage to be associated with diabetes and arthritis for women, and stroke, heart disease, and heart attack for men. Further, men and women in the sample actually reported similar rates of these diseases. The disease measures used were also self-reports of doctor diagnoses. Research demonstrates that lower income individuals are less likely to regularly seek care or visit a doctor (Dubay & Lebrun, 2012), meaning results may be conservative. Finally, this study considers the onset of four physical disease outcomes. Future research should also examine other physical and mental health outcomes.

Despite these limitations, this study is the first to examine whether the timing and duration of childhood exposure to economic hardship generates a process of cumulative disadvantage in health for both men and women. It has demonstrated the importance of measuring poverty as dynamic rather than static in that long-term and an initial high risk of childhood poverty appeared to be more consequential than other experiences of childhood economic hardship for women. Indeed, when childhood economic hardship is measured as dynamic, nuances emerge that have not been captured by other studies. Although this study is unable to concretely determine why different experiences of economic hardship in childhood matter for different disease outcomes, it clearly demonstrates that change and stability in childhood socioeconomic circumstances matter. Further, the link between childhood economic hardship and long-term negative health consequences may be more relevant to women's health over the life course.

Rather than focusing on how socioeconomic circumstances in adulthood explain differences in health between men and women, we focus on how

childhood adversity differentiates risk of onset within each group. Instead of simply controlling for gender, we explore the unique patterns of cumulative disadvantage among women and men. In so doing, we not only find childhood economic hardship to produce heterogeneity in women's chronic disease outcomes (Hamil-Luker & O'Rand, 2007), but also, that the impact of childhood poverty varies by age for women. Little empirical research has examined whether the process of cumulative disadvantage is the same across different sub-groups of the population over time or when the effects of childhood economic hardship may emerge for particular groups. This study suggests that cumulative disadvantage may be a gendered process, with age-dependent effects and heterogeneous health outcomes generally emerging for women, but not for men.

Acknowledgements

This study was supported by an operating grant from Canadian Institutes of Health Research as well as an Ontario Women's Health Scholars Award to Nicole Etherington. The collection of data used in this study was partly supported by the National Institutes of Health under grant number R01 HD069609 and the National Science Foundation under award number 1157698. The authors wish to thank the anonymous reviewers for their constructive and invaluable comments.

References

Allison, P.D. (2010). *Survival Analysis*. Retrieved from http://www.statisticalhorizons.com/wp-content/uploads/2012/01/Allison_SurvivalAnalysis.pdf

Bambra, C., Pope, D., Swami, V., Stanistreet, D., Roskam, A., Kunst, A., & Scott-Samuel, A. (2009). Gender, health inequalities and welfare state regimes: a cross-national study of 13 European countries. *Journal of Epidemiology & Community Health, 63,* 38044. http://dx.doi.org/10.1136/jech.2007.070292

Blackwell, D. L., Hayward, M., & Crimmins, E. (2001). Does childhood health affect chronic morbidity in later life? *Social Science & Medicine*, *52*(8), 1269–1284. http://dx.doi.org/10.1016/S0277-9536(00)00230-6

Beebe-Dimmer, J., Lynch, J.W., Turrell, G., Lustgarten, S., Raghunathan, T., & Kaplan, G.A. (2004). Childhood and adult socioeconomic conditions and 31-year mortality risk in women. *American Journal of Epidemiology*, *159*, 481–490.

Bird, C.E. & Rieker, P.P. (2008). *Gender and health: The effects of constrained choices and social policies*. New York: Cambridge University Press. http://dx.doi.org/10.1093/aje/kwh057

Borucka, J. (2013). Extensions of Cox Model for non-proportional hazards purpose. Retrieved from http://www.phusewiki.org/docs/Conference%202013%20SP%20Papers/SP07.pdf

Bowen, M.E. (2010). Coronary heart disease from a life-course approach: findings from the Health and Retirement Study, 1998-2004. *Journal of Aging and Health*, *22*, 219. http://dx.doi.org/10.1177/0898264309355981

Brown, T. H., O'Rand, A. M., & Adkins, D. E. (2012). Race-ethnicity and health trajectories: Tests of three hypotheses across multiple groups and health outcomes. *Journal of Health and Social Behavior*, 53(3), 359–377. http://dx.doi.org/10.1177/0022146512455333

Centers for Disease Control and Prevention. (2014). *Arthritis*. Retrieved from http://www.cdc.gov/arthritis/

Coltrane, S. (2000). Research on household labor: Modeling and measuring the social embeddedness of routine family work. *Journal of Marriage and the Family*, *62*, 1208-1233. http://dx.doi.org/10.1111/j.1741-3737.2000.01208.x

Corna, L. (2013). A life course perspective on socioeconomic inequalities in health: A critical review of conceptual frameworks. *Advances in Life Course Research*, *18*(2), 150-159. http://dx.doi.org/10.1016/j.alcr.2013.01.002

Crimmins, E.M., Kim, J.K., Sole-Auro, A. (2010). Gender differences in health: results from SHARE, ELSA and HRS. *European Journal of Public Health, 22*, 81-91.

Danese, A., Pariante, C. M., Caspi, A., Taylor, A., & Poulton, R. (2007). Childhood maltreatment predicts adult inflammation in a life-course study. *Proceedings of the National Academy of Sciences, 104*(4), 1319-1324. http://dx.doi.org/10.1073/pnas.0610362104

DeBoo, H.A., & Harding, J.E. (2006). The developmental origins of adult disease (Barker) hypothesis. *The Australian & New Zealand Journal of Obstetrics & Gynaecology, 46*, 4-14. http://dx.doi.org/10.1111/j.1479-828X.2006.00506.x

Denton, M., Prus, S., & Walters, V. (2004). Gender differences in health: A Canadian study of the psychosocial, structural and behavioral determinants of health. *Social Science & Medicine, 58(12)*, 2585–600. http://dx.doi.org/10.1016/j.socscimed.2003.09.008

deVries, B., & Watt, D. (1996). A lifetime of events: age and gender variations in thelife story. *International Journal of Aging and Human Development*, *42(2),* 81-102. http://dx.doi.org/10.2190/FM22-V5VT-B60Y-6UGC

Diprete, T. A., & Eirich, G. M. (2006). Cumulative advantage as a mechanism for inequality: A review of theoretical and empirical developments. *Annual Review of Sociology*, *32*, 271–297. http://dx.doi.org/10.1146/annurev.soc.32.061604.123127

Doumas, M., Papademetriou, V., Faselis, C., & Kokkinos, P. (2013). Gender differences in hypertension: Myths and reality. *Current Hypertension Reports*, *15*, 321-330. http://dx.doi.org/10.1007/s11906-013-0359-y

Drakopoulos, S.A., Lakioti, E., Thedossiou, I. (2011). Childhood socioeconomic deprivation and later adult health. *International Journal of Social Economics*, *28*, 23-38. http://dx.doi.org/10.1108/03068291111091945

Dubay, L.C. & Lebrun, L.A. (2012). Health, behavior, and health care disparities: Disentangling the effects of income and race in the United States. *International Journal of Health Services*, *42*(4), 607-625. http://dx.doi.org/10.2190/HS.42.4.c

Dupre, M. E. (2007). Educational differences in age-related patterns of disease: Reconsidering the cumulative disadvantage and age-as-leveler. *Journal of Health and Social Behavior*, *48*(1), 1–15. 241. **doi**: http://dx.doi.org/10.1177/002214650704800101

Elder, G.D, Johnson, M.K., & Crosnoe, R. (2003). The emergence and development of life course theory. In J.T Mortimer & M.J. Shanahan (Eds.), *Handbook of the Life Course* (pp.3-19). New York: Kluwer Academic/Plenum Publishers. http://dx.doi.org/10.1007/978-0-306-48247-2_1

Evans-Campbell, T., Lincoln, K.D., & Takeuchi, D.T. (2010). Race and mental health: Past debates, new opportunities. In W.R. Avison, J.D. McLeod, & B.A. Pescosolido (Eds). *Mental Health, Social Mirror*, (pp.169-190). New York: Springer.

Fitzgerald, J. (2011). Attrition in models of intergenerational links in health and economic status in the PSID. *The B.E. Journal of Economic Analysis & Policy*, *11*(3), 1–61. http://dx.doi.org/10.2202/1935-1682.2868

Fitzgerald, J., Gottschalk, P., & Moffitt, R. (1998). An analysis of sample attrition in panel data: The Michigan Panel Study of Income Dynamics. *The Journal of Human Resources*, *33*(2), 251–299. http://dx.doi.org/10.2307/146433

Fothergill, K. E., Ensminger, M. E., Green, K. M., Robertson, J. A., & Juon, H. S. (2009). Pathways to adult marijuana and cocaine use: A prospective study of African Americans. *Journal of Health & Social Behavior*, *50*(1), 65-81. http://dx.doi.org/10.1177/002214650905000105

Fuller, S., & Vosko, L.F. (2008). Temporary employment and social inequality in Canada: Exploring intersections of gender, race, and migration. *Social Indicators Research, 88*(1), 31-50. http://dx.doi.org/10.1007/s11205-007-9201-8

Galobardes, B., Lynch, J. W., & Davey Smith, G. (2004). Childhood socioeconomic circumstances and cause-specific mortality in adulthood: Systematic review and interpretation. *Epidemiologic Reviews*, *26*, 7–21. http://dx.doi.org/10.1093/epirev/mxh008

George, L.K. (2005). Socioeconomic status and health across the life course: Progress and prospects. *Journal of Gerontology, 60B,* 135–39. http://dx.doi.org/10.1093/geronb/60.Special_Issue_2.S135

Gilman, S. E., Kawachi, I., Fitzmaurice, G. M., & Buka, S. L. (2002). Socioeconomic status in childhood and the lifetime risk of major depression. *International Journal of Epidemiology*, *31*(2), 359–367. http://dx.doi.org/10.1093/ije/31.2.359

Gluckman P., & Hanson M. (2005). *The fetal matrix: Evolution, development and disease*. Cambridge, UK: Cambridge University Press.

Gustafsson, P. E., & Hammarström, A. (2012). Socioeconomic disadvantage in adolescent women and metabolic syndrome in mid-adulthood: An examination of pathways of embodiment in the Northern Swedish Cohort. *Social Science & Medicine*, *74*(10), 1630–8. http://dx.doi.org/10.1016/j.socscimed.2012.01.044

Hamil-Luker, J., & O'Rand, A. M. (2007). Gender differences in the link between childhood socioeconomic conditions and heart attack in adulthood. *Demography*, *44*(1), 137–158. http://dx.doi.org/10.1353/dem.2007.0004

Halliday, T. J., Kimmitt, M. C., & Kimmitt, C. (2012). Selective migration and health in the USA. *Population Studies*, *62*(3), 321–334. http://dx.doi.org/10.1080/00324720802339806

Hallqvist, J., Lynch, J., Bartley, M., Lang, T., & Blane, D. (2004). Can we disentangle life course processes of accumulation, critical period and social mobility? An analysis of disadvantaged socio-economic positions and myocardial infarction in the Stockholm Heart Epidemiology Program. *Social Science and Medicine*, *58*(8), 1555-1562. http://dx.doi.org/10.1016/S0277-9536(03)00344-7

Hannan, C. (2009). Women, gender equality, and diabetes. *International Journal of Gynecology and Obstetrics*, *104*, S4-S7. http://dx.doi.org/10.1016/j.ijgo.2008.11.021

Hayward, M.D, & Gorman, B.K. 2004. The long arm of childhood: The influences of early life social conditions on men's mortality. *Demography, 41*(1), 87–107. http://dx.doi.org/10.1353/dem.2004.0005

Heron, M.P. (2007). Deaths: leading causes for 2004. *National Vital Statistics Report*, *56(5)*,1-95.

Hogan, R., & Perrucci, C.C. (2007). Black women: Truly disadvantaged in the transition from employment to retirement income. *Social Science Research*, *36*, 1184–1199. http://dx.doi.org/10.1016/j.ssresearch.2006.07.002

Hunt, K. & Annandale, E. (1999). Relocating gender and morbidity: Examining men's and women's health in contemporary Western societies. *Social Science & Medicine, 48(1),*1–5. http://dx.doi.org/10.1016/S0277-9536(98)00284-6

Johnson, R. C., & Schoeni, R. F. (2011). Early-life origins of adult disease: national longitudinal population-based study of the United States. *American Journal of Public Health*, *101*(12), 2317–24. http://dx.doi.org/10.2105/AJPH.2011.300252

Kagotho, J. (2009) Examining the longitudinal impact of assets and income on immigrant health behaviors. *Washington University in St. Louis.*

Kivimaki, M., Lawlor, D.A., Davey Smith, G., Keltikangas-Jarvinen, L., Elovainio, M., Vahtera, J., Pulkki-Raback, L., Taittonen, L., Viikari, J.S.A. & Raitakari, O.T. (2006). Early socioeconomic position and blood pressure in childhood and adulthood: The cardiovascular risk in young Finns study. *Hypertension*, *47*, 39-44. http://dx.doi.org/10.1161/01.HYP.0000196682.43723.8a

Lemelin, E.T., Diez Roux, A.V, Franklin, T.G., Carnethon, M., Lutsey, P.L., Ni, H. & Shrager, S. (2009). Life-course socioeconomic positions and subclinical atherosclerosis in the multi-ethnic study of atherosclerosis. *Social Science & Medicine*, *68*(3), 444–51. http://dx.doi.org/10.1016/j.socscimed.2008.10.038

Lidfeldt, J., Li, T.Y., Hu, F.B., Manson, J.E., & Kawachi, I. (2007). A prospective study of childhood and adult socioeconomic status and incidence of Type 2 Diabetes in women. *American Journal of Epidemiology*, *165*(8), 882-889. doi: 10.1093/aje/kwk078

Lipowicz, A., Kozieł, S., Hulanicka, B., & Kowalisko, A. (2007). Socioeconomic status during childhood and health status in adulthood: the Wrocław growth study. *Journal of Biosocial Science*, *39*(4), 481–91. http://dx.doi.org/10.1017/S0021932006001799

Luo, Y. & Waite, L.J. (2005). The impact of childhood and adulthood SES on physical, mental, and cognitive well-being in later life. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, *60*(2),S93-S101. doi: 10.1093/geronb/60.2.S93.

Lundberg, U. (1996). The influence of paid and unpaid work on psychophysiological stress responses of men and women. *Journal of Occupational Health Psychology*, *1*, 117-130. http://dx.doi.org/10.1037/1076-8998.1.2.117

Lundbergy, U. & Parr, D. (2000). Neurohormonal factors, stress, health, and gender. In R.M. Eisler and M. Hersen (Eds.), *Handbook of Gender, Culture, and Health* (pp. 21-42). Mahwah, NJ: Lawrence Erlbaum Associates.

Lynch, S. M. (2003). Cohort and life-course patterns in the relationship between education and health: A hierarchical approach. *Demography*, *40*(2), 309–331. http://dx.doi.org/10.1353/dem.2003.0016

Lynch, S.M. (Ed). (2008). Special Issue: Race, Socioeconomic Status, and Health in Life-Course Perspective. *Research on Aging, 30*(2).http://dx.doi.org/10.1177/0164027507312086

Maty, S.C., Lynch, J.W., Raghunathan, T..E., & Kaplan, G.A. (2008). Childhood socioeconomic position, gender, adult body mass index, and incidence of Type 2 diabetes mellitus over 34 years in the Alameda County Study. *American Journal of Public Health*, *98*, 1486-1494. http://dx.doi.org/10.2105/AJPH.2007.123653

McDonough, P., & Berglund, P. (2003). Histories of poverty and selfrated health trajectories. *Journal of Health and Social Behavior*, *44*(2), 198–214. http://dx.doi.org/10.2307/1519808

McDonough, P., Sacker, A., & Wiggins, R. D. (2005). Time on my side? Life course trajectories of poverty and health. *Social Science & Medicine*, *61*(8), 1795–1808. http://dx.doi.org/10.1016/j.socscimed.2005.03.021

McGonagle, K.A., Schoeni, R.F., Sastry, N., & Freedman, V.A. (2013). The Panel Study of Income Dynamics: Overview, Recent Innovations, and Potential for Life Course Research. *Longitudinal and Life Course Studies*, *3*(2), 2-21.

Mckenzie, S.K., Carter, K.N., Blakely, T. & Ivoery, V. (2011). Effects of childhood socioeconomic position on subjective health and health behaviours in adulthood: how much is mediated by adult socioeconomic position? *BMC Public Health, 11*, 269. http://dx.doi.org/10.1186/1471-2458-11-269

Meer, J., Miller, D. L., & Rosen, H. S. (2003). Exploring the health-wealth nexus. *Journal of Health Economics*, *22*(5), 713–730. http://dx.doi.org/10.1016/S0167-6296(03)00059-6

Mensah, F.K. & Hobcraft, J. (2008). Childhood deprivation, health and development: associations with adult health in the 1958 and 1970 British prospective birth cohort studies. *Journal of Epidemiology & Community Health*, *62*, 599-606. http://dx.doi.org/10.1136/jech.2007.065706

Miech, R.A., & Shanahan, M.J. (2000). Socioeconomic status and depression over the life course. *Journal of Health and Social Behavior, 41*(2), 162-176. http://dx.doi.org/10.2307/2676303

Miller, G.E., Chen, E., & Parker, K.J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin*, *137*, 959-997. http://dx.doi.org/10.1037/a0024768

Moen, P., Robison, J., & Fields, V. (1994). Women's work and caregiving roles: A life course approach. *Journal of Gerontology*, *49*(4), S176–S186. http://dx.doi.org/10.1093/geronj/49.4.S176

Montez, J.K. & Hayward, M.D. (2014). Cumulative childhood adversity, educational attainment, and active life expectancy among U.S. adults. *Demography*, *51*, 413-435. http://dx.doi.org/10.1007/s13524-013-0261-x

Murray E.T., Mishra, G.D., Kuh, D., Guralnik, J., Black, S. & Hardy, R. (2011). Life course models of socioeconomic position and cardiovascular risk factors: 1946 birth cohort. *Annals of Epidemiology*, *21*, 589-597. http://dx.doi.org/10.1016/j.annepidem.2011.04.005

National Poverty Center. (2013). *Poverty in the United States*. Ann Arbour, MI: The University of Michigan.

National Center for Health Statistics. (2009). *Health, United States, 2008, with chartbook*. Hyatsville, MD: Author.

O'Rand, A. M. (1996). The precious and the precocious: understanding cumulative disadvantage and cumulative advantage over the life course. *The Gerontologist*, *36*(2), 230–8. http://dx.doi.org/10.1093/geront/36.2.230

Panel Study of Income Dynamics. (2013). Public use dataset. Produced and distributed by the Survey Research Center, Institute for Social Research, University of Michigan, Ann Arbor, MI.

Petrea, R.E., Beiser, A.S., Seshadri, S., Kelly-Hayes, M., Kase, C.S., & Wolf, P.A. (2009). Gender differences in stroke incidence and poststroke disability in the Framingham Heart Study. *Stroke*, *40*, 1032-1037. http://dx.doi.org/10.1161/STROKEAHA.108.542894

Phillips, J.A. & Sweeney, M.M. (2005). Premarital cohabitation and marital disruption among white, black, and Mexican American women. *Journal of Marriage and Family*, *67*(2), 296-314. doi: 10.1111/j.0022-2445.2005.00117.x

Prokos, A.H., Padavic, I., & Schmidt, A. (2009). Nonstandard work arrangements among women and men scientists and engineers. *Sex Roles, 61,* 653-665. http://dx.doi.org/10.1007/s11199-009-9680-y

Pudrovska, T., & Anishkin, A. (2013). Early-life socioeconomic status and physical activity in later life: evidence from structural equation models. *Journal of Aging and Health*, *25*(3), 383–404. http://dx.doi.org/10.1177/0898264312468601

Pudrovksa, T. & Anikputa, B. (2013). Early-life socioeconomic status and mortality in later life: An integration of four life-course mechanisms. *Journals of Gerontology. Series B: Psychological Sciences and Social Sciences*, *69*(3), 451-460.

Read, J.G. & Gorman, B.K. (2010). Gender and health inequality. *Annual Review of Sociology*, *36*, 371-386. http://dx.doi.org/10.1146/annurev.soc.012809.102535

Schafer, Markus H, & Ferraro, K. F. (2012). Childhood misfortune as a threat to successful aging: avoiding disease. *The Gerontologist*, *52*(1), 111–20. http://dx.doi.org/10.1093/geront/gnr071

Sen, G. & Ostlin, P. (2007). *Gender inequity in health: Why it exists and how we can change it*. World Health Organization.

Shuey, K. M., & Willson, A. E. (2008). Cumulative disadvantage and blackwhite disparities in life-course health trajectories. *Research on Aging*, *30*(2), 200–225. http://dx.doi.org/10.1177/0164027507311151

Shuey, K.M. & Willson, A.E. (2014). Economic hardship in childhood and adult health trajectories: An alternative approach to investigating life-course processes. *Advances in Life Course Research*, *22*, 49-61. http://dx.doi.org/10.1016/j.alcr.2014.05.001

Taylor,S.E., Klein, L.C., Lewis, B.P., Gruenewald, T.L., Gurung, R.A.R. & Updegraff, J.A. (2000). Biobehavioral responses to stress in females: Tend-and-befriend, not fight-or-flight. *Psychological Review, 107*, 411–429. http://dx.doi.org/10.1037/0033-295X.107.3.411

Turner, R.J. & Avison, W.R. (2003). Status variations in stress exposure: Implications for the interpretation of research on race, socioeconomic status, and gender. *Journal of Health and Social Behavior, 44*, 488-505. http://dx.doi.org/10.2307/1519795

Turner, R.J., Wheaton, B., & Lloyd, D.A. (1995). The epidemiology of social stress. *American Sociological Review*, *60*, 104-125. http://dx.doi.org/10.2307/2096348

Wagmiller, R. L., Lennon, M. C., Kuang, L., Alberti, P. M., & Aber, J. L. (2006). The dynamics of economic disadvantage and children's life chances. *American Sociological Review*, *71*(5), 847–866. http://dx.doi.org/10.1177/000312240607100507

Walsemann, K. M., Ailshire, J. a, Bell, B. a, & Frongillo, E. A. (2012). Body mass index trajectories from adolescence to midlife: differential effects of parental and respondent education by race/ethnicity and gender. *Ethnicity & Health*, *17*(4), 337–62. http://dx.doi.org/10.1080/13557858.2011.635374

Western, B., Bloome, D., Sosnaud, B., & Tach, L. (2012). Economic insecurity and social stratification. *Annual Review of Sociology*, *38*, 341–359. http://dx.doi.org/10.1146/annurev-soc-071811-145434

Willson, A. E., Shuey, K. M., & Elder, G. H., Jr. (2007). Cumulative advantage processes as mechanisms of inequality in life course health. *American Journal of Sociology*, *112*(6), 1886–1924. http://dx.doi.org/10.1086/512712

Zunzunegui, M.-V., Alvarado, B.-E., Béland, F., & Vissandjee, B. (2008). Explaining health differences between men and women in later life: a cross-city comparison in Latin America and the Caribbean. *Social Science & Medicine*, *68*(2), 235–42. http://dx.doi.org/10.1016/j.socscimed.2008.10.031

Endnotes

[i] Consistent with previous literature using the PSID, 125% of the US poverty threshold was used because the PSID consistently finds higher reported incomes than the Census Bureau (Wagmiller et al., 2006).

Temporal trends in energy and macronutrient distribution in meals eaten by children from the Avon Longitudinal Study of Parents and Children

Suzana Almoosawi Newcastle University, UK
suzana.almoosawi@newcastle.ac.uk
Victoria Cribb Bristol University, UK
Pauline Emmett Bristol University, UK
Alison Lennox University of Surrey, UK

(Received February 2014 Revised September 2015) http://dx.doi.org/10.14301/llcs.v7i1.292

Abstract

Cross-sectional studies have reported associations between frequency of eating, snacking, breakfast skipping, night-eating and obesity. However, there have been few investigations of longitudinal trends in time-of-day of energy and macronutrient intake. We investigated trends in time-of-day of energy and macronutrient intake in the Avon Longitudinal Study of Parents and Children. Diet was assessed using 3d estimated diaries at ages 10 and 13 years. Diaries were divided into seven time slots: breakfast, mid-morning, lunch, mid-afternoon, dinner, late evening and extras. Trends in energy and macronutrient intake at different eating occasions between ages 10 and 13 years were assessed using linear mixed models after adjustment for age, maternal employment and child's BMI. Lunch and dinner were found to contribute the greatest proportion of energy and macronutrient intake in both girls and boys at both ages. However, there was a shift in time-of-day of energy and macronutrient intake for both sexes, with greater proportions of intake between meals (mid-morning, late evening, extras) and lower proportion of intake at main meals (breakfast, dinner) at age 13 years compared to 10 years. Factors contributing to changes in energy and nutrient distribution warrant investigation and the implications of such changes in time-of-day of energy and nutrient intake on long-term health remains to be examined.

Keywords

Circadian rhythm, eating profile, eating pattern, time-of-day, energy, macronutrients, ALSPAC, prospective study

Introduction

It is increasingly recognised that the time, nutrient composition (Bray et al. 2010; Cho, Dietrich, Brown, Clark & Block, 2003; Deshmukh-Taskar, Nicklas, Radcliffe, O'Neil & Liu, 2012; Deshmukh-Taskar et al., 2010), frequency (Farshchi, Taylor & Macdonald, 2005a, 2005b) and regularity of meals (Farshchi et al. 2005a; Sierra-Johnson et al., 2008) can influence multiple metabolic parameters and consequently health (Almoosawi, Prynne, Hardy & Stephen, 2012; Almoosawi, Prynne, Hardy & Stephen, 2013a, 2013b). In both children and adults, skipping breakfast is associated with poorer micronutrient intake, poorer growth

and increased weight and body fatness (Deshmukh-Taskar, Nicklas, Radcliffe, O'Neil & Liu, 2012; Deshmukh-Taskar et al., 2010; Hallstrom et al., 2012; Rampersaud, Pereira, Girard, Adams & Metzl, 2005), while regular breakfast consumption is associated with lower body fatness and improved lipid, blood pressure and glucose profiles especially in boys (Hallstrom et al., 2012). The composition of breakfast has also been shown to influence total daily energy intake and weight (Cho, et al., 2003; Deshmukh-Taskar, Nicklas, Radcliffe, O'Neil & Liu, 2012) and cognitive performance at school (Mahoney, Taylor, Kanarek & Samuel, 2005), while eating after dinner has been related to multiple cardiometabolic risk factors and obesity (Almoosawi et al., 2013b; Sato et al., 2011). Thus, examining recent trends in energy and macronutrient distribution is important in contributing to our understanding of the role of time-of-day of eating on recent trends in obesity and other disorders.

The transition between childhood and adolescence marks a key stage in human development, and is characterised by multiple physiological, psychological and behavioural changes. Increased independence, concerns of body image and social activities outside the home can influence decisions regarding what and when to eat. In girls, the quality of the diet appears to decline (Mannino, Lee, Mitchell, Smiciklas-Wright & Birch, 2004) and the prevalence of breakfast skipping increases to a greater extent than in boys (Keski-Rahkonen, Kaprio, Rissanen, Virkkunen, & Rose 2003) with the move into teenage years. Dietary habits are known to track from adolescence to adulthood (Mannino et al., 2004; te Velde, Twisk & Brug, 2007). Consequently, poor eating patterns in adolescence can carry implications for health and disease development in adulthood. Despite this evidence, few studies have examined trends in energy and nutrient distribution through the day in children through their transition to adolescence.

The present paper aims to describe energy and macronutrient distribution across different eating occasions in the Avon Longitudinal Study of Parents and Children (ALSPAC). It will present data on the proportion of consumers and non-consumers within given eating occasions, and examine changes in time-of-day of energy and macronutrient intake across eating occasions between the ages of 10 and 13 years.

Methods

The Avon Longitudinal Study of Parents and Children (ALSPAC) is an on-going UK longitudinal cohort study which is designed to investigate the health and development of children. The study recruited 14,541 pregnant women resident in the former Avon Health Authority in South West England with expected delivery date between April 1991 and December 1992. This resulted in 13,988 children alive at 12-months, with an additional 548 new participants being recruited at age seven years giving a total of 14,536 children. Ethical approval for the study was obtained from the ALSPAC Law and Ethics Committee and the Local Research Ethic Committees. Details of the study have been published previously (Boyd et al., 2013). Please note that details of all ALSPAC data is available through a fully searchable data dictionary at http://www.bris.ac.uk/alspac/researchers/data-access/data-dictionary/

Dietary assessment

Detailed dietary assessments were carried out in the period between February 2002 and October 2003 (mean age 10.6 years), and January 2005 and September 2006 (mean age 13.8 years). Details of the dietary assessment method have been described elsewhere (Emmett, 2009). Briefly, prior to a clinic visit, children were asked, with the help of their carer to record all food and drinks consumed in a structured three-day diet diary (two weekdays and one weekend day) that was subdivided into eight eating occasions: pre-breakfast, breakfast, mid-morning, lunch, mid-afternoon, dinner, late evening and extras. Children were asked to describe portions using household measures. If the description was inadequate a portion size was allocated based on data on average portion sizes consumed by children of the same age derived from weighed dietary intakes (Wrieden et al., 2008) from a national survey of British children (Gregory, Collins, Davies, Hughes & Clarke, 1995; Gregory & Lowe, 2000). Further portion size information was obtained from manufacturers. Overall, 3,320 children completed three-day diet diaries (1,561 boys and 1,759 girls) at both ages 10 and 13 years, representing 23% of entire cohort, 44% of those with any dietary data collected at 10 years and 54% at 13 years. Children who completed dietary assessment at both time-points were more likely to have mothers with higher

education levels compared to those who did not complete assessment at both time-points. Diet records were coded using DIDO (Diet In, Data Out) developed by the Dunn Nutrition Unit in Cambridge (Emmett, 2009), and nutrient analysis used the 5th edition of McCancee and Widdowson's "The Composition of Foods" (Holland et al., 1991) and supplements (Chan, Brown & Buss, 1994; Chane, Brown, Lee & Buss, 1995; Holland, Unwin & Buss, 1991; Holland, Unwin & Buss, 1988; Holland, Unwin & Buss, 1989; Holland, Welsh & Buss, 1992a, 1992b; Holland, Brown & Buss, 1993).

Maternal employment, child anthropometry and mis-reporting

The above variables have been selected as potential confounders. Maternal employment has been previously shown to influence children's meal regularity and meal skipping (Gaina, Sekine, Chandola, Marmot & Kagamimori, 2009). Similarly, overweight and obese children appear to differ in their eating patterns and time-of-energy intake through the day compared to lean children (Bellisle, Rolland-Cachera, Deheeger & Guilloud-Bataille, 1988). Finally, there is evidence to suggest that mis-reporting can be selective with between-meal snacks more frequently under-reported (Poppitt, Swann, Black & Prentice, 1998). Maternal employment status was derived from a questionnaire completed when the child was aged 122-months (10.2 years). The question asked 'how many hours were worked in the last week?'. In this analysis maternal employment was grouped as minimal working (less than seven hours), part-time (seven-28 hours) and full-time (more than 28 hours). Mis-reporting was estimated using an individualised method as the ratio of reported energy intake to estimated energy requirements (Rennie, Coward & Jebb, 2007). Children with an energy intake to estimated energy requirement ratio between 0.79–1.21 were deemed as normal reporters (Johnson, Mander, Jones, Emmett & Jebb, 2008). Overall of the 3,321 children, 1,079 and 2,000 were found to under-report energy intake at age 10 and 13 years, respectively.

At the research clinic visit at ages 10 and 13 years, trained staff collected data on height, weight, waist circumference, and body composition. Height was measured to the last complete millimeter using the Harpenden Stadiometer and weight measured to the nearest 50g using the Tanita Body Fat Analyser (Model TBF 305). BMI was calculated as weight in kilograms divided by the square of height in meters. Underweight, overweight and obesity were defined using age and sex specific cut-off points from the International Obesity Task Force (Cole, Bellizzi, Flegal & Dietz, 2000; Cole, Flegal, Nicholls & Jackson, 2007).

Statistical analyses

Changes in proportion of consumers and non-consumers were assessed using McNemar's chi-square statistics. Multilevel models were used to investigate changes in the trajectory of energy or macronutrient intake at different eating occasions in boys and girls between ages 10 and 13years after adjustment for age, child's BMI and maternal employment. A random intercept and slope model was selected to allow each child to have their own intercept and slope. Age was centred at the mean in order to facilitate interpretation of the model intercept, without affecting the interpretation of the slope. Likelihood ratio tests were used to compare the multilevel models to a null single-level model. The advantage of using a random-effects model is that such models can separate between-subject variations from longitudinal changes and allow for missing data. All models were fitted separately for boys and girls. Additional analyses assessing differences in macronutrient intake between main meals (breakfast, lunch and dinner) and between-meal eating occasions (mid-morning, mid-afternoon, late evening and extras) were conducted including type of eating occasion as a binary predictor variable in the multilevel models. Sensitivity analysis including children with only accurate energy reporting were conducted. All statistical analyses were performed using STATA 13 and IBM SPSS version 21.

Results

Trends in energy intake

The distribution of energy and macronutrients by eating occasion in boys and girls at ages 10 and 13 years is shown in Table 1 for the entire sample, including non-consumers of particular eating occasions, and in table 2 for the sample excluding non-consumers. The percentage of non-consumers was higher at age 13 years at breakfast, mid-afternoon and dinner than at 10 years in both boys and girls, while the percentage of non-consumers

declined at mid-morning and for extras (figure 1). In girls, the percentage of non-consumers decreased in the late evening eating occasion between ages 10 and 13 years.

After adjustment for BMI and maternal employment, mean proportion of energy intake at age 10 years at breakfast was 16.5% for boys and 16.1% for girls (table 3). This proportion declined by 1.9% and 2.6% between ages 10 and 13 years for boys and girls, respectively. At child level, mean proportion of energy intake at breakfast varied with a standard deviation (SD) of 3.69 and 3.66, while the rate of change varied with a SD of 2.82 and 0.13 for both boys and girls, respectively. These random effects remained highly correlated, so children who obtained a high proportion of energy intake from breakfast at age 10 years tended to have lower rate of decline over time.

Lunch and dinner continued to contribute the greatest proportion of total daily energy intake at both ages 10 and 13 years. Mean proportion of energy intake at age 10 years at lunch was 28.2% for boys and 30.5% for girls. This declined at a rate of 2.2% for boys and 2.8% for girls, respectively between ages 10 and 13 years. At child level, mean proportion of energy intake at lunch varied with a SD of 3.35 for boys and 2.47 for girls, respectively, while the rate of change varied with a SD of 0.29 for boys and 3.9% for girls, respectively. These random effects remained highly correlated, so children who obtained a high proportion of energy intake from lunch at age 10 years tended to have a lower rate of decline over time. Mean proportion of energy intake at dinner at age 10 years was 28.7% for boys and 27.6% for girls. This declined at a rate of 2.9% and 2.0% between ages 10 and 13 years for boys and girls, respectively. At child level, mean proportion of energy intake at dinner varied with a SD of 4.48 and 4.03, while the rate of change varied with a SD of 3.38 and 2.24 for both boy and girls, respectively. In relation to boys, the random effects did not correlate after adjustment for individual level covariates, suggesting that a boy's BMI or maternal employment accounted for most of the variation. In relation to girls, the random effects remained highly correlated, so girls who obtained a high proportion of energy intake from dinner at age 10 years tended to have lower rate of decline over time. Mean proportion of energy intake at age 10 years at dinner was 0.13% and 0.10% higher for boy and girls, respectively, for every one unit increase in BMI.

In relation to between-meal eating occasions, mean proportion of energy intake at mid-morning increased at a rate of 3.1% for boys and 6.3% for girls, respectively, between ages 10 and 13 years. These random effects remained highly correlated, so children who obtained a high proportion of energy intake from mid-morning at age 10 years tended to have lower rate of decline over time. In relation to boys, mean proportion of energy intake at age 10 years at mid-morning was 0.09% lower for every one unit increase in BMI.

Mean proportion of energy intake at age 10 years at mid-afternoon was 10.5% for boys and 9.5% for girls. This intake declined at a rate of 1.6% for boys and 2.0% for girls, respectively, between ages 10 and 13 years. At child level, mean proportion of energy intake at mid-afternoon varied with a SD of 2.50 for boys and 1.90 for girls, respectively, while the rate of change varied with a SD of 3.31 and 0.97, respectively. These random effects remained highly correlated, so children who obtained a high proportion of energy intake from mid-afternoon at age 10 years tended to have higher rate of decline over time.

Between ages 10 and 13 years, mean proportion of energy intake at late-evening increased at a rate of 3.2% for boys and 3.3% for girls, respectively. These random effects remained highly correlated, so children who obtained a high proportion of energy intake from late-evening at age 10 years tended to have lower rate of decline over time.

Mean proportion of energy intake increased at the extra eating occasion at a rate of 2.4% and 2.9% between ages 10 and 13 years. Children who obtained a high proportion of energy intake from extras at age 10 years tended to have higher rate of decline over time.

Trends in macronutrient intake

In general, the distribution of macronutrients followed a similar pattern to that seen for energy (tables 1-2). Data from the linear mixed models are presented as supplementary material. However, the macronutrient composition of eating occasions differed significantly. In boys and girls, the proportion of energy intake from protein increased across all eating occasions between ages 10 and 13 years, with the exception of breakfast and extra eating occasions in girls (figure 2). By contrast, the proportion of energy intake from fat declined at

lunch in boys and at breakfast, lunch and dinner in girls, respectively. Similarly, the proportion of energy intake from carbohydrate declined at mid-morning and late evening in boys, and at mid-morning and late evening in girls, respectively. There was an increase in the proportion of energy from carbohydrate at breakfast in girls, and the proportion of energy from fat at mid-morning. Similarly, in boys, there was an increase in proportion of energy intake from fat at mid-morning and an increase in proportion of energy intake from carbohydrate at lunch.

The type of eating occasion (main meal vs. snack) was a significant predictor of the macronutrient composition, such that the average composition of between-meal eating occasions was higher in carbohydrate and lower in protein and fat than the average composition of main meals. These findings were similar across both years and in both boys and girls (see supplementary material).

Sensitivity analysis

Sensitivity analyses were conducted including only individuals with accurate reporting of energy intake (boys n= 1583, girls n=1778). Excluding under-reporters and over-reporters did not affect the rate of change (slope) in proportion of energy intake at different eating occasions, with the exception of dinner in girls wherein the rate of decline in proportion of energy intake was greater compared to previous models. In relation to the intercept, mean proportion of energy intake at age 10 was found to be higher at dinner and extras and lower at late evening for both boys and girls compared to previous models. At individual level, the random effects were also affected such that there were differences in the SD of the slope. These results are presented as supplementary material.

Discussion

To our knowledge this is the first study to describe changes in the distribution of energy and macronutrient composition of eating occasions in a cohort of boys and girls growing up in the United Kingdom during their transition from childhood to early adolescence. Lunch and dinner constituted the most important eating occasions, with the greatest contributions to energy intake. Breakfast contributed approximately 15% of total daily energy intake while lunch and dinner combined contributed around 60%. Overall, the contribution of lunch and dinner to energy intake seen in this study is consistent with previous cohort studies in the United States, the Netherlands and France (deCastro, Bellisle, Feunekes, Dalx & DeGraaf, 1997).

One of the main findings of the current study is the observed shift in time-of-day of energy and macronutrient intake for both sexes, with greater proportions of intake between meals (mid-morning, late evening, extras) and lower intakes at main meals (breakfast, dinner) at age 13 years compared to 10 years. The contribution of eating occasions between the main meals was almost double the contribution of breakfast to energy intake and increased between 10 and 13 years by about 7% of energy. Consistent with these findings, we observed a rise in the proportion of consumers in both the mid-morning period in both boys and girls, and late evening period in girls. These changes in time-of-day of energy intake are likely to be a reflection of age-related changes in eating behavior, although it is not possible to distinguish them from time trends given the design of this study. For instance, the findings are in agreement with the general trends in Nordic countries characterised by increased meal skipping and reduced regularity of main meals (Samuelson, 2000). Similarly, increases in snacking behavior between 1989–91 and 1994–98 and between 1994–98 and 2003–06 have been documented cross-sectionally in US children, with snacks contributing to 27% of children's total daily energy intake (Piernas & Popkin, 2010b). This rise in the contribution of snacking to total daily energy intake has also been observed in US adults, where snacks contribute 24% of total daily energy intake in 2006 compared to 18% in 1977 (Piernas & Popkin, 2010a). In adults, this rise was due to an increase in the number of people reporting consuming snacks from 71% to 97% (Piernas & Popkin, 2010a).

Regardless as to whether our findings are due to age or time trends, the above results are concerning since snacking typically occurs outside the home and family meals. Adolescents who snack frequently on the run or during leisure time are more likely to skip meals (Savige, Macfarlane, Ball, Worsley & Crawford, 2007). This can carry multiple health implications since regular family meals during early adolescence are known to be associated with the formation of healthy eating

habits in adulthood that include higher intakes of fruit and vegetables, less breakfast skipping especially in girls, more frequent dinner meals and greater priority to meal structure (Larson, Fulkerson, Story & Neumark-Sztainer, 2012).

In the present study, the average composition of between meals eating occasions was found to be higher in carbohydrate and lower in protein and fat than the average composition of main meals. This could be a point of concern if the main carbohydrate type was simple sugars, as regular between meal consumption of sugar-sweetened beverages has been shown to increase risk of overweight amongst preschool-aged children (Dubois, Farmer, Girard & Peterson, 2007). In ALSPAC, consumption of sugar-sweetened beverages has increased between ages five and seven years from 57g/d to 67g/d (Johnson et al., 2007). In National Diet and Nutrition Survey (NDNS) rolling programme, consumption of sugar-sweetened beverages was found to double from ages four-10 years to 11-18 years (Whitton et al., 2011). Together these findings may point towards between meal eating occasions as being important target points for future policies and dietary recommendation around healthy eating. However, further analyses are needed to examine type and sources of macronutrients consumed between meals in the ALSPAC cohort.

The decline in the proportion of daily energy intake from breakfast in both boys and girls and the rise in the proportion of daily energy intake from late evening meals in girls are also a matter of concern as breakfast skipping and eating later in the day are known risk factors for the development of chronic diseases and obesity. Overweight and obese children often report eating less at breakfast and more at dinner than their leaner counterparts (Bellisle et al., 1988). Eating breakfast is also frequently associated with a better diet quality, higher energy intake but lower body weight compared to breakfast skipping which is related to lower energy intake but higher BMI (Rampersaud et al., 2005). More recent evidence suggests that this unequivocal association between breakfast skipping, lower daily energy intake and higher BMI could be due to breakfast skipping being related to lower physical activity in adolescent girls (Corder et al., 2011). It is noteworthy that the prevalence of breakfast skipping in the current cohort is lower than that reported in US children, which averages 20% of children (Deshmukh-Taskar et al., 2010). Nonetheless, the increasing trend in breakfast skipping might constitute a risk for obesity, particularly when considering current evidence that demonstrates that an irregular pattern of meals produces lower postprandial energy expenditure compared with regular meal frequency (Farshchi, Taylor & Macdonald, 2004, 2005a). In relation to eating later in the day, recent evidence suggests that two-thirds of US children consume an evening snack (Nicklas, Baranowski, Cullen & Berenmson, 2001). Similarly, we have previously demonstrated a trend towards a shift in energy intake towards later in the day in adults over the course of two decades in the 1946 British Birth Cohort (Almoosawi, Winter, Prynne, Hardy & Stephen, 2012). Adults who reported having higher energy intake late in the evening had a greater rise in systolic and diastolic blood pressure between ages 43 and 53 years (Almoosawi et al., 2013b). Several explanations could be provided for the detrimental effects of eating late in the day. For instance, eating later in the day has a lower impact on satiety, resulting in higher intake of energy over the day (deCastro, 2007). Similarly insulin sensitivity declines progressively through the day. Thus, consumption of a standard meal in the evening produces greater postprandial glucose and insulin responses, an effect that is carried over to subsequent breakfast (Sato et al., 2011). Based on this evidence, it will be imperative to study the impact of skipping breakfast and eating later in the day on BMI trajectories in the ALSPAC cohort. Moreover, further research is required to identify children with an irregular meal pattern and study the relationship between such eating pattern and BMI.

In the current study, there was a reduction in the percentage of energy derived from fat at breakfast and lunch. The contribution of fat to total daily energy intake also declined between ages 10 and 13 years, in accordance with the current nutritional guidelines to reduce fat intake to 35% of total energy intake. Although differentiating between age- and time-effects is difficult in a birth cohort, it is likely that the observed changes in macronutrient composition reflect time-effects and changes in parents' or carer's food choices as a result of dietary recommendations and nutrition education campaigns. To emphasise, in the National Diet and Nutrition Survey (NDNS) rolling programme,

consumption of semi-skimmed milk has replaced consumption of whole milk in both children and adults (Whitton et al., 2011). There has also been a small reduction in the consumption of savoury snacks and chips in children, and an increase in consumption of reduced fat spreads (Whitton et al., 2011). In adults in National Survey of Health and Development (NSHD), the reduction in percentage of energy derived from fat has been attributed to a reduction in fat content of meat and meat products arising from improvements in butchery methods, animal feeds, and selective breeding (Prynne, Paul, Mishra, Greenberg & Wadsworth, 2005). Generally in the US and the UK, children's diets have improved as evident by the reduction in the consumption of fats/oils, breads/grains, red meat, chocolate confectionery, candy, and eggs, and increased quantities of fruit/fruit juices, beverages, and poultry (Nicklas et al., 2001; Whitton et al., 2011). However, it has been hypothesized that changes in specific eating patterns, such as increased frequency of meals eaten outside home, portion size, snacking and meal-skipping, may underlie the rise in obesity prevalence among children (Nicklas et al., 2001). This emphasises the need for research to investigate other aspects of eating behaviours such as meal frequency, composition, regularity and diurnal rhythms in energy and macronutrient distribution in relation to health.

The composition of breakfast and its relation to health has received growing attention in the past decades in relation to weight control (Deshmukh-Taskar et al., 2012; Deshmukh-Taskar et al., 2010) and cognitive function (Mahoney et al., 2005). In the Third National Health and Nutrition Examination Survey, Cho and colleagues reported that subjects who ate ready-to-eat cereals had significantly lower BMI compared to breakfast skippers and meat and egg eaters, while those who reported fruit or vegetable consumption had significantly lower daily energy intakes (Cho et al., 2003). Similar findings have been observed in other studies (Deshmukh-Taskar et al., 2012; Deshmukh-Taskar et al., 2010). Thus, the implications of the observed changes in energy and macronutrient distribution trends in ALSPAC, and of the observed sex-differences in trends, remain to be elucidated, particularly in relation to overweight and obesity. Circadian rhythms are central to the regulation of physiological and behavioural processes in humans, and disturbances in circadian rhythms have been reported to be associated with numerous chronic disease (Huang, Ramsey, Marcheva & Bass, 2011; Lowden, Moreno, Holmback Lennernas & Tucker, 2010; Scheer, Hilton, Mantzoros & Shea, 2009). As a consequence, further interventional and epidemiological research is required to understand the implications of our findings, and assess their association with long-term health outcomes and weight gain. Likewise, factors determining the time-of-day and nutrient composition of eating occasions and the impact of the context of eating need to be elucidated. In future, it will also be interesting to investigate the energy and nutrient density of eating occasion in attempt to examine whether consumption of meals higher in energy density later in the day combined with lower physical activity is associated with early biomarkers of cardiovascular disease and weight gain.

The strengths of the current analyses lies in the availability of dietary data over two time-points which permitted the examination of changes in energy and macronutrient distribution during the transition from childhood to adolescence in the same individuals, large sample size, and robustness of the dietary assessment method. Weaknesses include use of pre-defined eating occasions, which did no permit investigating changes in the frequency and number of eating occasions. The inclusion of an "extras" eating occasion also poses a strong limitation as children who failed to remember the eating occasion at which they consumed a certain food or drink may have opted to report the missed item within the extras eating occasion, thereby accounting for the change in the proportion of consumers between ages 10 and 13 years. We also did not investigate patterns of sleeping which may determine the time-of-day of eating. Furthermore, children in ALSPAC have been recruited from one prescribed area of the UK; hence the sample might not be representative of the entire population. Finally, the most significant limitation of the current study is the missing data as not all children provided data on diet, BMI or potential confounders. This may limit the generalisability of the current findings and suggests a need for further studies using other cohort samples.

In conclusion, breakfast and to a greater extent lunch and dinner contributed the greatest proportion of energy and macronutrient intake in

both girls and boys at both age 10 and 13 years. However, the contribution of breakfast and dinner to total daily energy intake declined between ages 10 and 13 years, with a switch towards greater energy consumption at mid-morning and late evening. Further studies are required to assess the implications of these findings. Further experimental studies are also required to examine the impact of manipulating time-of-day and nutrient composition of eating occasions on health.

Acknowledgements

We are extremely grateful to all the families who took part in this study, the midwives for their help in recruiting them and the whole ALSPAC team, which includes interviewers, computer and laboratory technicians, clerical workers, research scientists, volunteers, managers, receptionists and nurses. The UK Medical Research Council and the Wellcome Trust (grant ref 102215/2/13/2) and the University of Bristol provide core support for ALSPAC. This analysis was funded by the UK National Prevention Research Initiative (strategic grant G0701939).

References

Almoosawi, S., Prynne, C. J., Hardy, R., & Stephen, A. M. (2012). Time-of-day and nutrient composition of eating occasions: prospective association with the metabolic syndrome in the 1946 British birth cohort. *International Journal of Obesity (Lond),* 37(5), 725-31. http://dx.doi.org/10.1038/ijo.2012.103

Almoosawi, S., Prynne, C. J., Hardy, R., & Stephen, A. M. (2013a). Diurnal eating rhythms: Association with long-term development of diabetes in the 1946 British birth cohort. *Nutrition Metabolism and Cardiovascular Diseases, In Press*. http://dx.doi.org/10.1016/j.numecd.2013.01.003

Almoosawi, S., Prynne, C. J., Hardy, R., & Stephen, A. M. (2013b). Time-of-day of energy intake: association with hypertension and blood pressure 10 years later in the 1946 British Birth Cohort. *Journal of Hypertension,* 31(5), 882-92. http://dx.doi.org/10.1097/HJH.0b013e32835ecc06

Almoosawi, S., Winter, J., Prynne, C. J., Hardy, R., & Stephen, A. M. (2012). Daily profiles of energy and nutrient intakes: are eating profiles changing over time? *European Journal of Clinical Nutrition, 66*(6), 678-686. http://dx.doi.org/10.1038/ejcn.2011.210

Bellisle, F., Rolland-Cachera, M. F., Deheeger, M., & Guilloud-Bataille, M. (1988). Obesity and food intake in children: evidence for a role of metabolic and/or behavioral daily rhythms. *Appetite, 11*(2), 111-118. http://dx.doi.org/10.1016/S0195-6663(88)80010-2

Bray, M. S., Tsai, J. Y., Villegas-Montoya, C., Boland, B. B., Blasier, Z., Egbejimi, O., Kueht, M., & Young, M. E., (2010). Time-of-day-dependent dietary fat consumption influences multiple cardiometabolic syndrome parameters in mice. *International Journal of Obesity (Lond), 34*(11), 1589-1598. http://dx.doi.org/10.1038/ijo.2010.63

Chan, W., Brown, J., & Buss, D. (1994). *Miscellaneous Foods. Fourth supplement to 5th Edition of McCance & Widdowson's The Composition of Foods*. Cambridge: The Royal Soceity of Chemsitry. http://dx.doi.org/10.1039/9781849732604

Chane, W., Brown, J., Lee, S., & Buss, D. (1995). *Meat, Poultry and Game. Fifth supplement to 5th Edition of McCance & Widdowson's The Composition of Foods*. Cambridge: The Royal Society of Cambridge. http://dx.doi.org/10.1039/9781849732475

Cho, S., Dietrich, M., Brown, C. J., Clark, C. A., & Block, G. (2003). The effect of breakfast type on total daily energy intake and body mass index: results from the Third National Health and Nutrition Examination Survey (NHANES III). *Journal of the American College of Nutrition, 22*(4), 296-302. http://dx.doi.org/10.1080/07315724.2003.10719307

Cole, T. J., Bellizzi, M. C., Flegal, K. M., & Dietz, W. H. (2000). Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ, 320*(7244), 1240-1243. http://dx.doi.org/10.1136/bmj.320.7244.1240

Cole, T. J., Flegal, K. M., Nicholls, D., & Jackson, A. A. (2007). Body mass index cut offs to define thinness in children and adolescents: international survey. *BMJ, 335*(7612), 194. http://dx.doi.org/10.1136/bmj.39238.399444.55

Corder, K., van Sluijs, E. M. F., Steele, R. M., Stephen, A. M., Dunn, V., Bamber, D., Goodyer, I., Griffin, S. J., & Ekelund, U. (2011). Breakfast consumption and physical activity in British adolescents. *British Journal of Nutrition, 105*(2), 316-321. http://dx.doi.org/10.1017/S0007114510003272

deCastro, J. M. (2007). The time of day and the proportions of macronutrients eaten are related to total daily food intake. *British Journal of Nutrition, 98*(5), 1077-1083.

deCastro, J. M., Bellisle, F., Feunekes, G. I. J., Dalix, A. M., & DeGraaf, C. (1997). Culture and meal patterns: A comparison of the food intake of free-living American, Dutch, and French students. *Nutrition Research, 17*(5), 807-829. http://dx.doi.org/10.1016/S0271-5317(97)00050-X

Deshmukh-Taskar, P., Nicklas, T. A., Radcliffe, J. D., O'Neil, C. E., & Liu, Y. (2012). The relationship of breakfast skipping and type of breakfast consumed with overweight/obesity, abdominal obesity, other cardiometabolic risk factors and the metabolic syndrome in young adults. The National Health and Nutrition Examination Survey (NHANES): 1999-2006. *Public Health Nutrition*, 1-10.

Deshmukh-Taskar, P. R., Nicklas, T. A., O'Neil, C. E., Keast, D. R., Radcliffe, J. D., & Cho, S. (2010). The relationship of breakfast skipping and type of breakfast consumption with nutrient intake and weight status in children and adolescents: the National Health and Nutrition Examination Survey 1999-2006. *Journal of the American Dietic Association, 110*(6), 869-878. http://dx.doi.org/10.1016/j.jada.2010.03.023

Dubois, L., Farmer, A., Girard, M., & Peterson, K. (2007). Regular sugar-sweetened beverage consumption between meals increases risk of overweight among preschool-aged children. *Journal of the American Dietic Association, 107*(6), 924-934; discussion 934-925. http://dx.doi.org/10.1016/j.jada.2007.03.004

Emmett, P. (2009). Dietary assessment in the Avon Longitudinal Study of Parents and Children. *European Journal Clinical Nutrition, 63 Suppl 1*, S38-44. http://dx.doi.org/10.1038/ejcn.2008.63

Farshchi, H. R., Taylor, M. A., & Macdonald, I. A., (2004). Regular meal frequency creates more appropriate insulin sensitivity and lipid profiles compared with irregular meal frequency in healthy lean women. *European Journal of Clinical Nutrition, 58*(7), 1071-1077. http://dx.doi.org/10.1038/sj.ejcn.1601935

Farshchi, H. R., Taylor, M. A., & Macdonald, I. A. (2005a). Beneficial metabolic effects of regular meal frequency on dietary thermogenesis, insulin sensitivity, and fasting lipid profiles in healthy obese women. *American Journal of Clinical Nutrition, 81*(1), 16-24.

Farshchi, H. R., Taylor, M. A., & Macdonald, I. A. (2005b). Deleterious effects of omitting breakfast on insulin sensitivity and fasting lipid profiles in healthy lean women. *American Journal of Clinical Nutrition, 81*(2), 388-396.

Gaina, A., Sekine, M., Chandola, T., Marmot, M., & Kagamimori, S. (2009). Mother employment status and nutritional patterns in Japanese junior high schoolchildren. *International Journal of Obesity, 33*(7), 753-757. http://dx.doi.org/10.1038/ijo.2009.103

Boyd, A., Golding, J., Macleod, J., Lawlor, D. A., Fraser, A., Henderson, J., Molloy, L., Ness, A., Ring, S., & Davey Smith G. (2013). Cohort profile: The Children of the 90s - the index offspring of the Avon Longitudinal Study of Parents and Children. *International Journal of Epidemiology*, 42(1), 111-27.
Gregory, J. R., Collins, D. L., Davies, P. S. W., Hughes, J. M., & Clarke, P. C. (1995). *The Diet and Nutrition Survey: children aged 1.5-4.5 years* (Vol. 1 Report of the diet and nutrition survey). London: The Stationary Office.

Gregory, J. R., & Lowe, S. (2000). *National Diet and Nutrition Survey: young people aged 4–18 years* (Vol. 1 Report of the diet and nutrition survey.). London: The Statitionary Office.

Hallstrom, L., Labayen, I., Ruiz, J. R., Patterson, E., Vereecken, C. A., Breidenassel, C., Gottrand, F., Huybrechts, I., Manios, Y., Mistura, L., Widhalm, K., Kondaki, K., Moreno, L. A., & Sjostrom, M. (2012). Breakfast consumption and CVD risk factors in European adolescents: the HELENA (Healthy Lifestyle in Europe by Nutrition in Adolescence) Study. *Public Health Nutrition*, 1-10.

Holland, B., Unwin, I., & Buss, D. (1991). *Vegetables, Herbs and Spices. Fifth supplement to 4th Edition of McCance & Widdowson's The Composition of Foods*. Cambridge: The Royal Society of Chemistry. http://dx.doi.org/10.1039/9781849732499

Holland, B., Unwin, I., & Buss, D. . (1988). *Cereals and Cereal Products. Third supplement to 4th Edition of McCance & Widdowson's The Composition of Foods.* . Cambridge: The Royal Society of Chemistry.

Holland, B., Unwin, I., & D., Buss. (1989). Milk Products and Eggs. Fourth supplement to 4th Edition of McCance & Widdowson's The Composition of Foods. Cambridge: The Royal Society of Chemistry.

Holland, B., Welch, A., Unwin, I., Buss, D., Paul, A. , & Southgate, D. (1991). *McCance &Widdowson's The Composition of Foods, 5th Edition*. Cambridge: The Royal Society of Chemistry.

Holland, B., Welsh, A., & Buss, D. (1992a). *Fruits and Nuts. First supplement to 5th Edition of McCance & Widdowson's The Composition of Foods*. Cambridge: The Royal Society of Chemsitry. http://dx.doi.org/10.1039/9781849732482

Holland, B., Welsh, A., & Buss, D. (1992b). *Vegetable Dishes. Second supplement to 5th Edition of McCance & Widdowson's The Composition of Foods*. Cambridge: The Royal Society of Chemistry.

Holland, D., Brown, J., & Buss, D. (1993). *Fish and Fish Products. Third supplement to 5th Edition of McCance & Widdowson's The Composition of Foods*. Cambridge: The Royal Society of Chemistry. http://dx.doi.org/10.1039/9781849732611

Huang, W., Ramsey, K. M., Marcheva, B., & Bass, J. (2011). Circadian rhythms, sleep, and metabolism. *Journal of Clinical Investigation, 121*(6), 2133-2141. http://dx.doi.org/10.1172/JCI46043

Johnson, L., Mander, A. P., Jones, L. R., Emmett, P. M., & Jebb, S. A. (2007). Is sugar-sweetened beverage consumption associated with increased fatness in children? *Nutrition, 23*(7-8), 557-563. http://dx.doi.org/10.1016/j.nut.2007.05.005

Johnson, L., Mander, A. P., Jones, L. R., Emmett, P. M., & Jebb, S. A. (2008). A prospective analysis of dietary energy density at age 5 and 7 years and fatness at 9 years among UK children. *Int J Obes (Lond), 32*(4), 586-593. http://dx.doi.org/10.1038/sj.ijo.0803746

Keski-Rahkonen, A., Kaprio, J., Rissanen, A., Virkkunen, M., & Rose, R. J. (2003). Breakfast skipping and health-compromising behaviors in adolescents and adults. *European Journal of Clinical Nutrition, 57*(7), 842-853. http://dx.doi.org/10.1038/sj.ejcn.1601618

Larson, N., Fulkerson, J., Story, M., & Neumark-Sztainer, D. (2012). Shared meals among young adults are associated with better diet quality and predicted by family meal patterns during adolescence. *Public Health Nutrition*, 1-11.

Lowden, A., Moreno, C., Holmback, U., Lennernas, M., & Tucker, P. (2010). Eating and shift work - effects on habits, metabolism and performance. *Scandinavian Journal of Work, Environment & Health, 36*(2), 150-162. http://dx.doi.org/10.5271/sjweh.2898

Mahoney, C. R., Taylor, H. A., Kanarek, R. B., & Samuel, P. (2005). Effect of breakfast composition on cognitive processes in elementary school children. *Physiology & Behavior, 85*(5), 635-645. http://dx.doi.org/10.1016/j.physbeh.2005.06.023

Mannino, M. L., Lee, Y., Mitchell, D. C., Smiciklas-Wright, H., & Birch, L. L. (2004). The quality of girls' diets declines and tracks across middle childhood. *International Journal of Behavioral Nutrition and Physical Activity, 1*(1), 5. http://dx.doi.org/10.1186/1479-5868-1-5

Nicklas, T. A., Baranowski, T., Cullen, K. W., & Berenson, G. (2001). Eating patterns, dietary quality and obesity. *Journal of the American College of Nutrition, 20*(6), 599-608. http://dx.doi.org/10.1080/07315724.2001.10719064

Piernas, C., & Popkin, B. M. (2010a). Snacking increased among U.S. adults between 1977 and 2006. *J Nutrition, 140*(2), 325-332. http://dx.doi.org/10.3945/jn.109.112763

Piernas, C., & Popkin, B. M. (2010b). Trends in snacking among U.S. children. *Health Affairs (Millwood), 29*(3), 398-404. http://dx.doi.org/10.1377/hlthaff.2009.0666

Poppitt, S. D., Swann, D., Black, A. E., & Prentice, A. M. (1998). Assessment of selective under-reporting of food intake by both obese and non-obese women in a metabolic facility. *International journal of obesity and related metabolic disorders : journal of the International Association for the Study of Obesity, 22*(4), 303-311. http://dx.doi.org/10.1038/sj.ijo.0800584

Prynne, C. J., Paul, A. A., Mishra, G. D., Greenberg, D. C., & Wadsworth, M. E. (2005). Changes in intake of key nutrients over 17 years during adult life of a British birth cohort. *British Journal of Nutrition, 94*(3), 368-376. http://dx.doi.org/10.1079/BJN20041404

Rampersaud, G. C., Pereira, M. A., Girard, B. L., Adams, J., & Metzl, J. D. (2005). Breakfast habits, nutritional status, body weight, and academic performance in children and adolescents. *Journal of the American Dietic Association, 105*(5), 743-760; quiz 761-742. http://dx.doi.org/10.1016/j.jada.2005.02.007

Rennie, K. L., Coward, A., & Jebb, S. A. (2007). Estimating under-reporting of energy intake in dietary surveys using an individualised method. *British Journal of Nutrition, 97*(6), 1169-1176. http://dx.doi.org/10.1017/S0007114507433086

Samuelson, G. (2000). Dietary habits and nutritional status in adolescents over Europe. An overview of current studies in the Nordic countries. *European Journal of Clinical Nutrition, 54 Suppl 1*, S21-28. http://dx.doi.org/10.1038/sj.ejcn.1600980

Sato, M., Nakamura, K., Ogata, H., Miyashita, A., Nagasaka, S., Omi, N., Yamaguchi, S., Hibi, M., Umeda, T., Nakaji, S., & Tokuyama, K. (2011). Acute effect of late evening meal on diurnal variation of blood glucose and energy metabolism. *Obesity Research & Clinical Practice, 5*(3), E220-E228. http://dx.doi.org/10.1016/j.orcp.2011.02.001

Savige, G., Macfarlane, A., Ball, K., Worsley, A., & Crawford, D. (2007). Snacking behaviours of adolescents and their association with skipping meals. *International Journal of Behavioral Nutrition and Physical Activity, 4*, 36. http://dx.doi.org/10.1186/1479-5868-4-36

Scheer, F. A., Hilton, M. F., Mantzoros, C. S., & Shea, S. A. (2009). Adverse metabolic and cardiovascular consequences of circadian misalignment. *Proceedings of the National Academy of Sciences U S A, 106*(11), 4453-4458. http://dx.doi.org/10.1073/pnas.0808180106

Sierra-Johnson, J., Unden, A. L., Linestrand, M., Rosell, M., Sjogren, P., Kolak, M., De Faire, U., Fisher, R. M., & Hellenius, M. L. (2008). Eating meals irregularly: a novel environmental risk factor for the metabolic syndrome. *Obesity (Silver Spring), 16*(6), 1302-1307. http://dx.doi.org/10.1038/oby.2008.203

te Velde, S. J., Twisk, J. W., & Brug, J. (2007). Tracking of fruit and vegetable consumption from adolescence into adulthood and its longitudinal association with overweight. *British Journal of Nutrition, 98*(2), 431-438. http://dx.doi.org/10.1017/S0007114507721451

Whitton, C., Nicholson, S. K., Roberts, C., Prynne, C. J., Pot, G. K., Olson, A., Fitt, E., Cole, D., Teucher, B., Bates, B., Henderson, H., Pigott, S., Deverill, C., Swan, G., & Stephen, A. M. (2011). National Diet and Nutrition Survey: UK food consumption and nutrient intakes from the first year of the rolling programme and comparisons with previous surveys. *British Journal of Nutrition, 106*(12), 1899-1914. http://dx.doi.org/10.1017/S0007114511002340

Wrieden, W. L., Longbottom, P. J., Adamson, A. J., Ogston, S. A., Payne, A., Haleem, M. A., & Barton, K. L. (2008). Estimation of typical food portion sizes for children of different ages in Great Britain. *Britush Journal of Nutrition, 99*(6), 1344-1353. http://dx.doi.org/10.1017/s0007114507868516

Table 1. Unadjusted energy and macronutrient intake at different eating occasions in boys and girls aged 10 and 13 years in ALSPAC. Data includes non-consumers.

		Boys				Girls			
		Age 10 (n=1561)		Age 13 (n=1561)		Age 10 (n=1759)		Age 13 (n=1759)	
		Absolute intake	mean* (95% CI)	Absolute intake	mean (95% CI)	Absolute intake	mean (95% CI)	Absolute intake	mean (95% CI)
		g	%	g	%	g	%	g	%
Breakfast	Energy	327	16.6 (16.3,16.9)	317	14.7 (14.4,15.1)	280	15.8 (15.5,16.1)	235	13.3 (12.9,13.6)
	Carbohydrate	53	20.1 (19.8,20.5)	52	18 (17.5,18.4)	45	19 (18.7,19.4)	39	16.3 (15.9,16.8)
	Protein	10	15.8 (15.5,16.1)	11	13.8 (13.4,14.2)	9	14.9 (14.5,15.2)	8	12 (11.7,12.4)
	Fat	10	12 (11.7,12.4)	9	10.5 (10.1,10.9)	9	11.7 (11.4,12.1)	7	9.4 (9,9.7)
Mid-morning	Energy	96	4.9 (4.6,5.1)	170	7.8 (7.5,8.2)	99	5.6 (5.3,5.8)	147	8.3 (8,8.6)
	Carbohydrate	14	5.3 (5.1,5.6)	24	8.5 (8.1,8.8)	14	6.1 (5.8,6.4)	21	9 (8.7,9.4)
	Protein	2	3 (2.8,3.2)	4	5.6 (5.3,5.9)	2	3.4 (3.2,3.6)	4	5.8 (5.5,6.1)
	Fat	4	4.9 (4.7,5.2)	7	7.9 (7.5,8.3)	4	5.6 (5.3,5.9)	6	8.3 (7.9,8.7)
Lunch	Energy	580	29.7 (29.4,30.1)	591	27.6 (27.2,28.1)	544	31 (30.6,31.3)	499	28.3 (27.8,28.7)
	Carbohydrate	72	27.7 (27.4,28.1)	75	26.2 (25.8,26.7)	68	29.1 (28.7,29.4)	63	26.8 (26.4,27.3)
	Protein	19	28.9 (28.5,29.4)	21	26.7 (26.2,27.2)	18	30.2 (29.7,30.6)	17	27.4 (26.9,27.9)
	Fat	26	33 (32.5,33.5)	25	30.1 (29.5,30.6)	24	34 (33.5,34.5)	22	30.6 (30.1,31.2)
Mid-afternoon	Energy	178	9 (8.6,9.3)	159	7.2 (6.9,7.6)	167	9.4 (9,9.7)	138	7.7 (7.3,8.2)
	Carbohydrate	26	9.6 (9.3,10)	22	7.5 (7.2,7.9)	24	9.9 (9.6,10.3)	19	7.9 (7.5,8.3)
	Protein	4	6 (5.6,6.4)	4	5.7 (5.3,6.1)	4	6.9 (6.5,7.3)	4	6.5 (6,6.9)
	Fat	7	9.2 (8.8,9.6)	6	7.4 (7,7.8)	7	9.5 (9.1,9.9)	6	8.1 (7.6,8.5)
Dinner	Energy	601	30.7 (30.2,31.1)	596	28 (27.4,28.6)	528	29.8 (29.4,30.3)	493	28.1 (27.6,28.7)
	Carbohydrate	73	27.6 (27.2,28.1)	71	24.9 (24.3,25.4)	64	27.1 (26.6,27.5)	59	25.1 (24.6,25.7)
	Protein	25	38.2 (37.6,38.8)	27	34.9 (34.2,35.7)	22	37.1 (36.5,37.7)	22	35.4 (34.7,36.2)
	Fat	25	32 (31.5,32.6)	25	29.7 (29,30.4)	22	31 (30.4,31.5)	20	29.4 (28.7,30)
Late evening	Energy	158	7.9 (7.5,8.2)	239	10.9 (10.4,11.4)	134	7.4 (7,7.7)	188	10.4 (10,10.9)
	Carbohydrate	22	8 (7.7,8.4)	31	10.6 (10.1,11.1)	18	7.5 (7.2,7.8)	24	10.2 (9.7,10.6)
	Protein	5	7.5 (7.1,7.9)	9	11.5 (10.9,12.1)	4	7.1 (6.7,7.5)	7	11 (10.5,11.6)
	Fat	6	7.7 (7.3,8.1)	10	11.1 (10.5,11.7)	5	7.2 (6.9,7.6)	8	10.5 (10,11.1)
Extras	Energy	25	1.3 (1.1,1.4)	82	3.6 (3.4,3.9)	19	1.1 (0.9,1.2)	71	3.9 (3.6,4.2)
	Carbohydrate	4	1.5 (1.4,1.7)	13	4.3 (4,4.7)	3	1.3 (1.1,1.5)	11	4.6 (4.3,4.9)
	Protein	0	0.5 (0.5,0.6)	1	1.7 (1.6,1.9)	0	0.5 (0.4,0.5)	1	1.8 (1.7,2)
	Fat	1	1.1 (1,1.3)	3	3.4 (3.1,3.7)	1	0.9 (0.8,1.1)	3	3.7 (3.4,4)
Day	Energy	1965	-	2152	-	1772	-	1771	-
	Carbohydrate	264	-	288	-	237	-	236	-
	Protein	67	-	77	-	59	-	63	-
	Fat	79	-	85	-	72	-	71	-

* Mean of % of daily intake

Table 2. Unadjusted energy and macronutrient intake at different eating occasions in boys and girls aged 10 and 13 years in ALSPAC including only the consumers of each meal. (n shows the number of consumers within each eating occasion)

		Boys						Girls					
		Age 10			Age 13			Age 10			Age 13		
		N	Absolute intake	mean* (95% CI)	N	Absolute intake	mean (95% CI)	N	Absolute intake	mean (95% CI)	N	Absolute intake	mean (95% CI)
			g	%		g	%		g	%		g	%
Breakfast	Energy	1548	329.56	16.8 (16.5,17.1)	1509	327.92	15.2 (14.9,15.6)	1740	283.36	16 (15.7,16.3)	1638	251.96	14.2 (13.9,14.6)
	Carbohydrate	1548	53.49	20.3 (19.9,20.6)	1509	53.45	18.6 (18.2,19)	1740	45.58	19.2 (18.9,19.6)	1638	41.4	17.5 (17.1,17.9)
	Protein	1548	10.59	15.9 (15.6,16.3)	1509	11.01	14.3 (13.9,14.7)	1740	8.86	15 (14.7,15.3)	1638	8.12	12.9 (12.6,13.3)
	Fat	1548	9.69	12.1 (11.8,12.5)	1509	9.33	10.9 (10.5,11.3)	1740	8.62	11.8 (11.5,12.2)	1638	7.18	10 (9.7,10.4)
Mid-morning	Energy	1254	119.79	6.1 (5.8,6.3)	1313	201.63	9.3 (9,9.7)	1466	118.69	6.7 (6.4,6.9)	1552	166.31	9.4 (9.1,9.7)
	Carbohydrate	1254	17.6	6.6 (6.3,6.9)	1313	29.02	10.1 (9.7,10.4)	1466	17.38	7.3 (7,7.6)	1552	24.1	10.2 (9.9,10.6)
	Protein	1254	2.55	3.7 (3.5,4)	1313	5.21	6.7 (6.3,7)	1466	2.41	4.1 (3.8,4.3)	1552	4.06	6.5 (6.2,6.9)
	Fat	1254	5.26	6.2 (5.9,6.5)	1313	8.33	9.4 (9,9.8)	1466	5.14	6.7 (6.4,7)	1552	6.83	9.4 (9,9.8)
Lunch	Energy	1560	580	29.7 (29.4,30.1)	1554	593.35	27.8 (27.3,28.2)	1758	544.56	31 (30.6,31.4)	1748	502.61	28.4 (28,28.9)
	Carbohydrate	1560	72.48	27.7 (27.4,28.1)	1554	74.86	26.3 (25.9,26.8)	1758	68.1	29.1 (28.7,29.5)	1748	63.22	27 (26.6,27.4)
	Protein	1560	19.13	29 (28.5,29.4)	1554	20.64	26.8 (26.3,27.3)	1758	17.65	30.2 (29.7,30.6)	1748	17.26	27.6 (27.1,28.1)
	Fat	1560	25.75	33 (32.5,33.5)	1554	25.62	30.2 (29.6,30.8)	1758	24.29	34 (33.5,34.5)	1748	21.83	30.8 (30.3,31.4)
Mid-afternoon	Energy	1446	192.69	9.7 (9.3,10)	1206	205.27	9.4 (8.9,9.8)	1632	180.14	10.1 (9.7,10.4)	1318	184.49	10.3 (9.9,10.8)
	Carbohydrate	1446	27.93	10.4 (10.1,10.8)	1206	28.77	9.8 (9.4,10.2)	1632	25.63	10.7 (10.3,11)	1318	25.12	10.6 (10.1,11)
	Protein	1446	4.32	6.5 (6.1,6.9)	1206	5.81	7.4 (6.9,7.9)	1632	4.38	7.4 (7,7.8)	1318	5.42	8.6 (8.1,9.2)
	Fat	1446	8.24	9.9 (9.5,10.3)	1206	8.74	9.6 (9.1,10)	1632	7.78	10.3 (9.8,10.7)	1318	7.96	10.8 (10.2,11.3)
Dinner	Energy	1555	602.94	30.8 (30.3,31.3)	1523	610.43	28.7 (28.1,29.2)	1745	532.52	30.1 (29.6,30.5)	1716	505.47	28.8 (28.3,29.4)
	Carbohydrate	1555	72.81	27.8 (27.3,28.2)	1523	72.41	25.5 (25,26)	1745	64.3	27.3 (26.9,27.7)	1716	60.2	25.8 (25.3,26.3)
	Protein	1555	25.54	38.4 (37.7,39)	1523	27.51	35.8 (35.1,36.5)	1745	22.18	37.4 (36.8,38)	1716	22.86	36.3 (35.6,37)
	Fat	1555	25.32	32.2 (31.6,32.7)	1523	25.52	30.4 (29.8,31.1)	1745	22.56	31.2 (30.7,31.8)	1716	20.98	30.1 (29.5,30.8)
Late evening	Energy	1327	185.84	9.2 (8.9,9.6)	1325	281.43	12.9 (12.3,13.4)	1449	162.32	9 (8.6,9.3)	1497	220.89	12.3 (11.8,12.8)
	Carbohydrate	1327	25.47	9.4 (9.1,9.8)	1325	36.63	12.5 (12,13)	1449	22.04	9.1 (8.7,9.5)	1497	28.74	12 (11.5,12.4)
	Protein	1327	6.14	8.9 (8.4,9.3)	1325	10.8	13.6 (12.9,14.3)	1449	5.28	8.6 (8.2,9.1)	1497	8.36	13 (12.3,13.6)
	Fat	1327	7.76	9.1 (8.7,9.5)	1325	11.64	13 (12.4,13.7)	1449	6.86	8.8 (8.4,9.2)	1497	9.28	12.4 (11.8,12.9)
Extras	Energy	438	90.74	4.5 (4.1,4.9)	917	139.29	6.2 (5.8,6.6)	422	79.88	4.5 (4,4.9)	1150	108.6	6 (5.6,6.3)
	Carbohydrate	438	14.88	5.5 (5,5.9)	917	22.48	7.4 (6.9,7.8)	422	13.21	5.4 (4.9,6)	1150	16.99	7 (6.6,7.4)
	Protein	438	1.4	2 (1.7,2.2)	917	2.48	2.9 (2.7,3.2)	422	1.26	2 (1.7,2.2)	1150	1.98	2.8 (2.6,3)
	Fat	438	3.98	4 (3.6,4.5)	917	6.08	5.7 (5.3,6.2)	422	3.7	3.9 (3.4,4.4)	1150	5.17	5.7 (5.3,6.1)
Day	Energy	1561	1965.23	-	1561	2152.15	-	1759	1771.75	-	1759	1771.18	-
	Carbohydrate	1561	263.83	-	1561	287.75	-	1759	236.48	-	1759	235.74	-
	Protein	1561	66.51	-	1561	77.14	-	1759	58.95	-	1759	62.71	-
	Fat	1561	78.95	-	1561	84.94	-	1759	72.22	-	1759	70.67	-

- Mean of % of daily intake

Figure 1. Percentage of consumers and non-consumers at any given eating occasion in boys and girls aged 10 and 13 years.

For every eating occasions, asterisk denotes significant differences in proportion of consumers between ages 10 and 13 years; * P<0.01 ** P<0.001

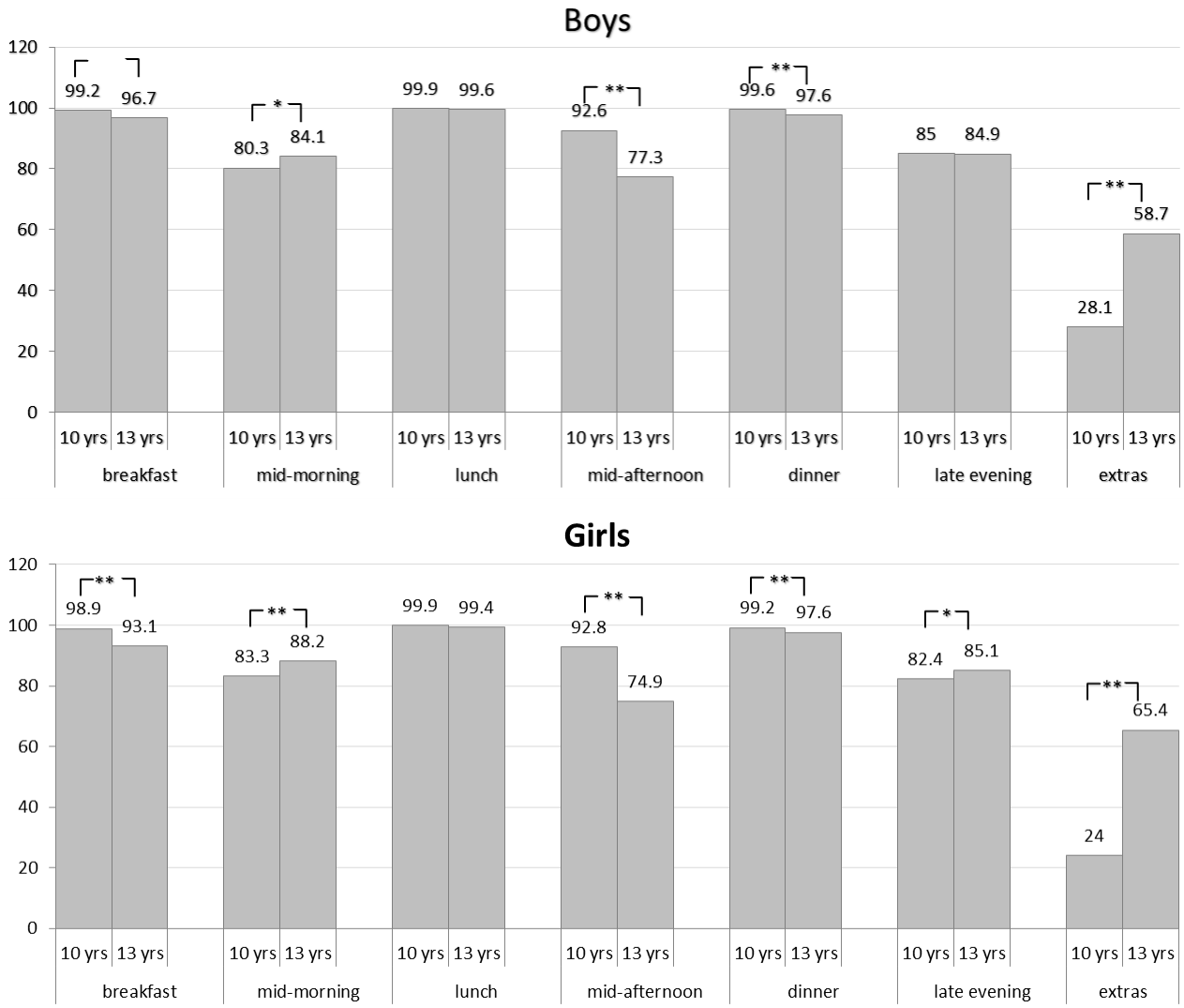


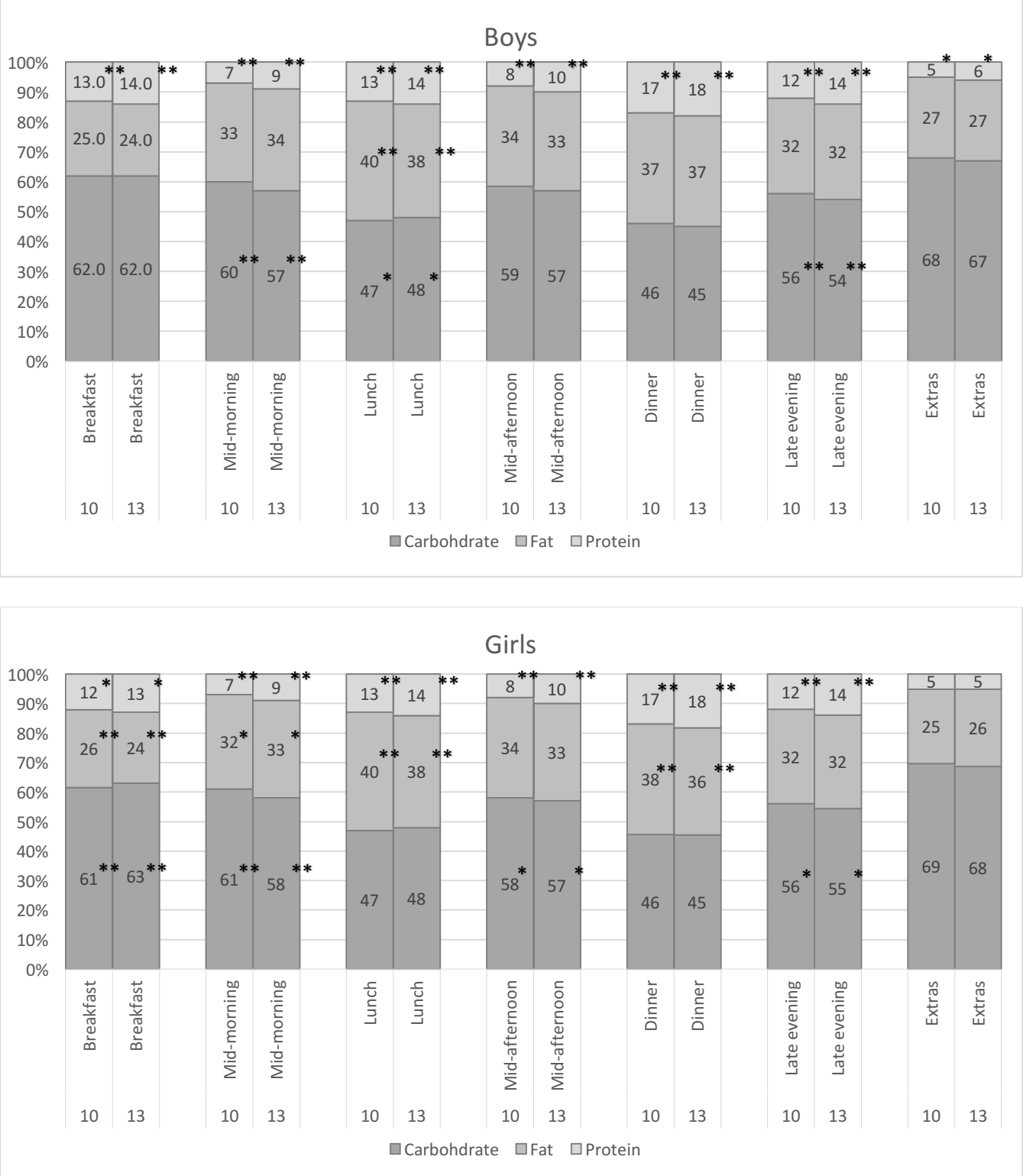
Table 3. Changes in energy intake and macronutrient composition of eating occasions in boy and girls between ages 10 and 13 years. Macronutrients expressed as proportion of energy intake.

			Energy			Carbohydrate (% Energy)			Protein (% Energy)			Fat (% Energy)		
			β*	95%CI	P-value	β	95%CI	P-value	β	95%CI	P-value	β	95%CI	P-value
Boys (n= 3122)	Breakfast	Constant	16.4	(15,17.9)	<0.001	61.6	(59.1,64.2)	<0.001	11.6	(10.8,12.4)	<0.001	26.8	(24.4,29.2)	<0.001
		Age	-1.9	(-2.3,-1.5)	<0.001	0.1	(-0.7,0.8)	0.824	0.5	(0.3,0.8)	<0.001	-0.6	(-1.3,0.1)	0.084
	Mid-morning	Constant	6.5	(5.2,7.7)	<0.001	60.1	(56,64.1)	<0.001	6.2	(5,7.4)	<0.001	33.7	(29.9,37.6)	<0.001
		Age	3.1	(2.7,3.5)	<0.001	-2.9	(-4.3,-1.5)	<0.001	1.8	(1.4,2.2)	<0.001	1.1	(-0.3,2.4)	0.112
	Lunch	Constant	28.2	(26.5,29.9)	<0.001	46.9	(45.2,48.7)	<0.001	12.6	(11.7,13.4)	<0.001	40.5	(38.9,42.2)	<0.001
		Age	-2.2	(-2.8,-1.6)	<0.001	0.8	(0.2,1.4)	0.008	0.6	(0.3,0.8)	<0.001	-1.4	(-1.9,-0.8)	<0.001
	Mid-afternoon	Constant	10.5	(9,12)	<0.001	57.7	(54.1,61.4)	<0.001	6.9	(5.7,8.2)	<0.001	35.4	(32.1,38.7)	<0.001
		Age	-1.6	(-2.1,-1.1)	<0.001	-1.4	(-2.6,-0.1)	0.037	2.1	(1.6,2.5)	<0.001	-0.7	(-1.9,0.5)	0.231
	Dinner	Constant	28.7	(26.5,31)	<0.001	46.7	(45,48.4)	<0.001	15.9	(14.7,17)	<0.001	37.4	(35.9,39)	<0.001
		Age	-2.9	(-3.7,-2.2)	<0.001	-0.7	(-1.3,-0.1)	0.015	0.9	(0.5,1.3)	<0.001	-0.2	(-0.7,0.4)	0.572
	Late evening	Constant	8.4	(6.5,10.3)	<0.001	53.8	(49.7,57.8)	<0.001	10.4	(8.8,12.1)	<0.001	35.8	(32.4,39.2)	<0.001
		Age	3.2	(2.6,3.8)	<0.001	-2.5	(-3.8,-1.2)	<0.001	1.7	(1.1,2.2)	<0.001	0.8	(-0.3,2)	0.138
	Extras	Constant	1.2	(0.3,2.1)	0.01	59.7	(53,66.4)	<0.001	5.2	(3.5,6.8)	<0.001	35	(28.8,41.2)	<0.001
		Age	2.4	(2.1,2.7)	<0.001	-2.2	(-4.7,0.3)	0.084	1	(0.4,1.7)	0.001	1.2	(-1.2,3.5)	0.338
Girls (n=3518)	Breakfast	Constant	16.1	(14.8,17.5)	<0.001	61.5	(59.2,63.9)	<0.001	12.1	(11.3,12.9)	<0.001	26.4	(24.1,28.6)	<0.001
		Age	-2.6	(-3.1,-2.2)	<0.001	1.5	(0.7,2.3)	<0.001	0.3	(0.1,0.6)	0.019	-1.8	(-2.6,-1.1)	<0.001
	Mid-morning	Constant	6.3	(5.2,7.4)	<0.001	58	(54.5,61.6)	<0.001	5.3	(4.3,6.3)	<0.001	36.7	(33.3,40)	<0.001
		Age	2.9	(2.5,3.3)	<0.001	-3.3	(-4.7,-2)	<0.001	1.5	(1.1,1.8)	<0.001	1.9	(0.6,3.2)	0.003
	Lunch	Constant	30.5	(28.9,32.1)	<0.001	46.3	(44.7,47.9)	<0.001	12.2	(11.5,12.9)	<0.001	41.6	(40.1,43.1)	<0.001
		Age	-2.8	(-3.4,-2.2)	<0.001	0.5	(0,1.1)	0.069	0.7	(0.4,0.9)	<0.001	-1.2	(-1.7,-0.6)	<0.001
	Mid-afternoon	Constant	9.5	(8,10.9)	<0.001	55.8	(52.4,59.1)	<0.001	6.8	(5.6,8)	<0.001	37.4	(34.4,40.4)	<0.001
		Age	-1.7	(-2.2,-1.1)	<0.001	-1.7	(-2.9,-0.4)	0.011	1.6	(1.1,2)	<0.001	0.1	(-1.1,1.2)	0.906
	Dinner	Constant	27.6	(25.5,29.7)	<0.001	47.8	(46.1,49.5)	<0.001	15.8	(14.8,16.9)	<0.001	36.4	(34.8,37.9)	<0.001
		Age	-2	(-2.7,-1.3)	<0.001	0	(-0.6,0.6)	0.944	1.2	(0.8,1.6)	<0.001	-1.2	(-1.7,-0.6)	<0.001
	Late evening	Constant	8.8	(7.1,10.5)	<0.001	54.7	(51,58.4)	<0.001	10.5	(8.9,12.1)	<0.001	34.9	(31.8,38)	<0.001
		Age	3.3	(2.7,3.9)	<0.001	-2	(-3.3,-0.7)	0.003	1.8	(1.2,2.3)	<0.001	0.3	(-0.9,1.4)	0.661
	Extras	Constant	1.2	(0.4,2)	0.005	66.3	(59.9,72.7)	<0.001	5.1	(3.5,6.6)	<0.001	28.7	(22.9,34.6)	<0.001
		Age	2.9	(2.6,3.2)	<0.001	-1.5	(-4.2,1.1)	0.261	0.1	(-0.5,0.8)	0.641	1.4	(-1,3.8)	0.250

- Coefficient from linear mixed model adjusted for maternal education and child's BMI.

Figure 2. Unadjusted means and 95% CI of macronutrient intake across different eating occasions in boys and girls at ages 10 and 13 years. Macronutrients expressed as percentage of energy consumed at a given eating occasion.

Within every eating occasion, a single * indicates a significant difference in the intake of a macronutrient between ages 10 and 13 years with a p-value <0.01. A double ** indicates a significant difference in the intake of a macronutrient between ages 10 and 13 years with a *p*-value of <0.001. P-values derived from mixed linear models adjusted for maternal education and child's BMI.



Bi-directional relationships between body mass index and height from three to seven years of age: an analysis of children in the United Kingdom Millennium Cohort Study

Laia Bécares University of Manchester, UK
Yvonne Kelly University College London, UK
Scott Montgomery Örebro University, Sweden
Amanda Sacker University College London, UK,
a.sacker@ucl.ac.uk

(Received January 2015 Revised September 2015) http://dx.doi.org/10.14301/llcs.v7i1.339

Abstract

Adiposity and height are known to correlate in childhood but it is less clear whether height and weight gain occur in synergy. We investigate the bidirectional relationships between measures of height and body mass index (BMI) – an indicator of adiposity - and their rates of change. The sample comprises singleton children in the Millennium Cohort Study (N = 11,357). Child anthropometrics measured by trained interviewers at ages three, five and seven years (2003-2009) were transformed to standardised scores based on 1990 British Growth Reference data from which piecewise linear models for height and BMI were jointly fitted. At three years of age, zHeight was positively related to subsequent zBMI velocities, whereas zBMI at three years was positively related to zHeight velocity to age five but inversely related to zHeight velocity from five to seven years of age. Age three zBMI predicted zHeight velocity from three to five years more strongly than age three zHeight predicted zBMI velocity over the same period. The rate of change in zHeight was positively correlated with subsequent zBMI velocity and vice versa. This new evidence on the bidirectional relationships between height and BMI velocities sheds light on the early childhood origins of obesity in adulthood and the need to monitor growth as well as weight gain.

Keywords

Body mass index, child growth, height, Millennium Cohort Study, United Kingdom

Introduction

It has long been known that a high degree of adiposity (an excessive accumulation of fat) and height are positively correlated in childhood (Hanks, Newton & Casazza, 2013; Lloyd, Wolff & Whelen, 1961; Samani-Radia & McCarthy, 2011; Wolff, 1955). Children who are taller with a higher body mass index (BMI) – signalling greater adiposity – in childhood have an earlier puberty followed by a slower than average rate of change in height in adolescence (He, He & Karlberg, 2001). This is said to account for the paradoxical positive correlation between height and BMI in childhood but a negative correlation in adulthood (Bosy-Westphal, Plachta-Danielzik, Doerhoefer & Mueller, 2009).

Growth in childhood is affected by many factors including genetics, hormones and nutrition (Fischbein & Pedersen, 1986; Forbes, 1977; Hindmarsh, Smith, Brook & Matthews, 1987).

Childhood obesity tracks into adulthood (Baird, et al., 2005; Serdula, et al., 1993), and is associated with an increased risk of adult morbidity and mortality (Must, Jacques, Dallal, Bajema & Dietz, 1992; Reilly & Kelly, 2011). Genetic markers of adult obesity have been associated with metabolism (Pearce, et al., 2013), with children's satiety and food intake (Cecil, Tavendale, Watt, Hetherington & Palmer, 2008; Wardle, et al., 2008), and have been linked with height and weight velocities in infancy (Elks, et al., 2010). These associations suggest that associated metabolic and/or appetitive characteristics result in more free energy that is used either for growth or stored as adipose tissue. Consequently, the temporality of rapid growth and accumulation of adiposity will depend in part on available energy and the growth phase of the child. As adipose tissue is an energy store, its accumulation also signals energy available for height-related growth (Wells, 2012).

Although height-related growth follows a relatively predictable path, the transition periods between growth phases (infantile, childhood, juvenile) are sensitive windows of developmental plasticity when metabolic and environmental factors are most influential (Hochberg, 2011).The prevalence of overweight across height quintiles has been found to increase ten-fold from three to ten years of age (Freedman, et al., 2004) suggesting synergy between adiposity and height as children mature. Longitudinally height is positively associated with higher obesity risk: pre-school children initially tall for their age were more likely to be obese at nine year follow-up (Stanojevic, Kain & Uauy, 2007). Studies also find BMI is associated with height velocity, as children's increased BMI preceded advancements in skeletal development and subsequent tall stature (Johnson, et al., 2012) and height velocities between eight and 18 years are predicted by changes in BMI from two to eight years of age (He, et al., 2001).

Longitudinal studies offer the potential to disentangle the relationship between height and adiposity, but to our knowledge, no study has investigated relationships between height and adiposity indicated by BMI and rates of change simultaneously. This study examines the bidirectional relationships between height and BMI z-scores (zHeight and zBMI, hereafter) from ages three to seven years old in a large population-based study of young children in the United Kingdom (UK). We hypothesise that i) as zBMI and zHeight are markers of the availability of free energy through over-nutrition, zHeight and zBMI will correlate positively with zBMI velocity and zHeight velocity; ii) zBMI will correlate more strongly with zHeight velocity than zHeight will correlate with zBMI velocity as zBMI is a more reliable indicator of the availability of energy stored in adipose tissue; and iii) zBMI velocity and zHeight velocity will correlate negatively within the same time period and positively across time periods as energy usage is directed towards increasing height and laying down fat in turn.

Methods

Data

The Millennium Cohort Study (MCS) is a nationally representative longitudinal study of infants born in the UK between September 2000 and January 2002. Families with children who were living in the UK at age nine months were identified through the Department of Work and Pensions Child Benefit system and selected on the basis of where the family was resident shortly after the time of birth. The sample is clustered at the electoral ward (an administrative unit level) such that disadvantaged areas and areas with a high proportion of ethnic minorities are over-represented. More detail on the survey design, recruitment and fieldwork are found elsewhere (Hansen, 2012)

There have been survey sweeps when cohort members were aged about nine months (MCS1), three (MCS2), five (MCS3), seven (MCS4), and 11 years (MCS5). At MCS1, 18,552 families were recruited to the study (85% interview rate). At MCS2 a further 692 families joined the survey, but there were 3,655 unproductive interviews, giving a total of 15,589 (81%). At MCS3 15,246 families (79.2%) and at MCS4 13857 families (72%) were interviewed. This study uses data from MCS2 to MCS4, and successively excluded cohort members if they dropped out of the study before MCS2 (n=1798), were not in the study at MCS4 (n=3,403), were not singletons (n=360), had missing covariate data (n=710) or anthropometric measurement dates were unknown (n=1,648), resulting in an analysis sample comprising 11,357 cohort members (range 11,192 to 11,357 for individual models).

Ethical approval for the MCS was obtained from the relevant ethics committees and parents gave

informed consent before interviews took place, and separate written consent for anthropometric measurements.

Measures

Anthropometric measures, including weight, height, waist circumference and %fat, were taken by trained interviewers. The children were weighed without shoes or outdoor clothes using Tanita HD-305 scales in MCS2 and MCS3 (Tanita UK Ltd, Middlesex, UK), recorded to the nearest 0.1 kg. Weight and body fat in MCS4 was measured using Tanita BF-522W scales. Height was measured using Leicester Height Measure Stadiometers (Seca Ltd, Birmingham, UK), recorded to the nearest 0.1 cm. For MCS2 and MCS3, we replaced missing height and weight measures with data taken from the Personal Child Health Record (n<50) (Walton, Bedford & Dezateux, 2006) In MCS3 and MCS4, waist circumference was measured twice to the nearest completed millimetre on bare skin or over light clothing, using a non-elasticated SECA tape (SECA, Hamburg, Germany) positioned horizontally midway between the costal margin and the iliac crest. If the difference between the two measurements was ≥ 2 cm, a third measurement was taken; the mean of the two closest measures was used. BMI (kg/m^2) was calculated from the height and weight measures after conversion to metric scales where necessary. Child anthropometrics were transformed to standardised scores with the LMS method using 1990 British Growth Reference data (Cole, 1990) Measurement dates were used to derive age at measurement in months.

Growth in height and weight is affected by sex, birth weight, household income and ethnicity (Sacker & Kelly, 2012; Saxena, Ambler, Cole & Majeed, 2004) and measures of these were included in statistical models. Birth weight in kilograms was collected from the main respondent at first contact (MCS1 or MCS2 for participants new to the study). Household income was measured at each age and equivalised using the modified OECD scale to account for the number of people in the household (Hansen, 2012) and log transformed. Ethnicity was grouped into White British, Indian, Pakistani, Bangladeshi, Black Caribbean, Black African and Other.

Analysis

To examine the bidirectional relationships between standardised height scores (zHeight) and standardised BMI scores (zBMI) from ages three to seven, we use piecewise latent growth curve models, an extension of latent growth curves that model nonlinear relations over time by using two or more linear piecewise splines. We set the knot at age five after sensitivity analysis comparing competing models with the knot at the MCS3 mean age (5.2 years), and above the mean age (5.4 years) showed placement of the knot did not affect results.

We jointly model zHeight and zBMI by fitting two piecewise linear models with individually-varying times of observation and two linear slope factors to describe trajectories of change in zHeight and zBMI occurring over two time periods (from age three to five, and from age five to seven). Intercepts and slopes are treated as random variables and allowed to covary. The models included adjustment for birth weight, ethnicity and income and were estimated separately for boys and girls. All equations are provided in the online supplementary material.

Piecewise growth curve models were estimated in Mplus version 7.2 using the full-information robust maximum likelihood (MLR) estimator that adjusts for non-response assuming data are missing at random and produces standard errors robust to non-normality and the non-independence of observations. Analyses take account of the stratified, clustered sample design and are weighted to account for the unequal probability of being sampled.

To ease interpretation we present correlation coefficients and standard errors (SE) between zHeight and zBMI, as well as between baseline measures and growth over time for each individual measure. The correlation coefficients were calculated using the Mplus “Model Constraint: New” option, allowing their standard errors to be estimated using the formula in Kendal and Stuart (1977 p247). All model estimates are presented in the online supplementary material. Because data on waist circumference (zWaist) and %fat are not available at all ages, reduced forms of the zBMI models are estimated and available in online supplementary material.

Results

Table 1 shows the available anthropometric data at ages three, five and seven years in the MCS. Mean values at each age are presented in their original metric and where appropriate in standard deviate scores. Table 2 breaks down these data further for the growth trajectories sample, showing mean zHeight and zBMI by each covariate, confirming relationships reported elsewhere.

Table 1. Anthropometrics at three, five and seven in the MCS

	Age three		Age five		Age seven	
	Raw	Standardised [1]	Raw	Standardised [1]	Raw	Standardised [1]
Boys						
Height (cm)	95.48	0.00	109.62	0.02	122.68	0.14
Weight (kg)	15.56	0.40	19.70	0.35	24.79	0.37
BMI (kg/m^2)	17.03	0.55	16.35	0.48	16.39	0.40
Waist (cm)	n/a	n/a	53.46	0.49	56.52	0.70
%fat [2]	n/a	n/a	n/a	n/a	20.02	n/a
Girls						
Height (cm)	94.26	-0.07	108.78	-0.02	121.88	0.10
Weight (kg)	14.83	0.29	19.36	0.28	24.69	0.28
BMI (kg/m^2)	16.66	0.46	16.30	0.40	16.53	0.31
Waist (cm)	n/a	n/a	53.43	0.63	56.62	0.73
%fat [2]	n/a	n/a	n/a	n/a	22.16	n/a

[1] Standard deviate scores derived using LMS standardisation (Cole, 1990)
[2] Total fat mass/total body mass

zHeight and zBMI growth trajectories

The modelled conditional growth trajectories of zHeight for boys and girls are shown in figure 1. Detailed model estimates are given in online supplementary tables S1 (boys) and S2 (girls). Boys were taller than girls, although both sexes followed similar trajectories of increased zHeight with age and acceleration in zHeight after age five. Baseline zHeight was not significantly related to change between three and five years (Tables 3 and 4, top left quadrant), but was inversely correlated with change between five and seven years, so that children who were taller at age three grew more slowly after five years of age (boys r = -0.06, SE = 0.03; girls r = -0.12, SE = 0.04). Height velocity from three to five years was inversely correlated with height velocity from five to seven years, although this was only significant for girls (boys r = -0.20, SE = 0.14; girls r = -0.29, SE = 0.15).

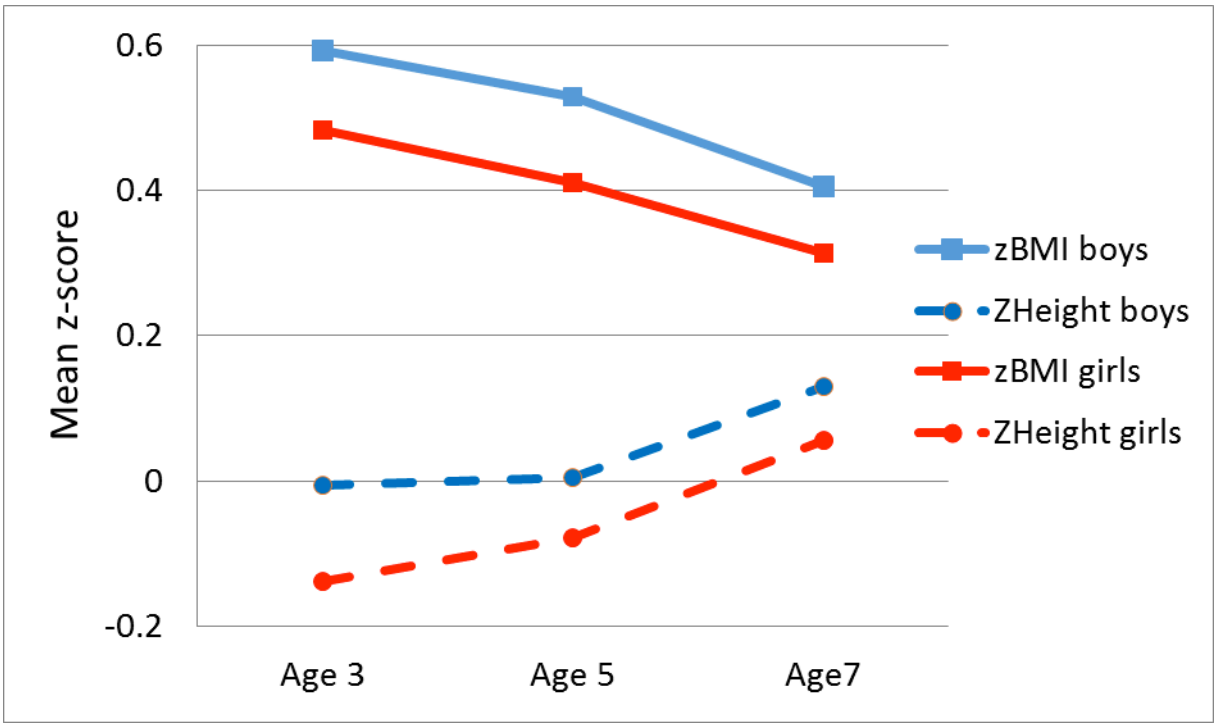
The conditional growth trajectories of zBMI for boys and girls (figure 1) show that boys had higher zBMI than girls, with both following similar trajectories of decreased zBMI with age. The rate that zBMI decreased accelerated somewhat after age five. Baseline zBMI measures were inversely correlated with changes over time (tables 3 and 4, bottom right quadrant): Children with higher zBMI at age three had a greater decline in zBMI from age three to five (boys r = -0.28, SE = 0.14; girls r = -0.29, SE = 0.14) and boys had a greater decline from age five to age seven (r = -0.11, SE 0.04).

Table 2. Mean (95% confidence intervals) of zHeight and zBMI at three, five and seven years by covariate values for 11,357 children in the MCS

		Age 3 years		Age 5 years		Age 7 years	
	N (%) [1]	zHeight	zBMI	zHeight	zBMI	zHeight	zBMI
Sex							
Male	5717 (51)	0.02(-0.02, 0.06)	0.56 (0.52, 0.60)	0.04 (0.00, 0.07)	0.49 (0.45, 0.53)	0.16 (0.12, 0.20)	(0.39 (0.35, 0.43)
Female	5640 (49)	-0.07(-0.11, -0.04)	0.47 (0.44, 0.50)	-0.02 (-0.06, 0.01)	0.40 (0.37, 0.43)	0.10 (0.06, 0.14)	0.31 (0.28, 0.35)
Ethnicity							
White British	9703 (87)	-0.05 (-0.08, -0.03)	0.55 (0.52, 0.58)	-0.02 (-0.05, 0.01)	0.47 (0.45, 0.50)	0.11 (0.08, 0.14)	0.36 (0.34, 0.39)
Indian	298 (2)	0.16 (-0.04, 0.36)	-0.09 (-0.28, 0.10)	0.20 (0.01, 0.38)	0.00 (-0.23, 0.23)	0.30 (0.12, 0.49)	0.14 (-0.07, 0.34)
Pakistani	483 (3)	0.10 (0.00, 0.19)	0.12 (-0.02, 0.25)	0.09 (0.01, 0.17)	0.12 (-0.06, 0.29)	0.14 (0.04, 0.24)	0.04 (-0.12, 0.19)
Bangladeshi	171 (1)	-0.05 (-0.28, 0.18)	0.21 (-0.18, 0.60)	-0.02 (-0.27, 0.22)	0.23 (-0.04, 0.50)	-0.03 (-0.29, 0.23)	0.24 (-0.03, 0.51)
Black Caribbean	225 (2)	0.12 (-0.03, 0.28)	0.67 (0.46, 0.88)	0.23 (0.08, 0.38)	0.59 (0.39, 0.79)	0.35 (0.20, 0.51)	0.60 (0.40, 0.80)
Black African	215 (2)	0.80 (0.64, 0.95)	0.62 (0.41, 0.84)	0.80 (0.67, 0.93)	0.67 (0.48, 0.87)	0.85 (0.68, 1.02)	0.72 (0.50, 0.94)
Other	262 (2)	-0.14 (-0.29, 0.02)	0.26 (0.08, 0.43)	-0.09 (-0.26, 0.08)	0.19 (0.01, 0.37)	0.03(-0.13, 0.19)	0.09 (-0.11, 0.29)
Birth weight							
≥ 2500 g	10668 (94)	0.00 (-0.02, 0.03)	0.54 (0.51, 0.57)	0.03 (0.01, 0.06)	0.47 (0.44, 0.50)	0.15 (0.13, 0.18)	0.37 (0.34, 0.40)
< 2500 g	689 (6)	-0.50 (-0.60, -0.39)	0.10 (-0.02, 0.21)	-0.42 (-0.52, -0.32)	0.07 (-0.04, 0.17)	-0.26 (-0.36, -0.15)	0.05 (-0.05, 0.15)
Income (age 3)							
> 60% median	8625 (76)	0.00 (-0.03, 0.03)	0.53 (0.50, 0.56)				
≤ 60% median	2732 (24)	-0.12 (-0.17, -0.07)	0.48 (0.41, 0.55)				
Income (age 5)							
> 60% median	8724 (76)			0.04 (0.01, 0.07)	0.52 (0.50, 0.55)		
≤ 60% median	2633 (24)			-0.10 (-0.16, -0.04)	0.48 (0.41, 0.55)		
Income (age 7)							
> 60% median	8639 (77)					0.16 (0.13, 0.19)	0.53 (0.50, 0.56)
≤ 60% median	2718 (23)					0.01 (-0.04, 0.07)	0.47 (0.41, 0.53)

[1] unweighted sample size; weighted percentage

Figure 1. zHeight and zBMI growth trajectories for boys and girls in the UK Millennium Cohort Study (2003-2009)



zHeight and zBMI expressed in standard deviate scores derived using LMS standardisation (Cole, 1990)

Table 3. Correlations (standard errors) between baseline zHeight and zBMI [1] and changes over time for 5,717 boys in the MCS

	zHeight 3	zHeight 3-5	zHeight 5-7	zBMI 3	zBMI 3-5	zBMI 5-7
zHeight 3 years	1.00					
zHeight 3-5 years	-0.07 (0.09)	1.00				
zHeight 5-7 years	-0.06* (0.03)	-0.20 (0.14)	1.00			
zBMI 3 years	-0.00 (0.03)	0.40*** (0.07)	-0.06* (0.03)	1.00		
zBMI 3-5 years	0.24*** (0.04)	-0.66*** (0.13)	0.43*** (0.09)	-0.28* (0.14)	1.00	
zBMI 5-7 years	0.07 (0.04)	0.57*** (0.17)	-0.46*** (0.14)	-0.11** (0.04)	-0.16 (0.21)	1.00

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$

[1] zHeight and zBMI expressed in standard deviate scores derived using LMS standardisation (Cole, 1990)

Table 4. Correlations (standard errors) between baseline zHeight and zBMI [1] and changes over time for 5,640 girls in the MCS

	zHeight 3	zHeight 3-5	zHeight 5-7	zBMI 3	zBMI 3-5	zBMI 5-7
zHeight 3 years	1.00					
zHeight 3-5 years	0.70 (0.12)	1.00				
zHeight 5-7 years	-0.12*** (0.04)	-0.29* (0.15)	1.00			
zBMI 3 years	0.06** (0.03)	0.40*** (0.09)	-0.10** (0.04)	1.00		
zBMI 3-5 years	0.19*** (0.04)	-0.63*** (0.19)	0.61*** (0.20)	-0.29* (0.14)	1.00	
zBMI 5-7 years	0.17 (0.11)	0.65 (0.52)	-0.37 (0.33)	-0.04 (0.05)	-0.13 (0.20)	1.00

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$

[1] zHeight and zBMI expressed in standard deviate scores derived using LMS standardisation (Cole, 1990)

Relationship between height and BMI growth trajectories

There was a significant correlation between baseline zHeight and zBMI at age 3 for girls only, conditional on ethnicity, birth weight and household income. There was evidence linking zHeight to subsequent zBMI velocities: The correlation between zHeight at age three and zBMI velocity between ages three to five was 0.24 (SE = 0.04, $P < 0.0005$, Table 3) for boys and 0.19 (SE = 0.04, $P < 0.0005$, Table 4) for girls. The correlation between zHeight at age three and zBMI velocity between ages five to seven was weaker and of marginal significance for boys ($r = 0.07$, SE = 0.04; $P = 0.05$). For girls, the correlation was stronger but imprecisely estimated ($r = 0.17$, SE = 0.11).

Examining correlations between baseline BMI and height velocities, we find that higher zBMI at age three was positively correlated with increased zHeight gain from age three to five; children who were fatter at age three grew taller between ages three and five. The strength of the correlation between baseline zBMI and zHeight velocity was about twice that between baseline zHeight and zBMI velocity (boys: $r = 0.40$ versus 0.24; girls: $r = 0.40$ versus 0.19), a significant difference (boys: $\Delta r = 0.16$, $P = 0.02$; girls: $\Delta r = 0.22$, $P = 0.01$). However, the correlation reversed direction between ages five to seven, whereby children who were fatter at age three grew more slowly later.

Increased zHeight between ages three and five was correlated with increased zBMI in the next time period. Boys who grew taller more quickly between ages three and five put on more weight from ages five to seven. The relationship was stronger but imprecisely estimated for girls. There was also a positive correlation between zBMI velocity from three to five years and zHeight velocity from five to seven years (again stronger for girls), such that children who put on more weight between ages three to five, grew taller at a faster rate thereafter.

Analyses of the growth trajectories between height, waist and %fat present additional support for the interrelation between height and adiposity. Baseline zWaist was not related to zWaist velocity subsequently (Online supplementary tables S3 and S4). Results show that taller zHeight at age three was correlated with higher zWaist at age five and %fat measured at age seven in both boys and girls. There was weak evidence ($P < 0.10$) that higher zHeight velocity to age five was positively correlated with higher zWaist at age five in girls and that higher zHeight velocity from five to seven years was positively correlated with higher zWaist

velocity over the same period in boys. The evidence was stronger that zHeight velocities to age five were positively correlated with %fat at age seven (online supplementary tables S5 and S6).

Discussion

In a large representative sample of UK children, the relationship between zHeight and zBMI in childhood was dynamic and bidirectional. Our first hypothesis was partially supported by evidence showing zHeight at three years of age to be positively correlated with subsequent zBMI velocities and zBMI at three years positively correlated with zHeight velocity to age five. However, zBMI at three was inversely correlated with zHeight velocity from five to seven years of age. Consistent with our second hypothesis, the longitudinal relationship between zBMI and zHeight velocity from three to five years was stronger than that between zHeight and zBMI velocity over the same period. In support of the third hypothesis, zHeight velocity from three to five years was negatively correlated with zBMI velocity over the same age range and positively correlated with subsequent zBMI velocity, and vice versa for associations of zBMI velocity from three to five years with zHeight velocity from three to five years and from five to seven years.

Strengths of the study are the large representative sample and the use of objectively measured anthropometric data. Previous studies have cautioned against using parental reports of their child's height and weight as this may bias results (Brettschneider, Ellert & Schaffrath Rosario, 2012; Weden, et al., 2013). The present study has simultaneously modelled bidirectional longitudinal associations between zHeight and zBMI, overcoming some of the limitations of previous work that assumed a unidirectional relationship. Some weaknesses must be acknowledged. Data on the children were only taken at three time points, preventing us from modelling changes with a parametric non-linear function. More frequent measurements would have also allowed us to detect onsets of rebound or growth spurts. We made an assumption about the position of the knot for the piecewise linear models. For simplicity we modelled the knot at five years of age; however sensitivity analyses with alternative knot positions did not reveal any differences to our findings. We had no information on length at birth although birth weight was included as a covariate. BMI is the most commonly used measure of adiposity, yet its use in childhood has supporters and critics (Metcalf, et al., 2011) and it is influenced by other factors such as muscle mass; although a further strength of the study is that findings based on waist circumference and %fat corroborate those using zBMI. It is still possible that the findings might differ if growth curve models using other more specific measures of adiposity were estimated (Cole, Faith, Pietrobelli & Heo, 2005).

We found that, on average in the UK, young children born around the year 2000 were of similar stature to children of the same age used to derive the 1990 British Growth Standards, but they had much higher BMI's. Children in the MCS grew taller than the 1990 standards by age seven, while at the same time differences in BMI slightly narrowed. The secular increases in height and BMI shown here are similar to those from other European Union countries over the same period (Cardoso & Padez, 2008; Heude, et al., 2003). The present study did not address the question of changing trends. However increases in BMI over such a relatively short period are likely to be a consequence of the free energy available from simultaneous increases in children's energy intake together with decreases in energy expenditure (Anderson & Butcher, 2006). An alternative suggestion is provided by Buchan, Bundred, Kitchiner and Cole (2007). In their study of time trends in BMI and height, they note the relationship between tall stature and increases in BMI and hypothesise a role for appetite regulation while at the same time acknowledging that children did not become taller over their period of study.

Although we found baseline zHeight and zBMI to be uncorrelated, zHeight and zBMI were related cross-sectionally at later ages (not shown), similar to the findings of Rolland-Cachera Deheeger, Maillot, and Bellisle,(1982). Our results on the relationship of zHeight and zBMI with zHeight and zBMI velocities are also consistent with other research (Buchan, et al., 2007; Stanojevic, et al., 2007; Walker, Gaskin, Powell & Bennett, 2002) but we add new evidence of bidirectional associations between zHeight and zBMI and that zBMI predicts zHeight velocity more strongly than zHeight predicts zBMI velocity. Our results are also consistent with longitudinal relationships between height and BMI velocities reported previously (Dietz & Hartung, 1985; He, et al., 2001; Metcalf, et al.,

2011). Although the correlation between zHeight velocity between ages three and five years and zBMI velocity between ages five and seven years was greater than that between zBMI velocity between ages three and five years and zHeight velocity thereafter, the differences were not significant (boys: $\Delta r = 0.14$, $P = 0.23$; girls: $\Delta r = 0.04$, $P = 0.47$).

There was an unexpected inverse correlation of age three zBMI and zHeight velocity from five to seven years of age. Studies of the adiposity rebound may offer some insight into this finding. On average, adiposity increases in infancy and then declines followed by a rebound around the age of six years (Rolland-Cachera, Deheeger, Maillot & Bellisle, 2006). An earlier adiposity rebound is associated with a higher BMI before, during and after the rebound (Freedman, Khan, Serdula, Srinivasan & Berenson, 2001; Williams, 2005). This phenomenon can be observed in the MCS (see Table 1). If children who have a higher zBMI at age three have an earlier adiposity rebound, then it follows that they could be laying down fat from five to seven years. The free energy hypothesis would predict a resultant diversion from height growth during this phase of development.

There were two instances where the magnitude of correlations were larger for girls than boys but their precision was much weaker, suggesting diversity in girls' synergy between height and adiposity. Both instances involved relationships with zBMI velocity between ages five and seven years. The mechanisms underlying this finding are unknown and warrant further investigation.

These data are of relevance to clinicians concerned with growth or obesity risk in childhood. The evidence from a small clinical study is that height velocity can be slowed down by weight loss (Dietz & Hartung, 1985). Indeed, Dietz emphasised that obese children's height should be monitored carefully if placed on even a slightly restrictive diet. The interplay, or competition, between height growth and the laying down of adipose tissue that we have shown is considered to represent a physiologically adaptive trait that enables the body to choose between investing energy in growth or saving energy for storage (Ralt, 2007). This competition is observed even under the 'normal' average population level environmental conditions in our study and indicates that alternatives to severe calorie restriction might be advisable during some phases of childhood development.

The findings also have implications for health inequalities in later life. In general, household socioeconomic position (SEP) is inversely related to obesity in childhood (Shrewsbury & Wardle, 2008). Yet interactions between SEP and height have been observed such that the association between SEP and obesity is greater in taller children (Murasko, 2009). Our longitudinal analysis suggests how high calorie diets might bring about these interactions and contribute to greater social inequalities in obesity in adulthood. Disturbingly, 40-year trends in the US show that BMI and height increases have been greatest in children from more disadvantaged households (Murasko, 2011), and have led to a call for more research on the extent to which these growth-related processes in childhood underlie developmental origins of health disparities (Hanks, et al., 2013). If the trends are mirrored in the UK then we concur with previous suggestions that childhood height for age might help identify children who could become overweight adults (Freedman, Khan, Serdula, Srinivasan, & Berenson 2001; Navti, Samani-Radia & McCarthy, 2014), but add that targeting overweight taller children from more disadvantaged homes might prevent health gradients from becoming steeper.

Conclusions

This study provides evidence on the relationship between height and BMI in early childhood and suggests that clinicians should be made aware that an increase in height velocity, whether rapid catch-up in early childhood or disproportionate peripubertal growth, is a marker of an obesogenic environment that may be associated with contemporaneous or future unhealthy weight gain.

Acknowledgements

This work was supported by the United Kingdom Economic and Social Research Council grants ES/J019119/1; RES-177-25-0012 and ES/K001582/1. LB was also supported by a Hallsworth Research Fellowship.

The work is based on the United Kingdom Millennium Cohort Study (MCS), which is funded by the United Kingdom Economic and Social Research Council and a consortium of government departments. The original data creators, depositors or copyright holders, the funders of the MCS, and the UK Data Archive bear no responsibility for the accuracy or comprehensiveness of the analyses.

We thank Professor Russell Viner at the Institute of Child Health, University College London, for his helpful comments on the design of the study.

References

Anderson, P. M., & Butcher, K. F. (2006). Childhood obesity: trends and potential causes. *The Future of children, 16*(1), 19-45. http://dx.doi.org/10.1353/foc.2006.0001

Baird, J., Fisher, D., Lucas, P., Kleijnen, J., Roberts, H., & Law, C. (2005). Being big or growing fast: systematic review of size and growth in infancy and later obesity. *BMJ, 331*(7522), 929. http://dx.doi.org/10.1136/bmj.38586.411273.E0

Bosy-Westphal, A., Plachta-Danielzik, S., Doerhoefer, R.-P., & Mueller, M. J. (2009). Short stature and obesity: positive association in adults but inverse association in children and adolescents. *British Journal of Nutrition, 102*(3), 453-461. http://dx.doi.org/10.1017/S0007114508190304

Brettschneider, A.-K., Ellert, U., & Schaffrath Rosario, A. (2012). Comparison of BMI Derived from Parent-Reported Height and Weight with Measured Values: Results from the German KiGGS Study. *International Journal of Environmental Research and Public Health, 9*(2), 632-647. http://dx.doi.org/10.3390/ijerph9020632

Buchan, I. E., Bundred, P. E., Kitchiner, D. J., & Cole, T. J. (2007). Body mass index has risen more steeply in tall than in short 3-year olds: serial cross-sectional surveys 1988-2003. *International Journal of Obesity, 31*(1), 23-29. http://dx.doi.org/10.1038/sj.ijo.0803435

Cardoso, H. F. V., & Padez, C. (2008). Changes in height, weight, BMI and in the prevalence of obesity among 9-to 11-year-old affluent Portuguese schoolboys, between 1960 and 2000. *Annals of Human Biology, 35*(6), 624-638. http://dx.doi.org/10.1080/03014460802464200

Cecil, J. E., Tavendale, R., Watt, P., Hetherington, M. M., & Palmer, C. N. (2008). An obesity-associated FTO gene variant and increased energy intake in children. *New England Journal of Medicine, 359*(24), 2558-2566. http://dx.doi.org/10.1056/NEJMoa0803839

Cole, T. J. (1990). The LMS method for constructing normalized growth standards. *European Journal of Clinical Nutrition, 44*, 45-60.

Cole, T. J., Faith, M. S., Pietrobelli, A., & Heo, M. (2005). What is the best measure of adiposity change in growing children: BMI, BMI %, BMI z-score or BMI centile? *European Journal Clinical Nutrition, 59*(3), 419-425. http://dx.doi.org/10.1038/sj.ejcn.1602090

Dietz, W. H., Jr., & Hartung, R. (1985). Changes in height velocity of obese preadolescents during weight reduction. *American Journal of Diseases in Childhood, 139*(7), 705-707. http://dx.doi.org/10.1001/archpedi.1985.02140090067031

Elks, C. E., Loos, R. J., Sharp, S. J., Langenberg, C., Ring, S. M., Timpson, N. J., Ness, A. R., Davey Smith, G., Dunger, D. B., Wareham, N. J., & Ong, K. K. (2010). Genetic markers of adult obesity risk are associated with greater early infancy weight gain and growth. *PLoS Medicine, 7*(5), e1000284. http://dx.doi.org/10.1371/journal.pmed.1000284

Fischbein, S., & Pedersen, N. (1986). Multivariate analysis of genetic and environmental influences for longitudinal height and weight data. *Acta Geneticae Medicae et Gemellologiae, 36*(2), 171-180.

Forbes, G. B. (1977). Nutrition and growth. *The Journal of Pediatrics, 91*(1), 40-42. http://dx.doi.org/10.1016/S0022-3476(77)80440-X

Freedman, D. S., Khan, L. K., Serdula, M. K., Dietz, W. H., Srinivasan, S. R., & Berenson, G. S. (2004). Inter-relationships among childhood BMI, childhood height, and adult obesity: the Bogalusa Heart Study. *International Journal of Obesity, 28*(1), 10-16. http://dx.doi.org/10.1038/sj.ijo.0802544

Freedman, D. S., Khan, L. K., Serdula, M. K., Srinivasan, S. R., & Berenson, G. S. (2001). BMI rebound, childhood height and obesity among adults: the Bogalusa Heart Study. *International Journal of Obesity, 25*(4), 543-549. http://dx.doi.org/10.1038/sj.ijo.0801581

Hanks, L. J., Newton, A. L., & Casazza, K. (2013). Getting to the height of the matter: The relationship between stature and adiposity in pre-pubertal children. *Ethnicity & Disease, 23*(1), 71-76.

Hansen, K. (2012). Millennium Cohort Study First, Second, Third and Fourth Surveys: A Guide to the Datasets. (7th ed.). London: Centre for Longitudinal Studies, Institute of Education, University of London.

He, Q., He, J., & Karlberg. (2001). BMI in Childhood and Its Association with Height Gain, Timing of Puberty, and Final Height. *Pediatric Research, 49*(2), 244-251. http://dx.doi.org/10.1203/00006450-200102000-00019

Heude, B., Lafay, L., Borys, J. M., Thibult, N., Lommez, A., Romon, M., Ducimetiere, P., & Charles, M. A. (2003). Time trend in height, weight, and obesity prevalence in school children from Northern France, 1992-2000. *Diabetes & Metabolism, 29*(3), 235-240. http://dx.doi.org/10.1016/S1262-3636(07)70032-0

Hindmarsh, P., Smith, P. J., Brook, C. G. D., & Matthews, D. R. (1987). The relationship between height velocity and growth hormone secretion in short pre-pubertal children. *Clinical Endocrinology, 27*(5), 581-591. http://dx.doi.org/10.1111/j.1365-2265.1987.tb01188.x

Hochberg, Z. e. (2011). Developmental Plasticity in Child Growth and Maturatiom. [Review]. *Frontiers in Endocrinology, 2*.

Johnson, W., Stovitz, S. D., Choh, A. C., Czerwinski, S. A., Towne, B., & Demerath, E. W. (2012). Patterns of linear growth and skeletal maturation from birth to 18 years of age in overweight young adults. *International Journal of Obesity (Lond), 36*(4), 535-541. http://dx.doi.org/10.1038/ijo.2011.238

Kendal, M., & Stuart, A. (1977). *The advanced theory of statistics* (4th ed. Vol. Vol.1, Distribution theory). London: Griffin.

Lloyd, J. K., Wolff, O. H., & Whelen, W. S. (1961). Childhood Obesity: A Long-Term Study Of Height And Weight. *The British Medical Journal, 2*(5245), 145-148. http://dx.doi.org/10.1136/bmj.2.5245.145

Metcalf, B. S., Hosking, J., Fremeaux, A. E., Jeffery, A. N., Voss, L. D., & Wilkin, T. J. (2011). BMI was right all along: taller children really are fatter (implications of making childhood BMI independent of height) EarlyBird 48. *International Journal Obesity (Lond), 35*(4), 541-547. http://dx.doi.org/10.1038/ijo.2010.258

Murasko, J. E. (2009). Socioeconomic status, height, and obesity in children. *Economics & Human Biology, 7*(3), 376-386. http://dx.doi.org/10.1016/j.ehb.2009.04.004

Murasko, J. E. (2011). Trends in the associations between family income, height and body mass index in US children and adolescents: 1971-1980 and 1999-2008. *Annals of Human Biology, 38*(3), 290-306. http://dx.doi.org/10.3109/03014460.2010.537698

Must, A., Jacques, P. F., Dallal, G. E., Bajema, C. J., & Dietz, W. H. (1992). Long-term morbidity and mortality of overweight adolescents: a follow-up of the Harvard Growth Study of 1922 to 1935. *New England journal of medicine, 327*(19), 1350-1355. http://dx.doi.org/10.1056/NEJM199211053271904

Navti, L. K., Samani-Radia, D., & McCarthy, H. D. (2014). Children's body fatness and prevalence of obesity in relation to height for age. *Annals of Human Biology, 41*(1), 84-90. http://dx.doi.org/10.3109/03014460.2013.856474

Pearce, Laura R., Atanassova, N., Banton, Matthew C., Bottomley, B., van der Klaauw, Agatha A., Revelli, J.-P., Hendricks, A., Keogh, Julia M., Henning, E., Doree, D., Jeter-Jones, S., Garg, S., Bochukova, Elena G., Bounds, R., Ashford, S., Gayton, E., Hindmarsh, Peter C., Shield, Julian P. H., Crowne, E., Barford, D., Wareham, Nick J., O'Rahilly, S., Murphy, Michael P., Powell, David R., Barroso, I., & Farooqi, I. S. (2013). KSR2 Mutations Are Associated with Obesity, Insulin Resistance, and Impaired Cellular Fuel Oxidation. *Cell, 155*(4), 765-777. http://dx.doi.org/10.1016/j.cell.2013.09.058

Ralt, D. (2007). Low muscle mass - Tall and obese children - A special genre of obesity. *Medical Hypotheses, 68*(4), 750-755. http://dx.doi.org/10.1016/j.mehy.2006.05.071

Reilly, J. J., & Kelly, J. (2011). Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: systematic review. *International Journal of Obesity, 35*(7), 891-898. http://dx.doi.org/10.1038/ijo.2010.222

Rolland-Cachera, M. F., Deheeger, M., Maillot, M., & Bellisle, F. (2006). Early adiposity rebound: causes and consequences for obesity in children and adults. *International Journal of Obesity, 30*(S4), S11-S17. http://dx.doi.org/10.1038/sj.ijo.0803514

Rolland-Cachera, M. F., Sempé, M., Guilloud-Bataille, M., Patois, E., Péquignot-Guggenbuhl, F., & Fautrad, V. (1982). Adiposity indices in children. *The American Journal of Clinical Nutrition, 36*(1), 178-184.

Sacker, A., & Kelly, Y. J. (2012). Ethnic differences in growth in early childhood: an investigation of two potential mechanisms. *The European Journal of Public Health, 22*(2), 197-203. http://dx.doi.org/10.1093/eurpub/ckr032

Samani-Radia, D., & McCarthy, H. D. (2011). Comparison of children's body fatness between two contrasting income groups: contribution of height difference. *International Journal of Obesity, 35*(1), 128-133. http://dx.doi.org/10.1038/ijo.2010.116

Saxena, S., Ambler, G., Cole, T. J., & Majeed, A. (2004). Ethnic group differences in overweight and obese children and young people in England: cross sectional survey. *Archives of Disease in Childhood, 89*(1), 30-36.

Serdula, M. K., Ivery, D., Coates, R. J., Freedman, D. S., Williamson, D. F., & Byers, T. (1993). Do obese children become obese adults? A review of the literature. *Preventative Medicine, 22*(2), 167-177. http://dx.doi.org/10.1006/pmed.1993.1014

Shrewsbury, V., & Wardle, J. (2008). Socioeconomic Status and Adiposity in Childhood: A Systematic Review of Cross-sectional Studies 1990–2005. *Obesity, 16*(2), 275-284. http://dx.doi.org/10.1038/oby.2007.35

Stanojevic, S., Kain, J., & Uauy, R. (2007). The association between changes in height and obesity in Chilean preschool children: 1996-2004. *Obesity, 15*(4), 1012-1022. http://dx.doi.org/10.1038/oby.2007.611

Walker, S. P., Gaskin, P. S., Powell, C. A., & Bennett, F. I. (2002). The effects of birth weight and postnatal linear growth retardation on body mass index, fatness and fat distribution in mid and late childhood. *Public Health Nutrition, 5*(3), 391-396. http://dx.doi.org/10.1079/PHN2002275

Walton, S., Bedford, H., & Dezateux, C. (2006). Use of personal child health records in the UK: findings from the millennium cohort study. *BMJ, 332*(7536), 269-270. http://dx.doi.org/10.1136/bmj.332.7536.269

Wardle, J., Carnell, S., Haworth, C. M. A., Farooqi, I. S., O'Rahilly, S., & Plomin, R. (2008). Obesity Associated Genetic Variation in FTO Is Associated with Diminished Satiety. *The Journal of Clinical Endocrinology & Metabolism, 93*(9), 3640-3643. http://dx.doi.org/10.1210/jc.2008-0472

Weden, M. M., Brownell, P. B., Rendall, M. S., Lau, C., Fernandes, M., & Nazarov, Z. (2013). Parent-Reported Height and Weight as Sources of Bias in Survey Estimates of Childhood Obesity. *American Journal of Epidemiology*. First published online June 19th, 2013. http://dx.doi.org/10.1093/aje/kws477

Wells, J. C. (2012). The evolution of human adiposity and obesity: where did it all go wrong? *Disease Models & Mechanisms, 5*(5), 595-607. http://dx.doi.org/10.1242/dmm.009613

Williams, S. M. (2005). Weight and height growth rate and the timing of adiposity rebound. *Obesity Research, 13*(6), 1123-1130. http://dx.doi.org/10.1038/oby.2005.131

Wolff, O. H. (1955). Obesity in Childhood. *Quarterly Journal of Medicine, 24*, 109.

Handling attrition and non-response in longitudinal data with an application to a study of Australian youth

Joy Cumming Australian Catholic University
Joy.cumming@acu.edu.au
Harvey Goldstein Australian Catholic University and University of Bristol, UK

(Received February 2015 Revised September 2015) http://dx.doi.org/10.14301/llcs.v7i1.342

Abstract

A standard concern with long term longitudinal studies is that of attrition over time. Together with initial non-response this typically leads to biased model estimates unless a suitable form of adjustment is carried out. The standard approach to this has been to compute weights based upon the propensity to respond and to drop out and then carry out weighted analyses to compensate for response bias. In the present paper we argue that this approach is statistically inefficient, because it drops incomplete data records, is inflexible, and in practice gives rise to undue complexity involving a proliferation of weighting systems for different analyses. Instead we set out an alternative approach that makes use of recently developed imputation procedures for handling missing data and show how this can be used to improve the quality of the statistical analysis. An example analysis is given using the Longitudinal Study of Australian Youth (LSAY).

Keywords

Longitudinal data, life course, further education, attrition, missing data, imputation, weighting

Introduction

Attrition in longitudinal studies is typically viewed as a serious problem for two reasons. First, the loss of individuals over time will often result in a sample size, after a few occasions or 'sweeps', very much smaller than the initial sample size. For those analyses that utilise data at more than one occasion, the use of only those individuals with data at all such occasions in the analysis will result in a loss of efficiency. We use the term 'attrition' to mean any pattern of loss of individual records over time, including those cases where individuals may return to a study after missing measurement occasions.

Secondly, loss may not occur at random so that the remaining sample may be biased with respect to the variables being analysed. In longitudinal studies, at any given occasion the characteristics of subsequent losses will be known and these can be compared with those who are followed up. If biases are detected then suitable weights can be introduced to compensate for this, and this is the traditional approach to dealing with attrition.

The present paper sets out a general model-based approach to dealing with attrition in longitudinal studies. It does this by embedding the problem within a general approach to handling missing data and the procedure will, in principle, handle both the loss of individual records over time and the loss of individual data items, as well as initial non-response at wave 1. Further, this approach provides efficient estimates based upon a one-pass Markov Chain Monte Carlo (MCMC) algorithm that avoids the creation of multiply imputed data sets.

Data

These procedures are applied to a long term study the "Longitudinal Study of Australian Youth' (LSAY) with up to 12 waves of data collection. This is a study that is tracking the pathways of young Australians as they move from school to further study, work and other destinations. Data are collected on variables related to education, training, work, financial matters, health, social activities and attitudes. LSAY started in 1995 by sampling year nine students, average age 14.5 years, in Australian secondary schools and following them up every year on a further 11 occasions (LSAY, 2013). In the present paper we analyse the second cohort commencing in 1998 using data up to wave 6 in 2003. The principal aim of the paper is to explore ways in which the richness of the dataset can be utilised efficiently, in the light of extensive non-random sample attrition over time. We concentrate on a single outcome, whether or not the respondent is in part time or full time study and relate this to a number of characteristics. A series of reports by Sheldon Rothman presents basic tabulations for outcomes up to wave 6 (Rothman, 2005).

Australia has six states and two territories with very unequal population sizes. The sample design used in LSAY is one where there was oversampling from some smaller states and territories leading to unequal student selection probabilities (table 1). Thus a set of sample design weights was derived so that inferences could be made to the actual Australian population. For every child in a school the weights are equal so that we can treat this as a single level weight in any weighted analyses. In addition the study provides weights that attempt to control for non-random attrition. A detailed discussion of how to derive such weights can be found in Plewis (2007).

Table 1 shows the characteristics of the sample, including the distribution by state. The initial sample consists of 296 schools.

Table 1: Characteristics of 1998 LSAY cohort

		Unweighted sample	All Australian schools
Average age of respondents 30 /6/1998 (yrs)		14.5	
Total no of respondents		14117	
State/territory (%)	New South Wales	24.0	32.8
	Victoria	20.9	23.5
	Queensland	22.0	20.1
	South Australia	8.8	7.8
	Western Australia	12.0	10.8
	Tasmania	5.1	2.7
	Northern Territory	3.3	0.9
	Australian Capital Territory	4.0	1.9
Sex (%)	Male	51.2	
	Female	48.2	
	Unknown	0.6	
Indigenous status	Indigenous	3.1	
(%)	Non-Indigenous	96.9	
Geographic region	Metropolitan	60.0	
(%)	Regional	22.4	
	Rural and remote	17.5	
Country of birth (%)	Australia	85.0	
	Other	15.0	
School sector (%)	Government	63.0	
	Catholic	22.1	
	Independent	14.9	

Source: LSAY - Longitudinal Surveys of Australian Youth, Y98 cohort to 2009, released April 2010, updated January 2011.

Since the corresponding sample weights apply at the state level, we can effectively eliminate the need for weights by explicitly fitting state (as a series of dummy variables) in our models. Only if we wish to provide country level estimates will we then need to marginalise over the state distributions, but we do not consider this at the present stage. In fact, the use of weights does not change any of our inferences very much. Ignoring weights and not fitting effects for state will provide inferences for a (hypothetical) population where the state population sizes are proportional to the chosen state sample sizes.

In all our subsequent models we have also studied whether there are interactions among the explanatory (predictor) variables and found little evidence for these, so that they will be omitted.

Table 2. Percentages of original sample of 14117 year 9 students in 1998 remaining at waves 2-6

Year (wave)	Per cent remaining
1999 (2)	65.8
2000 (3)	67.6
2001 (4)	62.2
2002 (5)	55.0
2006 (6)	48.9

As table 2 shows the attrition is considerable, especially in the first year of the study with just 49% remaining by wave 6, and it is clearly necessary to make adjustments for this. We note that attrition is not completely monotonic with some students returning to the study. This will not affect the estimation model.

The LSAY user guide (LSAY, 2013) also discusses deriving weights to compensate for non-random attrition over time in the cohort. Weighting, however, is not entirely satisfactory (Goldstein, 2009) since it will in general require a different set of weights to be computed for every different combination of waves entering any given (longitudinal) model and this is not practically very feasible. What LSAY itself provides is essentially only weights that correct the data at each wave separately. Thus these will allow adjustment for cross sectional analyses at each wave but not for longitudinal analyses. In any case these weights are only computed based on overall achievement (average of student outcomes on tests of mathematics and reading comprehension) and gender at wave 1. Plewis (2007) proposes an extension of this by computing a set of weights specific for each of a number of longitudinal analyses involving different combinations of occasions or waves and different sets of variables. Such approaches are intended to deal with the problem of biases that may arise from differential attrition. In the present paper we are additionally concerned with efficiency, and in particular the loss of efficiency that is implicit in such weighting procedures, as we explain below.

Modelling the probability of participation in further and higher education

As noted, our principal aim in this article is methodological, namely to explore an alternative approach to weighting to improve efficiency. A secondary aim is to explore factors that predict the probability of participating in further or higher education after school. To address the methodological issue, in the present analysis we are only considering two time points, so that we can use weights based upon the probability of remaining in the sample at wave 6 as described below. Inspection of the data shows that other variables are associated with attrition, as shown in table 3.

Table 3. Variables predicting the probability of remaining in the sample at wave 6. Two level model. Reference categories in brackets. Probit link function. 9027 out of 14117 cases used.

Parameter	Estimate	Standard error
Intercept	-0.617	0.050
Female (male)	0.111	0.030
Catholic (Government) school	0.179	0.050
Independent (Government) school	-0.019	0.059
Maths score year 9	0.023	0.004
Reading score year 9	0.030	0.004
Non-Australia country of birth of mother (Australia)	-0.116	0.031
SES ANU3 score father	0.0022	0.0007
Level 2 variance	0.060	0.009

We see that additionally to the test scores and gender, country of birth of mother, type of school and father's socioeconomic status (SES) are predictors of attrition. We shall be using these variables in subsequent analyses either fitting into the model or using in the imputation model to correct for attrition bias. Adding some other terms in this model such as home language (English/other) does not add to the prediction so we will use the above variables as standard.

One way to utilise the results in table 3 is to use the inverse predicted probabilities of inclusion as weights when modelling wave 6 outcomes in order to correct for any attrition bias. A problem with such an analysis is that it is based only upon the students who remain at wave 6 and hence does not increase efficiency, especially as the predictors in table 3 are also those used in our model of interest given in table 4.

The methodological approach we use as an alternative to weighting is a recently proposed extension of multiple imputation (Goldstein, Carpenter, & Browne, 2014). In longitudinal data Goldstein (2009) discusses procedures for handling attrition and item missing data in longitudinal studies and contrasts weighting with multiple imputation. Seaman and White (2014) discuss the use of inverse probability weighting to adjust for biases that may arise when values are missing not at random, where the weights are derived from a model that predicts the probability of an individual having no missing values in the model of interest. Both these latter two papers point out that the weighted analysis uses only individuals with complete cases. For this reason, apart from issues of bias, imputation is generally recognised to be more efficient and the statistically most satisfactory method for handling attrition and in fact any type of 'missing' data (Moodie, Delany, Lefevre, & Platt, 2008). Carpenter and Kenward (2012) also discuss this and in particular consider the question of bias reduction and increasing efficiency using a combination of multiple imputation and inverse probability weighting, doubly robust estimation, although in the present case there is little to be gained since the prediction of the weights utilises the same variables as are in the model of interest.

In the present paper we demonstrate the increase in efficiency from using imputation. A particular advantage of imputation is that, in addition to attrition, nonresponses to individual questions can also be incorporated straightforwardly (see Goldstein, 2009 for a discussion). In our final analyses we will compare imputation with a complete cases only model, with and without weighting. We shall be utilising a

recent development in imputation that obviates the need for multiply imputed datasets and allows quite general models with missing data, as described below. Software for this has been developed by the Centre for Multilevel Modelling in Bristol and specifically the STATJR software package (STATJR, 2015), Alternative imputation methods, notably 'chained equation' procedures, are based upon multiple imputed datasets and are unable properly to handle functions of variables having missing data, such as power and interaction terms (Goldstein et al., 2014).

The imputation algorithm

The method described by Goldstein et al. (2014) extends the standard joint model for multiple imputation procedure (Carpenter & Kenward, 2013) by obviating the need to produce a set of multiply imputed datasets and also allows interaction and polynomial terms in the model of interest. It is a one-pass method using a single MCMC algorithm and is fully Bayesian with a faster implementation in software. The following is a simple summary of the procedure, avoiding undue technicality.

For simplicity consider a single level model of interest where the response and explanatory variables have a joint normal distribution. If there were no missing data then this can be readily fitted using standard multiple regression, either via maximum likelihood or in a Bayesian model, for example with default diffuse prior distributions. Consider the simple regression, joint model

$$Y = \beta_0 + \beta_1 X + e_{Y|X}$$
$$X = \alpha_0 + e_X \qquad (1)$$

The first line of (1) is the usual regression model and the second line specifies a model for the explanatory variable *X*. The residual terms express the usual conditional distribution $Y|X$ and the distribution for *X* itself. Note that, unlike in standard linear regression where it is not needed, we have explicitly introduced a distribution for *X*, since this will be required when we have missing data in this predictor. Where there are missing values in the response the record is omitted, although where there are several responses in a multivariate model we would impute missing responses where other responses are present. It is assumed that $e_X, e_{Y|X}$ are independent.

We initiate a MCMC algorithm, with suitable starting values, that at each iteration uses a 'metropolis' step, to propose, for each missing value in *X* (or *Y* in the multivariate case), a value, which is used in (1) to update both lines if accepted using the implied likelihood values. Suitable starting values can be derived from the complete cases. For each missing value, in turn, the Metropolis step will accept a proposed value based upon a comparison of the joint likelihood for *X* and *Y* based respectively on the current and proposed value. Where there are several *X* variables, the second line of (1) will express a conditional relationship among the *X* variables for each one in turn.

Model (1), with several predictors, is readily generalised to several explanatory variables and to further hierarchical levels or cross classifications. If there are interactions or polynomial terms in the model of interest these are only present in the first line of (1) and so are easily incorporated. Where we have non-normally distributed variables we adopt a 'latent normal' transformation that introduces additional steps in the algorithm to sample from underlying normal distributions: for a binary variable this is the usual probit model, and is used in our models. Note that the requirement for compatibility (congeniality) of the imputation model and the model of interest in standard multiple imputation is automatically satisfied using this algorithm. One of the assumptions made in imputation models is that, conditionally on the variables included in the imputation component, any propensity to missingness is effectively random, the so called Missing at Random (MAR) assumption. We may also include auxiliary variables, not required in the model of interest, in the imputation component of the joint model where these are needed to ensure MAR. Full details are given by Goldstein et al. (2014), including choice of prior distributions.

Our subsequent analyses, therefore, include all the variables from table 3 associated with attrition.

Predicting education and training participation at wave 6

Our model of interest is a comparison between those in tertiary education currently studying part time or full time, and those not.

The model of interest is

$$g(\pi_{ij}) = X_{ij}\beta + u_j$$
$$\pi_{ij} = \int_{-\infty}^{X_{ij}\beta} \phi(t)dt$$
$$u_j \sim N(0, \sigma_u^2) \qquad (2)$$

where π_{ij} is the probability that the *i*th student in the *j*th (1998) school is in tertiary education, and $\phi(t)$ is the standard normal distribution with g the probit link function, relating this probability to the set of covariates listed in table 4. The subscripts i, j index students and schools respectively. The probit function rather than the logit is convenient for modelling with missing data and allows interpretation of parameters on an underlying standard normal (N(0,1)) scale.

The imputation model can be written, excluding subscripts, as

$$X^* \sim MVN(\alpha Z, \Omega_X) \qquad (3)$$

This is a two level multivariate normal model where the responses are the predictors, apart from the intercept and school type, *Z*, where there are no missing values, in table 4. For the maths and reading scores, these are already normalised. For the remaining binary variables the X^* are obtained using a step in the algorithm that randomly samples from the underlying 'latent' normal distribution corresponding to the binary variable. The joint model is thus the combination of (2) and (3).

We have explored a number of analyses with different predictors and table 4 presents a final fitted model that contains a set of predictor variables that jointly predict propensity to remain studying. Gender is included for completeness. We looked at student's country of birth, father's country of birth and also student location in urban/semi urban/rural region and these had small effects and have been omitted. The continuous ANU3 score is used for SES status as recommended by Marks (1999). In addition to variables shown in table 3 on probability of remaining in the sample at wave 6, two additional variables, Home language not English (English), and mother's SES, are in the model of interest.

Table 4. Full or part time study in 2003 by student, parental, environmental and school characteristics in year 9, 1998. MCMC estimates with burnin=5,000, iterations=25,000. Listwise deletion with 3407 out of 14117 cases used. Two level model. Reference categories in brackets. Single level estimates in brackets.

Parameter	Estimate	Standard error
Intercept	-0.938 (-0.952)	0.098 (0.088)
Female (male)	0.052 (0.053)	0.047 (0.046)
Catholic (Government) school	0.182 (0.174)	0.062 (0.052)
Independent (Government) school	0.236 (0.222)	0.080 (0.070)
Maths score year 9	0.049 (0.048)	0.006 (0.006)
Reading score year 9	0.024 (0.023)	0.006 (0.006)
Non-Australia country of birth of mother (Australia)	0.185 (0.181)	0.059 (0.058)
Home language not English (English)	0.412 (0.408)	0.119 (0.115)
SES ANU3 score father	0.0049 (0.0041)	0.0011 (0.0011)
SES ANU3 score mother	0.0025 (0.0027)	0.0014 (0.0013)
Level 2 variance	0.034	0.016

The results in the above model are not unexpected. Country of birth of mother outside Australia is associated with a greater propensity to remain studying and this could be explored further in terms of actual country of origin.

Being in Catholic or independent schools likewise is positively associated with increased propensity to remain studying, as is having a main language in the home that is not English, and having high maternal and paternal occupational status.

We see that the school level variance is 0.034 with an equivalent standard deviation of 0.18. This is on a standard normal scale and is relatively small. Omitting the school level we have also fitted a single level model whose estimates are given in brackets and it can be seen that the inferences are almost identical. Thus the actual school attended in 1998 appears to have little effect on propensity to be studying five years later. The overall percentage studying is 61% and a variance component analysis, just fitting an intercept term in the model gives us an estimate for the between school variance (standard deviation) of 0.121 (0.35) so we see that most of the school effect is accounted for by the predictor variables.

The main methodological problem with this analysis is that the effective sample size is only 3407 out of a possible 14117 (24%) of all cases. In particular 33% of father's and 45% of mother's SES are missing (table 5).

Table 5. Percentage of missing values for selected variables

Studying full or part time at wave 6	51.1
Sex	0.6
School type	0
Maths score year 9	2.3
Reading score year 9	2.5
Country of birth of mother	4.7
Mother's SES ANU3 score	44.7
Father's SES ANU3 score	33.5

Cases with any missing data are omitted from the analysis. Thus, even if we had appropriate weights that corrected for any biases we would still have a very reduced efficiency due to the fact that only 24% of the sample cases can be used.

We demonstrate this in table 6 by repeating the analysis in table 4 with one that uses the inverse probability weights derived from table 3 where we predict the probability of remaining in the sample until wave 6. Bayesian models do not allow us to incorporate weights so that the estimates are second order quasi-likelihood estimates (Goldstein, 2011) which do approximate very closely, in the unweighted case, to the Bayesian MCMC estimates.

Table 6 Full or part time study in 2003 by student, parental, environmental and school characteristics in year 9, 1998. Second order PQL (Quasi-likelihood) estimates. Listwise deletion with 3,407 out of 14,117 cases used. Two level model. Reference categories in brackets. Standard errors in brackets

Parameter	Unweighted estimates	Inverse probability weighted estimates
Intercept	-0.942(0.093)	-0.927(0.095)
Female (male)	0.054(0.047)	0.040(0.047)
Catholic (Government) school	0.184(0.057)	0.209(0.057)
Independent (Government) school	0.232(0.090)	0.230(0.090)
Maths score Year 9	0.050(0.006)	0.050(0.006)
Reading score Year 9	0.024(0.006)	0.024(0.006)
Non-Australia country of birth of mother (Australia)	0.184(0.060)	0.177(0.061)
Home language not English (English)	0.412(0.120)	0.409(0.121)
SES ANU3 score father	0.0050(0.0011)	0.0048(0.0011)
SES ANU3 score mother	0.0026(0.0013)	0.0025(0.0013)
Level 2 variance	0.034(0.014)	0.036(0.014)

We see that the weighted and unweighted point estimates are very similar, with virtually identical standard errors.

In the next model, in table 7, we have used our imputation procedure to both adjust for bias and utilise all the information in a statistically efficient manner.

Incorporating missing data using imputation

Out of the total sample, 51% are not present at wave 6 and hence missing the response in the model of interest, 'in full or part time study', which is available for all those remaining in the study. In the present case, even though we fit only the sample of 6901 who remain at wave 6, we do actually obtain results where the parameter estimates and standard errors are essentially the same as fitting the full data, that is, imputing responses for those not present at wave 6. This is to be expected since where the response, in a model with covariates, is missing, there is no further information available from such records.

Table 7. Full or part time study in 2003 by student, parental, environmental and school characteristics in year 9, 1998. Missing data model. MCMC estimates with burnin=500, iterations=2,500. Two level model. Reference categories in brackets. Single level estimates in brackets. Sample size=6,901.

Parameter	Estimate	Standard error
Intercept	-1.040 (-0.951)	0.065 (0.057)
Female (male)	0.058 (0.047)	0.034 (0.032)
Catholic (Government) school	0.210 (0.159)	0.047 (0.040)
Independent (Government) school	0.255 (0.214)	0.057 (0.049)
Maths score year 9	0.049 (0.051)	0.004 (0.004)
Reading score year 9	0.028 (0.026)	0.004 (0.004)
Non-Australia country of birth of mother (Australia)	0.179 (0.186)	0.040 (0.040)
Home language not English (English)	0.457 (0.497)	0.089 (0.072)
SES ANU3 score father	0.0046 (0.0045)	0.0009 (0.0009)
SES ANU3 score mother	0.0025 (0.0018)	0.0010 (0.0006)
Level 2 variance	0.036	0.009

While there are some small differences in the parameters estimates, notably for school type, the reading score, home language and father's SES, the main difference lies in the considerable reduction in standard errors. These reductions are of an order up to about 50%, reflecting the efficiency gain from the imputation-based modelling. Fitting the imputation model is not onerous, the two level model took approximately 80 minutes to fit, on a 2.4 Ghz PC running windows 7.

Discussion

The Longitudinal Study of Australian Youth represents a major investment in data collection over a 12 year period to track the fortunes of students from school year 9 annually into early adulthood. In the present paper we have explored a very limited set of variables looking at antecedents of whether in the years after leaving school, the students are still in full or part time education. We find that test scores, parental country of birth, home language and SES all affect the propensity to be studying.

The particular focus of the paper, however, lies in its methodology. By wave 6 half the students who were sampled originally in wave 1 had left the study, and the propensity to leave was not random. We have shown how the use of complete cases, whether using inverse probability weights or not, results in estimates that are considerably less efficient than a fully imputation based approach. In the present case bias appears not to be an issue since neither the weighted analysis nor the imputation analysis lead to very different estimates from the unweighted complete cases analysis.

More generally, our analysis illustrates the usefulness of a missing data approach to both missing data item values and dropout. When there are a relatively small number of complete cases it is not efficient to base an analysis solely on such cases. Utilising weights in a complete cases analysis may provide a means of correcting potential biases, but where such weights, as in the case of attrition,

are derived from observed data at wave 1 (or later) we show that greater simplicity and greater statistical efficiency can be obtained by an imputation based approach that incorporates all the observed data in a single model.

The assumption of missingness at random is an important one. To deal with non-random attrition in a weighting approach we would normally seek to satisfy this by incorporating 'auxiliary' variables when modelling the propensity. In an imputation approach we would incorporate such auxiliary variables in the imputation component of the model. In our analysis all the variables that are significant predictors of dropout by wave six are also in the model of interest.

In some longitudinal studies the data analyst may be confronted with data that contain design or non-response weights of unknown provenance. In such cases, unlike the present, these weights will generally need to be incorporated into both the imputation component and that for the model of interest. Within a fully Bayesian framework it is not clear how to do this, and this problem is currently the focus of further research. Carpenter and Kenward (2012) provide some guidance.

Acknowledgements

We are most grateful to referees for their helpful comments.

References

Carpenter, J., & Kenward, M. (2012). *Multiple imputation and its application*. Chichester: John Wiley & Sons.

Carpenter, J., Goldstein, H., & Kenward, M. (2011). REALCOM-IMPUTE software for multilevel multiple imputation with mixed response types. *J. Statistical Software*, *45*(5), 1–14. http://dx.doi.org/10.18637/jss.v045.i05

Goldstein (2009). Handling attrition and non-response in longitudinal data. *International Journal of Longitudinal and Life Course Studies, 1,* 63–72.

Goldstein, H. (2011). Multilevel statistical models (Fourth ed.). Chichester, England: Wiley.

Goldstein, H., Carpenter, J. R. and Browne, W. J. (2014). Fitting multilevel multivariate models with missing data in responses and covariates that may include interactions and non-linear terms. *Journal of the Royal Statistical Society: Series A* (Statistics in Society). http://dx.doi.org/10.1111/rssa.12022

Goldstein, H., Carpenter, J., Kenward, M., & Levin, K. (2009). Multilevel models with multivariate mixed response types. *Statistical Modelling*, *9*(3), 173–197. http://dx.doi.org/10.1177/1471082X0800900301

LSAY. (2013). *Longitudinal Surveys of Australian Youth.* 1998 cohort user guide. Accessed 12th February 2015 from http://www.lsay.edu.au/publications/2199.html

Marks, G. (1999). *The measurement of socioeconomic status and social class in the LSAY project [Technical Report #14].* Melbourne: ACER.

Moodie, E.F.M., Delany, J.A.C., Lefevre, G., & Platt, R.E. (2008). Missing confounding data in marginal structural models: a comparison of inverse probability weighting and multiple imputation. *International Journal of Biostatistics*, *4*, 1–23. http://dx.doi.org/10.2202/1557-4679.1106

Plewis, I. (2007). Non-response in a birth cohort study: the case of the Millennium Cohort Study. *International Journal of Social Research Methodology*, *10*, 325–334. http://dx.doi.org/10.1080/13645570701676955

Rothman, S. (2005). Accessed 12th February 2015 from http://www.lsay.edu.au/publications/1844.html

Seaman, S., & White, I. (2014). Inverse probability weighting with missing predictors of treatment assignment or missingness. *Communications in Statistics—Theory and Methods*, *43*, 3499–3515. http://dx.doi.org/10.1080/03610926.2012.700371

STATJR. (2015). Centre for Multilevel Modelling, University of Bristol. Accessed 12th February 2015 from http://www.bristol.ac.uk/cmm/software/statjr/

STUDY PROFILE

The Swiss Household Panel Study: Observing social change since 1999

Robin Tillmann Swiss Centre of Expertise in the Social Sciences FORS, Switzerland
robin.tillmann@fors.unil.ch
Marieke Voorpostel Swiss Centre of Expertise in the Social Sciences FORS, Switzerland
Erika Antal Swiss Centre of Expertise in the Social Sciences FORS, Switzerland
Ursina Kuhn Swiss Centre of Expertise in the Social Sciences FORS, Switzerland
Florence Lebert Swiss Centre of Expertise in the Social Sciences FORS, Switzerland
Valérie-Anne Ryser Swiss Centre of Expertise in the Social Sciences FORS, Switzerland
Oliver Lipps Swiss Centre of Expertise in the Social Sciences FORS, Switzerland
Boris Wernli Swiss Centre of Expertise in the Social Sciences FORS, Switzerland

(Received July 2015 Revised September 2015) http://dx.doi.org/10.14301/llcs.v7i1.360

Abstract

Collecting data on households and individuals since 1999, the Swiss Household Panel (SHP) is an ongoing, unique, large-scale, nationally representative, longitudinal study in Switzerland (N=7,383 households and N=12,119 persons interviewed in 2014). The data of the SHP provide a rich source of information to study social change in Switzerland over a significant period on a wide variety of topics. The SHP aims to provide both continuity and innovation in measurement and data collection, with the combination of retrospective and prospective longitudinal data in the most recent refreshment sample as one notable example of such an innovation. This paper provides an overview of the SHP – focusing on its origin, aims, design, content, data collection and adjustments, possibilities for cross-national comparisons, data use and accomplishments.

Keywords

Swiss Household Panel, household panel survey, design, longitudinal data

Introduction

The Swiss Household Panel (SHP)[1] is a study of major importance in the Swiss social survey landscape. Collecting data on households and individuals since 1999, the SHP is a unique longitudinal study that allows for the analysis of mid- to long-term micro-social change on a wide variety of topics. Together with a few other nationally representative household panels from other nations, the SHP provides unique analytical opportunities to researchers in the social sciences. The SHP combines continuity with innovation. The majority of questions are asked annually, allowing for longitudinal analysis over a period now spanning 16 years. Additional research instruments, such as biographical questionnaires, provide an even longer-term context.

This paper provides an overview of the SHP. We focus first on its origin and aims. Next, we describe the SHP's design, questionnaire content, data

collection, and data adjustments. Finally, we discuss possibilities for cross-national comparisons, data use, and accomplishments.

Origin and aims

The Swiss Priority Program "Switzerland Towards the Future" implemented the SHP as one of its key structural measures in 1999 (Budowski et al., 1998; Budowski et al., 2001; Joye & Scherpenzeel, 1997). Experiences made from existing European panel surveys informed the SHP's design, notably the German Socio-Economic Panel (SOEP) (Schupp & Wagner, 2007) and the British Household Panel Study (BHPS), which is now integrated into Understanding Society, the UK Household Longitudinal Survey (UKHLS) (Buck & McFall, 2011).

Initially, the SHP was a joint project run by the Swiss National Science Foundation, the Swiss Federal Statistical Office, and the University of Neuchâtel. Between 2004 and 2007 the SHP developed a joint venture project "Living in Switzerland-2020" aimed at conducting a pilot study of the Statistics of Income and Living Conditions (CH-SILC) 2004-2005 survey in collaboration with the Swiss Federal Statistical Office. Since 2008, still funded by the Swiss National Science Foundation, the SHP has been integrated into the Swiss Centre of Expertise in the Social Sciences (FORS) hosted by the University of Lausanne.

Two main aims guide the SHP (Farago, 1996; Joye & Scherpenzeel, 1997). The first is to ensure a solid database for social reporting on stability and changes in living arrangements and wellbeing in Switzerland to complement the data collected by the Swiss Federal Statistical Office. Indeed, the SHP is a unique longitudinal study, because it offers data to comprehensively analyse mid- to long-term micro-social change; other surveys in Switzerland offer a smaller range of topics, follow a restricted subgroup, or allow only the study of short-term transitions (Tillmann et al., 2012). It also aims to promote opportunities for quantitative social science research by making high-quality data available to Swiss social scientists and to the international social science research community.

Data collected from household panels not only allow for the estimation of gross transitions but also provide an understanding of those transitions, including the circumstances (family events, a change in the activity status, health events, etc.) causing movements in and out of a given state (for example, transitions into and out of poverty). The SHP is an important tool for fine-tuning our conceptions and analyses of social dynamics (Berthoud & Gershuny, 2000; Budowski, et al., 2001; Rose, 1995) and changes across time (Menard, 2002).

The SHP features a broad range of fields and a variety of topics. This makes the SHP a valuable source of information for studies in different disciplines and allows for cross-domain analyses. To keep up with changes in the field, the SHP occasionally modifies the questionnaire and adds newly constructed variables to the dataset. A major criterion for any change to the questionnaire is that it should not compromise the comparability of the data over time. A second strong feature of the SHP is that all members of the households in the panel are interviewed. This allows for intra-household and intergenerational studies, such as the study of mutual influence of household members' attitudes and behaviours over time.

Design

General design

At present, the SHP comprises three samples: the SHP_I (5,074 households and 7,799 individuals interviewed first in 1999), the SHP_II (2,538 households and 3,654 individuals interviewed first 2004) and the SHP_III (3,989 households and 6,090 individuals interviewed first in 2013) (Voorpostel et al., 2014). See tables 3 and 4 for more details on the samples.

There are three types of questionnaires in the SHP: a household grid questionnaire to assess household composition, a household questionnaire, and an individual questionnaire. All household members aged 14 or older are eligible to answer the individual questionnaire. Each household has a reference person[2] who completes the household grid and the household questionnaire. The household questionnaire includes in addition a proxy questionnaire on household members younger than 14 years, who are absent for the time of the field work, or who are unable to respond themselves due to illness or disability.

Sampling procedure and follow-up procedure

For the first sample of the SHP in 1999 (SHP_I), the Swiss Federal Statistical Office drew a random sample of private households on the basis of the

Swiss telephone directory (SRH – Stichprobenregister für Haushalterhebungen – the sampling frame for household surveys). At the time of the SHP_I sample's selection, the SRH's coverage rate was about 95%. In order to compensate for selective nonresponse and attrition (e.g., deaths, hospitalisation, or migration) and to include new population groups, a random refreshment sample of households was added in 2004 (SHP_II) following the same sampling procedure. The sampling frame for SHP_II was CASTEM (Cadre de Sondage pour le Tirage d'Echantillons de Ménages), the follow-up to SRH, that is owned by the Swiss Federal Statistical Office; CASTEM also represents a telephone directory. CASTEM covered about 92% of the Swiss private households at the time of SHP_II sampling. A second refreshment sample was added in 2013 (SHP_III). This sample was drawn from the SRPH (Stichprobenrahmen für die Personen- und Haushaltserhebungen), which consists of data coming from the cantonal and communal register of residents; the SRPH is owned by the Swiss Federal Statistical Office. As this sampling frame is on an individual basis, the selection units of the SHP_III were individuals rather than households. The complete households of the selected individuals were subsequently included in the sample. The SHP_III sample also was no longer restricted by the availability of landline telephone connections.

All three random samples are stratified by the seven major statistical regions of Switzerland. Within each major geographic region, each household (SHP_I and SHP_II) or individual (SHP_III) had the same inclusion probability, independent of the size of the household.

The SHP's reference population includes all private households whose members represent the non-institutional resident population in Switzerland. Individuals living in old peoples' homes, institutions, or prisons, are not part of the reference population.

Follow-up procedure

For the first sample, all households that were interviewed in the first wave (with at least the household questionnaire and one individual questionnaire completed) formed the initial panel to be followed over time. For the second and third samples, all households that completed at least the grid questionnaire in the first wave were approached again. Households that were not reached at all during the first wave or those that did not supply any information at the time of the first wave were not included in the panel in later waves. Households were no longer approached if they could not be contacted for seven waves, refused to participate any longer, moved away from Switzerland, or moved to an institution.

On the individual level, the SHP initially only followed original sample members[3] (OSMs) from the first wave and their children; cohabitants[4] were only (re-)interviewed as long as they lived with an OSM. Since 2007, cohabitants have also been followed and have been entered into the panel as a new household upon leaving the original household. As a general rule, OSMs are followed indefinitely until they leave the target population (e.g., in the case of death or institutionalisation).

Survey content

Survey content overview

The household and individual questionnaires cover a broad range of topics.[5] They are also designed to collect both 'objective' data, such as financial resources, social position, and participation, and subjective data, such as satisfaction scores, values, and attitudes. The whole constitutes an operationalisation of different elements on the micro-social level: living conditions, life events, attitudes, perceptions, and lifestyles (Budowski, et al., 1998).

The questionnaire at *the household level* covers the following areas:

1. *composition of the household:* basic information (collected in the grid questionnaire) about all the members of the household, such as their age, sex, relations, nationality, level of education, and occupational status;
2. *accommodation:* the type and size of the accommodation, home ownership or tenancy, cost of and/or the subsidies received for housing, satisfaction with the accommodation, and evaluation of the state of the accommodation;
3. *standard of living*: possession of various goods such as cars, televisions or computers, and participation in various activities, such as holidays, meals at restaurants, or dentist visits, and the reasons (financial or otherwise) households do not have these goods or carry out these activities;
4. *the household's financial situation*: financial difficulties, indebtedness (and the reasons for

it), total household income, payments to other households, expenses (e.g. for childcare), satisfaction with income, an estimate of the minimum income the household considers necessary, and an evaluation of how the household's financial situation has evolved;
5. *the household and the family*: external help available to the household for housework, childcare, or care for other household members, the division of housework and childcare, and decision-making within the household.

The *individual questionnaires* cover the following topics[6]:
1. *the household and the family:* information on children living outside the household, time spent on housework, and satisfaction with private life and the share of housework;
2. *health and quality of life:* general illness and health problems, doctor and hospital visits, long-term handicaps, threats or attacks endured, self-perceived state of health, estimated evolution of the state of health, satisfaction with health and with life in general, feelings of safety, tobacco consumption, and physical activities;
3. *social origin (asked at first interview only):* information related to each respondent's parents, including profession, professional position, educational level, political positioning, nationality and any financial difficulties in the family of origin (at the reference age of 15);
4. *education:* the respondent's native language(s), level of education completed, education currently being pursued, and participation in on-the-job training;
5. *employment:* information on the respondent's profession, such as working conditions, number of hours worked, work schedule, atypical work, status in the labour market, previous jobs, job satisfaction, job insecurity, and personal qualifications;
6. *income:* total personal income, total professional income, social security pensions, social and private transfers, and other income, plus satisfaction with the financial situation and evaluation of changes in it;
7. *participation, integration, and networks:* frequency of social contacts, unremunerated work outside the home, participation in associations, membership of and participation in groups, assessment of social capital by means of evaluation of potential practical help and emotional support (from various social network ties) and general trust in people;
8. *politics and values:* political participation, membership, party identification, political positioning, satisfaction with the political system, evaluation of issues and political values;
9. *leisure and media:* leisure activities, amount of leisure and holiday time, use of media, and satisfaction with leisure and free time.
10. *psychological scales*: (from 2009 onwards) dimensions of self-perception (such as self-mastery and self-esteem) and other aspects like the Big Five personality traits.

Since the second wave, the questionnaire has also included a life events module assessing the occurrence of events such as the termination of relationships, deaths of family or friends, and conflicts with relatives; and an occupational calendar module assessing (on a monthly basis) the respondent's employment situation in the twelve months prior to the interview.

Unique, annual, and rotating content

In 2009 the SHP introduced a new system of modularization for the individual questionnaire. The SHP now contains three different types of questions: *unique* questions asked only once (usually in the first interview), *core* questions asked each wave and *rotating core* questions asked regularly (but not each year). Table 1 shows the different types of questions.

Table 1: Questionnaire content

Topics	Unique	Core	Rotating core
Last job*	x		
Social origin	x		
Socio-demographics		x	
Life events		x	
Health		x	
Education		x	
Current job		x	
Occupational calendar		x	
Income		x	
Social network			x
Leisure			x
Social participation			x
Politics			x
Religion			x
Psychological scales			x

* Last job refers to the last job held prior to entering the panel for those respondents who were not employed at the time of the first interview.

The rotating core questions are arranged in different modules, i.e. social network, religion, social participation, political behaviour and values, leisure and culture, and psychological scales. Table 2 shows the rotation calendar.

Table 2: Rotation calendar of the SHP modules from 2010 to 2020

Module	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020
Social network	X			X			X			X	
Religion			X			X			X		
Social participation		X			X			X			X
Political behaviour and values		X			X			X			X
Leisure and culture	X			X			X			X	
Psychological scales			X			X			X		

Life calendars

The questionnaires used in the first wave of the SHP_III differed from those used in SHP_I and SHP_II. In the first wave of the SHP_III, retrospective individual biographical data were collected. In addition to the regular grid and the household questionnaire, respondents in the SHP_III sample completed a life calendar covering their entire life course starting from birth. The SHP_III life calendar is presented as a two-way grid on paper with the temporal dimension (in years) for the rows and various life domains in the columns. Respondents were asked to report events for each domain in this grid. This questionnaire has been developed with the NCCR LIVES, a Swiss National Centre of Competence dedicated to life course research. Thus, the SHP_III has an original design, combining retrospective biographical data with prospective longitudinal data.

The grid provides a visual structure which enhances memory retrieval (Berney & Blane, 2003; Blane, 1996; Caspi et al., 1996). The SHP_III participants can visualize their life trajectories in all domains and can therefore link the occurrence and duration of events in different domains. Interrelatedness facilitates recall for distinct events because interrelated themes reflect individual autobiographical memories (Belli, 1998; Belli, Lee,

Stafford, & Chou, 2004). The visual structure also helps to detect gaps and inconsistencies. Overall, this method produces high-quality retrospective data (Freedman, Thornton, Camburn, Alwin, & Young-DeMarco, 1988).

The life calendar covers the following domains of life:

1. *residential trajectory*: residential mobility;
2. *residence permit*: the different residence permits of non-Swiss respondents;
3. *living arrangements*: with whom the respondent lived during the life course;
4. *partner relationships*: partner relationships and changes in civil status;
5. *family events*: the occurrence and timing of family-related events such as births of children, parents' separation or divorce, and deaths of family members;
6. *professional activities*: professional activities and periods during which the respondent received social benefits;
7. *health*: the occurrence and duration of different health problems.

Retrospective data also exist on a subsample of the SHP_I respondents (n=5,560). In 2001-2002, all SHP_I respondents were approached by mail with a self-completion questionnaire. This questionnaire collected information on education, work, and family history.

Data collection

Survey mode and interview length

The panel survey is conducted annually from September to February by the institute M.I.S. Trend in Lausanne and Bern. Interviews are conducted in (Swiss) German, French, and Italian. The main mode of interviewing is computer-assisted telephone interviewing (CATI). Alternative modes were offered only in the first wave of the SHP_III sample; if no telephone number was available, respondents completed the household questionnaire by computer assisted personal interviewing (CAPI). The biographical questionnaire in the first wave of the SHP_III was self-administered or administered with an interviewer present. In addition to the alternative modes in the SHP_III, since 2010 (wave 12), CAPI and computer assisted web interview (CAWI) have been offered as alternative survey modes to those who initially refused to participate.[7] The interviews require around 15 minutes to administer the grid and the household questionnaire and around 35 minutes to complete the individual questionnaires.

Fieldwork and measures to increase response

The fieldwork starts with sending a letter to the participating households informing them of the upcoming interviews. Enclosed with this letter, participants receive a newsletter containing some results of recent analyses of the SHP data. Since 2010, to enhance survey participation, each eligible respondent has received an *unconditional* incentive enclosed with the preliminary letter (a 20 CHF voucher for a popular chain of supermarkets for the SHP_I and the SHP_II, and 10 CHF voucher for the SHP_III).[8] Between wave 12 and wave 14, an additional incentive was offered to *complete households* for households of at least two eligible persons. This additional incentive (50 CHF) was given to the participants at the end of the fieldwork. For budgetary reasons the additional incentive was dropped in 2013.

To guarantee the smooth functioning of the fieldwork, M.I.S. Trend ensures a strict selection of only experienced interviewers who are native speakers. To increase the interviewers' motivation they can earn two collective bonuses. One bonus is based on the general response rate: all interviewers together have to complete at least 95% of the number of interviews carried out in the previous wave. The second bonus is geared towards interviewers who are engaged in refusal calls; it is based on the refusal conversion rate.

The SHP also invests significantly in refusal conversion, and makes great efforts to maintain contact with the households. Households that have not participated for at least one year are re-approached progressively. These households are sent a preliminary letter with the request to take part in the survey again. Only the most successful and specially trained interviewers are selected to contact these households. Households and individuals who refuse participation in the current wave are also re-contacted at a later point by interviewers trained in refusal conversion. This has resulted in a high refusal conversion rate; for example, about 45% of these households eventually participated in the survey in 2014.

Panel maintenance

To avoid households dropping out from the panel because of unsuccessful tracing (due to

moving, changed phone numbers, household splits, etc.), several measures ensure that contact can be re-established with the respondents in later waves. In addition, a newsletter is enclosed with the advance letter at the start of each fieldwork phase, and respondents are asked to provide their mobile number and their e-mail address. If respondents are not willing to give this information or do not have a mobile number or e-mail address, they are asked to leave the address of an auxiliary (e.g., a family member living outside the household or a close friend) who can help in case of losing track of the respondent. Third, during the fieldwork period, households are called on different days of the week and at different times during the day in order to minimise non-contact. Fourth, a bilingual interviewer is responsible for relocating lost respondents. This interviewer tries to contact the respondent by mobile phone, e-mail, or through the auxiliary, searching different directories and registers to locate the respondent.

Initial wave response and sample attrition

At the household level, response rates were 64% in the first wave of the first sample (1999), 65% in the first wave of the second sample (2004), and 60% in the first wave of the third sample (2013). On the individual level, response rates (conditional upon household participation) were 85%, 76%, and 81% respectively. Appendix A provides information on how the initial sample of SHP_III compared to the population distributions in 2013 on several characteristics[9]. The distribution in the sample is very close to that of to the general population, with a slight underrepresentation of those in the age category 25-34, men, and foreigners. The initial samples of the SHP also had a higher share of married individuals and a lower share of divorced individuals compared to the population.

Tables 3 and 4 show the number of households and individuals interviewed in every wave for the three samples. The tables show that sample sizes in the SHP, as in most panel studies, declined over time. After 16 waves of data collection, the number of households and individuals interviewed for the SHP_I corresponds to 55% of the initial number of households and 60% of the initial number of individuals interviewed. For the SHP_II, these figures are 55% and 59%, respectively, after 11 waves. SHP_III has only two waves, after which 81% of original households and 87% of original individuals remain.

Table 3: Number of households interviewed in SHP_I, SHP_II and SHP_III (1999-2014)

Year	Wave	SHP_I n	% of sample size in 1999	SHP_II n	% of sample size in 2004	SHP_III n	% of sample size in 2013	SHP_I+II +III n
1999	1	5,074	100					5,074
2000	2	4,425	87					4,425
2001	3	4,139	82					4,139
2002	4	3,582	71					3,582
2003	5	3,227	64					3,227
2004	6/1	2,837	56	2,538	100			5,375
2005	7/2	2,457	48	1,799	71			4,256
2006	8/3	2,537	50	1,684	66			4,221
2007	9/4	2,817	56	1,494	58			4,311
2008	10/5	2,718	54	1,546	61			4,264
2009	11/6	2,930	58	1,476	58			4,406
2010	12/7	2,985	59	1,557	61			4,542
2011	13/8	2,977	59	1520	60			4,495
2012	14/9	2,968	58	1,493	59			4,461
2013	15/10/1	2,881	57	1,488	57	3,989	100	8,358
2014	16/11/2	2,778	55	1,385	55	3,220	81	7,383

Table 4: Number of persons interviewed in SHP_I, SHP_II and SHP_III (1999-2014)

Year	Wave	SHP_I n =	% of sample size in 1999	SHP_II n =	% of sample size in 2004	SHP_III n =	% of sample size in 2013	SHP_I+II +III n =
1999	1	7,799	100					7,799
2000	2	7,073	91					7,073
2001	3	6,601	85					6,601
2002	4	5,700	73					5,700
2003	5	5,220	67					5,220
2004	6/1	4,413	57	3,654	100			8,067
2005	7/2	3,888	50	2,649	72			6,537
2006	8/3	4’091	52	2,568	70			6,659
2007	9/4	4,630	59	2,350	64			6,980
2008	10/5	4,494	58	2,410	66			6,904
2009	11/6	4,800	62	2,309	63			7,109
2010	12/7	5,057	65	2,489	68			7,546
2011	13/8	5,103	65	2,481	68			7,584
2012	14/9	5,032	65	2,414	66			7,446
2013	15/10/1	4,880	63	2,327	64	6,090[a]	100	13,297
2014	16/11/2	4,678	60	2,150	59	5,291	87	12,119

[a]) This number refers to the number of life calendars collected among respondents aged 16 and older.

The numbers in these tables reflect the number of observations in each wave, but do not give information on the respondents’ longitudinal participation. Table 5 shows that respondents in SHP_I (on average) participated in 7.3 waves; on average the respondents in SHP_II and SHP_III responded in 5.5 and 1.6 waves, respectively. Table 5 also gives the number of respondents who participated in every wave (n=1,598 in SHP_I, n=957 in SHP_II and n=4,453 in SHP_III). The table also shows that a significant number of participants are still being followed despite having missed some waves of observation. Finally, the table provides the number of respondents who have not been interviewed in the last three waves, suggesting the number of dropouts from the study (5,404 for SHP_I, and 2,129 for SHP_II) –although these respondents may still re-enter at a later point in time.

Table 5: Characteristics of the SHP_I, SHP_II and SHP_III

	SHP_I	SHP_II	SHP_III
Households interviewed in first wave	5,074	2,538	3,989
Individuals interviewed in first wave	7,799	3,654	6,090
Years of data released	1999-2014	2004-2014	2013-2015
Total number of waves currently available	16	11	2
Mean number of waves in which individual sample members[a] participated	7.3	5.5	1.6
Number of individuals who participated in all waves (fully longitudinal respondents)	1,598	957	4,453
Number of individuals who participated in all waves for which they were eligible	2,470	1,287	4,590
Number of individuals who missed at least one wave but were present in one of the three most recent waves	3,061	1,415	n/a
Number of individuals who did not participate in the last three waves	5,404	2129	n/a
Number of individuals known to be deceased, to be institutionalized or to have emigrated	536	187	23

[a]) This percentage is based on all sample members who have participated at least once. Household members who never completed an individual questionnaire are not included.

Comparing the different response patterns (respondents who always participate, those who sometimes participate, and those who have not participated in the last three waves), dropouts and wave non-respondents are clearly somewhat selective (Voorpostel, 2010). As is found in other studies (Groves, 2006; Lillard & Panis, 1998; Neukirch, 2002; Watson & Wooden, 2009), SHP non-respondents are more likely to be younger, male, less educated, unemployed, and in poorer health; they are also less likely to be married or own a home (see Appendix C in the SHP Userguide, Voorpostel, et al., 2014). Nonetheless, these factors contribute only marginally to explaining response to the survey. For the most part, response seems random (Voorpostel, 2010).

Data adjustments: Weighting and imputation

It is essential to use weights in order to have estimates that are representative of the underlying population. The SHP provides several weights. First, weights are assigned to households and to individuals. Second, a distinction is made between cross-sectional and longitudinal weights. Cross-sectional weights assure that the sample is representative for any given year of data collection and can be used for cross-sectional analysis (for example, to calculate the percentage of households living in poverty or the population's general satisfaction with life in 2013). Longitudinal weights are meant for longitudinal analysis and refer to the first wave of the panel. These weights are used if respondents are followed over time from the first wave. These weights hence refer to the population in the first wave of a particular panel (1999, 2004, and 2013 for SHP_I, SHP_II, and SHP_III, respectively). For the SHP_II, for example, these weights should be used when conducting a longitudinal analysis starting in 2004 and continuing to any later wave (e.g., to study changes in general satisfaction with life). Both cross-sectional and longitudinal weights are assigned to individuals, but households are given only cross-sectional weights.[10] For each of the type of weight, the SHP delivers two

versions: one weight to obtain the size of the relevant Swiss population and one to maintain the sample size.

The weights are constructed in four steps. First, the inclusion probabilities are determined for every unit of the reference population, and then the inverse is taken as the initial weight. In a second step, the weights are adjusted for non-response, both in the initial wave and in later waves. The method used for modelling non-response is analysis by segmentation, as proposed by Kass (1980). Information on the non-respondents comes from the official registers from which the samples were drawn, and from questionnaires completed in previous waves.[11] To obtain weights for OSMs' cohabitants – whose inclusion probability is unknown – the generalised weight share method (GWSM) is used for the cross-sectional individual weight and the household weight (Lavallée, 2002). The third step consists of combining the three panels using a factor allocating a relative importance to each of the samples based on its size. Finally, all weights are adjusted so that the estimated population sums are equal to the actual sums of the non-institutionalised Swiss population. In rare cases, Winsorisation (Hastings, Mosteller, Tukey, & Winsor, 1947) is needed to correct for negative or extreme weights.[12]

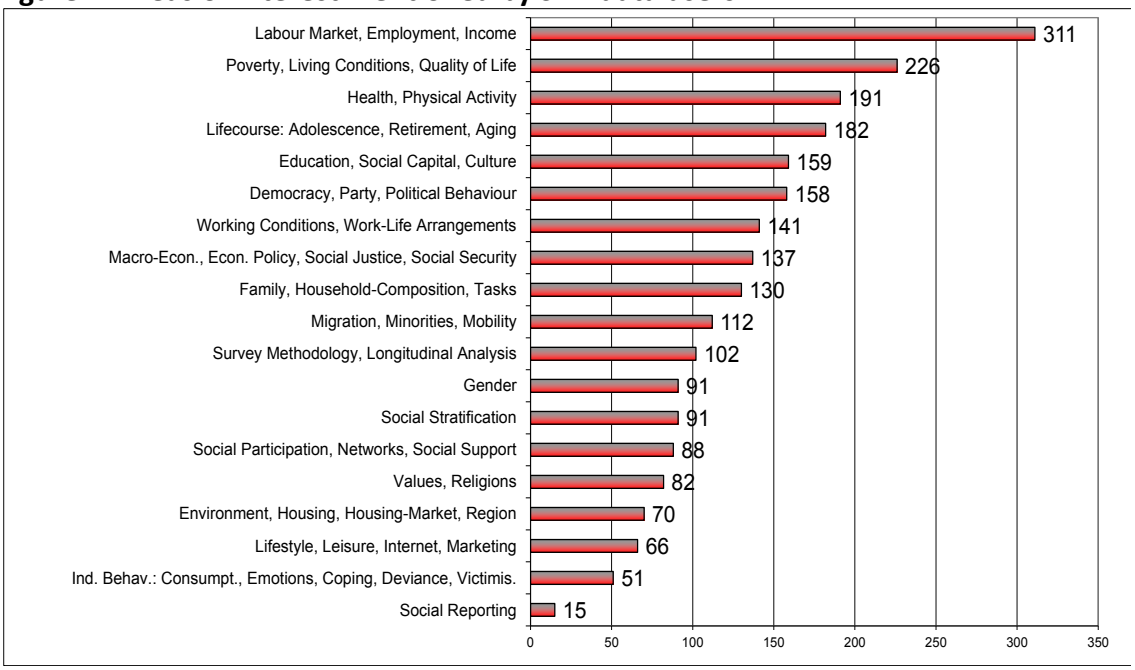
As the longitudinal weights can only be used when analysing change since the first wave of any given panel, they are not suitable if the period analysed starts at a later wave. For the development of longitudinal samples that start after the first wave, transitional factors are provided. These transitional factors enable the researcher to create custom-made individual longitudinal weights over several consecutive waves. More information on the construction of these transitional factors, their applicability, and their limitations is available on the SHP homepage. Full documentation about the SHP's weighting scheme of the SHP can also be found on the SHP website.

Data use

SHP data are available free of charge. Users must sign a contract (available on the SHP website) and report the research topics for which they intend to use the SHP data. The currently registered SHP data users (n=1,762) have reported a wide variety of topics.

Figure 1 shows the relative importance of the single topic categories given by the SHP data users. The category with the topics "Labour Market, Employment, Income" leads the table. "Poverty, Living Conditions, Quality of Life", "Health, Physical Activity" and "Life Course: Adolescence, Retirement, Aging" are also among the most frequently listed topics. Moreover, around 300 users mentioned using the data for seminars and courses.

Figure 1: Areas of interest mentioned by SHP data users



The value of the SHP as a tool to investigate social change is also demonstrated in the large number of publications in various disciplines that have been based on the data. These studies have addressed a wide variety of topics, including changes in wellbeing following major life events (Anusic, Yap, & Lucas, 2014); changes in social contacts over the life course (Kalmijn, 2012); mutual influence between mothers, fathers and children with respect to support for political parties (Fitzgerald, 2011); the impact of parental divorce on political and civic participation (Voorpostel & Coffé, 2014); the relation between conspicuous consumption in the neighbourhood and income satisfaction (Winkelmann, 2012); and the determinants of change in physical activity levels in young adults (Zimmermann-Sloutskis, Wanner, Zimmermann, & Martin, 2010). The SHP has also featured in many cross-national comparative studies. Examples include studies on the causal connection between civic engagement and generalised trust in several countries (van Ingen & Bekkers, 2015); the stability of political interest over time in different countries (Prior, 2010); the relationship between unemployment and wellbeing in Germany and Switzerland (Oesch & Lipps, 2012); and the impact of political discord in the family on engagement in politics in Switzerland, Germany, and the UK (Fitzgerald & Curtis, 2012).[13]

International comparison

The SHP was designed to allow cross-national comparisons with other household studies. In 2008 the SHP was included in the Cross-National Equivalent File (CNEF), which provides harmonized data from several household studies. To date the CNEF comprises the German SOEP, the BHPS, the US Panel Study of Income Dynamics (PSID), the Canadian Survey of Labour and Income Dynamics (SLID), the Household Income and Labour Dynamics in Australia (HILDA), the Korean Labor and Income Panel Study (KLIPS) and the Russia Longitudinal Monitoring Survey (RLMS-HSE) (Frick, Jenkins, Lillard, Lipps, & Wooden, 2007). The CNEF covers the following main topics: demographics, employment, income, and health.[14]

Moreover, the SHP contains variables in various domains that can be compared to other panels, such as political behaviour and values (for example, the left-right self-placement scale, interest in politics, participation in polls, and general trust in people), social participation (for example, participation in sport/leisure associations, unions, political parties or charitable organisations), leisure and culture (different items broadly comparable with those of the SOEP and the UKHLS), religion (with usual questions on religious affiliation and participation in religious services), and psychological scales (for example, the Big Five personality traits, the Morally Debatable Behaviour Scale, satisfaction scales, and sense of control). In addition, the SHP provides internationally comparable constructed variables for research in social stratification in particular (such as Treiman's prestige scale or the European Socio-Economic Classification (ESeC)).

Conclusion

This paper described the main aspects of the SHP and analysed how it is distinctive as a long-term longitudinal survey. The SHP has been collecting high-quality data on Swiss households for 16 years, and it has good prospects to continue for many more years to come. With 16 years of observation, the SHP is an extremely valuable source of data for researchers in the social sciences in Switzerland and abroad. The SHP aims to provide both continuity and innovation in measurement and data collection. One notable innovation was the design of the SHP_III, which combined retrospective and prospective longitudinal data.

The importance of the SHP for the research community is borne out by its large number of active data users, who have contributed to an impressive list of publications based on the SHP data, covering a very broad spectrum of research domains. This is a strong indication that the multidisciplinary SHP survey serves the research needs of a diverse and interdisciplinary academic community, both nationally and internationally.

Acknowledgements

The Swiss Household Panel (SHP) is based at the Swiss Centre of Expertise in the Social Sciences (FORS). The project is supported by the Swiss National Science Foundation.

References

Anusic, I., Yap, S. Y., & Lucas, R. (2014). Testing set-point theory in a Swiss national sample: Reaction and adaptation to major life events. *Social Indicators Research, 119*(3), 1265-1288. http://dx.doi.org/10.1007/s11205-013-0541-2

Belli, R. F. (1998). The structure of autobiographical memory and the event history calendar: Potential improvements in the quality of retrospective reports in surveys. *Memory, 6*(4), 383-406. http://dx.doi.org/10.1080/741942610

Belli, R. F., Lee, E. H., Stafford, F. P., & Chou, C.-H. (2004). Calendar and question-list survey methods: Association between interviewer behaviors and data quality. *Journal of Official Statistics, 20*(2), 185.

Berney, L., & Blane, D. (2003). The life grid method of collecting retrospective information from people at older ages. *Research Policy and Planning, 21*(2), 13-22.

Berthoud, R., & Gershuny, J. (2000). *Seven years in the lives of British families: Evidence on the dynamics of social change from the British Household Panel Survey*: Policy Press.

Blane, D. B. (1996). Collecting retrospective data: development of a reliable method and a pilot study of its use. *Social science & medicine, 42*(5), 751-757. http://dx.doi.org/10.1016/0277-9536(95)00340-1

Buck, N., & McFall, S. (2011). Understanding Society: design overview. *Longitudinal and Life Course Studies, 3*(1), 5-17.

Budowski, M., Niklowitz, M., Scherpenzeel, A., Tillmann, R., Wernli, B., & Zimmermann, E. (1998). *Aims and architecture of the Swiss Household Panel*. Neuchatel: Swiss Household Panel.

Budowski, M., Tillmann, R., Zimmermann, E., Wernli, B., Scherpenzeel, A., & Gabadinho, A. (2001). The Swiss Household Panel 1999-2003: Data for research on micro-social change. *ZUMA-Nachrichten, 50*, 100-125.

Caspi, A., Moffitt, T. E., Thornton, A., Freedman, D., Amell, J. W., Harrington, H., Smeijers, J., & Silva, P. A. (1996). The life history calendar: A research and clinical assessment method for collecting retrospective event-history data. *International Journal of Methods in Psychiatric Research, 6*, 101-114. http://dx.doi.org/10.1002/(SICI)1234-988X(199607)6:2<101::AID-MPR156>3.3.CO;2-E

Farago, P. (1996). *Gesellschaftliche dauerbeobachtung im SP "Zukunft Schweiz/Demain la Suisse" [Swiss Priority Programme "Switzerland towards the future"]*. Bern: Swiss National Science Foundation.

Fitzgerald, J. (2011). Family dynamics and Swiss parties on the rise: Exploring party support in a changing electoral context. *The Journal of Politics, 73*(3), 783-796. http://dx.doi.org/10.1017/S0022381611000454

Fitzgerald, J., & Curtis, K. A. (2012). Partisan discord in the family and political engagement: A comparative behavioral analysis. *The Journal of Politics, 74*(01), 129-141. http://dx.doi.org/10.1017/S0022381611001150

Freedman, D., Thornton, A., Camburn, D., Alwin, D., & Young-DeMarco, L. (1988). The life history calendar: A technique for collecting retrospective data. *Sociological methodology, 18*(1), 37-68. http://dx.doi.org/10.2307/271044

Frick, J. R., Jenkins, S. P., Lillard, D. R., Lipps, O., & Wooden, M. (2007). The Cross-National Equivalent File (CNEF) and its member country household panel studies. *Schmollers Jahrbuch, 127*, 627-654.

Groves, R. M. (2006). Nonresponse rates and nonresponse bias in household surveys. *Public Opinion Quarterly, 70*(5), 646-675. http://dx.doi.org/10.1093/poq/nfl033

Hastings, C., Mosteller, F., Tukey, J. W., & Winsor, C. P. (1947). Low Moments for Small Samples: A Comparative Study of Order Statistics. *The Annals of Mathematical Statistics, 18*(3), 413-426. http://dx.doi.org/10.1214/aoms/1177730388

Joye, D., & Scherpenzeel, A. (1997). *Observation à long terme: Projet de panel. Pro-gramme Prioritaire Demain la Suisse*. Bern: Swiss National Science Foundation.

Kalmijn, M. (2012). Longitudinal analyses of the effects of age, marriage, and parenthood on social contacts and support. *Advances in Life Course Research, 17*(4), 177-190. http://dx.doi.org/10.1016/j.alcr.2012.08.002

Kass, G. (1980). An exploratory technique for investigating large quantities of categorical data. *Applied Statistics, 29*(2), 119-127. http://dx.doi.org/10.2307/2986296

Lavallée, P. (2002). *Le sondage indirect ou la méthode généralisée du partage des poids*. Paris: Ellipses.

Lillard, L. A., & Panis, C. W. A. (1998). Panel attrition from the Panel Study of Income Dynamics: Household income, marital status, and mortality. *Journal of Human Resources, 13*(2), 437-457. http://dx.doi.org/10.2307/146436

Menard, S. (2002). *Longitudinal research*. Thousand Oaks: Sage.

Neukirch, T. (2002). *Nonignorable attrition and selectivity biases in the Finnish subsample of the ECHP: An empirical study using additional register information. Chintex Working Paper #5.* Frankfurt-am-Main: J.W. Goethe-Universität.

Oesch, D., & Lipps, O. (2012). Does unemployment hurt less if there is more of it around? A panel analysis of life satisfaction in Germany and Switzerland. *European Sociological Review, 29*(5), 955-967. http://dx.doi.org/10.1093/esr/jcs071

Prior, M. (2010). You've either got it or you don't? The stability of political interest over the life cycle. *The Journal of Politics, 72*(03), 747-766. http://dx.doi.org/10.1017/S0022381610000149

Rose, D. (1995). Household panel studies: An overview. *Innovation: The European Journal of Social Science Research, 8*(1), 7-24. http://dx.doi.org/10.1080/13511610.1995.9968428

Schupp, J., & Wagner, G. (2007). The German Socio-Economic Panel Study (SOEP): Scope, evolution and enhancements. *Schmollers Jahrbuch, 127*(1), 139-169.

Tillmann, R., Voorpostel, M., Bloch, D., Kuhn, U., Lebert, F., Rothenbühler, M., Ryser, V.-A., Schmid, F., & Wernli, B. (2012). *Swiss Household Panel : Scientific Report 2011*. Lausanne: FORS.

van Ingen, E., & Bekkers, R. (2015). Generalized trust through civic engagement? Evidence from five national panel studies. *Political Psychology, 36*(3), 277-294. http://dx.doi.org/10.1111/pops.12105

Voorpostel, M. (2010). Attrition in the Swiss Household Panel by demographic characteristics and levels of social involvement. *Swiss Journal of Sociology, 36*(2), 359-377.

Voorpostel, M., & Ryser, V.-A. (2011). *Mixed mode data collection as a strategy to decrease panel attrition in the Swiss Household Panel*. Lausanne: FORS.

Voorpostel, M., & Coffé, H. (2014). The effect of parental separation on young adults' political and civic participation. *Social Indicators Research*, *124*(1), 295-316. http://dx.doi.org/10.1007/s11205-014-0770-z

Voorpostel, M., Tillmann, R., Lebert, F., Kuhn, U., Lipps, O., Ryser, V.-A., Schmid, F., Antal, E., & Wernli, B. (2014). *Swiss Household Panel User Guide (1999-2013), Wave 15*. Lausanne: FORS.

Watson, N., & Wooden, M. (2009). Identifying factors affecting longitudinal survey response. In P. Lynn (Ed.), *Methodology of longitudinal surveys* (pp. 157-182). London: Wiley. http://dx.doi.org/10.1002/9780470743874.ch10

Winkelmann, R. (2012). Conspicuous consumption and satisfaction. *Journal of Economic Psychology, 33*(1), 183-191. http://dx.doi.org/10.1016/j.joep.2011.08.013

Zimmermann-Sloutskis, D., Wanner, M., Zimmermann, E., & Martin, B. W. (2010). Physical activity levels and determinants of change in young adults: a longitudinal panel study. *International Journal of Behavioral Nutrition and Physical Activity, 7*(2), 1-13. http://dx.doi.org/10.1186/1479-5868-7-2

Appendix A

Table A.1: Representativeness of the SHP_III distributions in 2013

Age	**Population**	**SHP_III**
14-19	7.46%	8.99%
20-24	6.87%	6.97%
25-34	15.98%	11.61%
35-44	17.09%	16.96%
45-54	18.74%	19.49%
55-64	14.34%	14.94%
65-	19.53%	21.06%
	100.00%	**100.00%**
Gender		
Male	49.64%	48.92%
Female	50.36%	51.08%
	100.00%	**100.00%**
Swiss nationality		
Swiss	75.84%	82.51%
Foreigner	24.17%	17.49%
	100.00%	**100.00%**
Region		
Geneva, Vaud, Valais	18.80%	18.46%
Bern, Fribourg, Jura, Neuchâtel, Solothurn	22.20%	23.20%
Aargau, Basel-Landschaft, Basel-Stadt	13.63%	13.51%
Zürich	17.89%	16.01%
Appenzell Ausserrhoden, Appenzell Innerrhoden, Glarus, Graubünden, Schaffhausen, St. Gallen, Thurgau	13.82%	14.26%
Luzern, Nidwalden, Obwalden, Schwyz, Uri, Zug	9.38%	10.24%
Ticino	4.29%	4.30%
	100.00%	**100.00%**
Civil status		
Single	43.08%	43.19%
Married	44.79%	46.67%
Widower	4.36%	3.63%
Divorced	7.77%	6.51%
	100.00%	**100.00%**

Endnotes

[1] www.swisspanel.ch
[2] 61% of the reference persons in 2013 were female. Among households including couples this was 58%.
[3] These include all eligible household members living in the selected households in the first wave (in 1999, 2004 or 2013).
[4] Cohabitants are persons who entered the selected households after the first wave, and who are not children of any OSM.
[5] Questionnaires can be searched online, and are also available in pdf format.
[6] The first wave of the SHP_III did not include an individual questionnaire. The first time an individual questionnaire was administered was in Wave 2 (2014). In Wave 1 in addition to the grid and household questionnaire, respondents completed a biographical questionnaire (see the Life calendars section).
[7] In 2010, the SHP tested these alternative modes of data collection (CAPI and CAWI) among initial refusals as a first step toward a potential mixed-modes strategy of data collection. An evaluation of this mixed-mode approach showed that it is not useful and even risky, at least in terms of participation, to implement a full mixed-modes system of data collection for the SHP survey (see Voorpostel and Ryser, 2011).
[8] The incentive for the SHP_III is lower for budgetary reasons only. Between wave 8 and wave 12 participants received a conditional incentive after completion of the individual questionnaire; before wave 8 there were no incentives.
[9] The distributions of the initial samples of the SHP_I and SHP_II were comparable. For more detailed information on representativeness and weight construction of the SHP_I and SHP_II samples, full documentation can be found here and here.
[10] Because the household composition changes over time, it does not make sense to assign them longitudinal weights.
[11] In addition, at the first wave of the SHP_II and SHP_III a non-response survey (PAPI) was sent to all the households who could not be contacted or who refused to participate.
[12] This procedure is used to replace extreme values by the values at the first and the 99th percentile of the weight distribution.
[13] A complete list of publications based on the SHP can be found here.
[14] See the variable list and availability for each country file at http://cnef.ehe.osu.edu/files/2012/11/CNEF_codebooks_All.pdf.

COMMENT AND DEBATE

Origins of heath inequalities: the case for Allostatic Load

Cyrille Delpierre Université of Toulouse III Paul Sabatier, France
cyrille.delpierre@inserm.fr
Cristina Barbosa-Solis Université of Toulouse III Paul Sabatier, France
Jerome Torrisani Cancer Research Center of Toulouse, France
Muriel Darnaudery University of Bordeaux, France
Melanie Bartley University College London, UK
David Blane Imperial College London, UK
Michelle Kelly-Irving Université of Toulouse III Paul Sabatier, France
Linn Getz Norwegian University of Science and Technology, Norway
Margret Olafia Tomasdottir University of Iceland, and Primary Health Care of the Capital Area, Iceland
Tony Roberston University of Stirling, UK
Per E. Gustafsson Umeå University, Sweden

http://dx.doi.org/10.14301/llcs.v7i1.325

Abstract

In an opening paper Delpierre et al. explore the concept of allostatic load. The impact of the environment on our biological systems is summarised by the concept of embodiment. The biological embedding of social conditions could therefore be a relevant mechanism to partly explain the social gradient in health. A key issue is how to measure the 'physiological reality' – the biological expression of embodiment at individual and population levels. Allostatic load (AL) has been proposed as a measure of the overall cost of adapting to the environment and may be a relevant tool or concept for measuring the way we have embodied our environment. Social inequalities in health may be partly explained by the embodiment of social environments, and AL may allow us to measure and compare embodiment between socioeconomic groups. However, before operationalising AL, a number of issues deserve further exploration. Among these, the choice of biological systems, and variables within each system, that should be included to remain 'loyal' to the theory of biological multisystem wastage underlying AL and the most appropriate methodological approach to be used to build an AL score, are particularly important. Moreover, studies analysing the link between adverse environments (physical, chemical, nutritional, psychosocial) across the life course and AL remain rare. Such studies require cohorts with data on socioeconomic and psychosocial environments over the life course, with multiple biological measures, made at various stages across the life span. The development and maintenance of these cohorts is essential to continue exploring the promising results that could enhance our understanding of the genesis of the social gradient in health by measuring embodiment. These points are then debated in commentaries by Linn Getz and Margret Olafia Tomasdottir, Tony Robertson and Per Gustafson. The commentaries are followed by a response from the authors of the opening paper.

Keywords

Allostatic load, embodiment, social epidemiology

Allostatic load as a measure of social embodiment: conceptual and empirical considerations

Cyrille Delpierre Université of Toulouse III Paul Sabatier, France
cyrille.delpierre@inserm.fr
Cristina Barbosa-Solis Université of Toulouse III Paul Sabatier, France
Jerome Torrisani Cancer Research Center of Toulouse, France
Muriel Darnaudery University of Bordeaux, France
Melanie Bartley University College London, UK
David Blane Imperial College London, UK
Michelle Kelly-Irving Université of Toulouse III Paul Sabatier, France

(Received October 2014 Revised July 2015)

Introduction

The impact of the environment on our biological systems is summarised by the concept of embodiment. Krieger (2005) described embodiment as "how we, like any living organism, literally incorporate, biologically, the world in which we live, including our societal and ecological circumstances". The notion of embodiment refers to the fact that every human being is both a social and a biological organism that incorporates the world in which (s)he lives.

In consequence, an adverse socioeconomic environment may be implicated in the development of future diseases by modifying certain biological processes especially when exposures occur early in life. In the 1990s Barker (1990) showed that intrauterine growth retardation was associated with an increased risk of cardiovascular and metabolic diseases in adulthood, introducing the foetal origin of disease hypothesis. This postulates that environmental conditions during specific windows of development can have long-term effects on organogenesis, and metabolic and physiological processes. However, embodiment is a continuous process that occurs throughout life, with some periods of life being more sensitive than others to changes induced by the environment. As a phenomenon occurring over the life course, embodiment may partly explain the social gradient observed for the vast majority of chronic diseases. Hertzman (1999) wrote "the process whereby differential human experiences systematically affect the healthfulness of life across the life cycle has been termed *biological embedding*". If embodiment, or biological embedding, refers to the concept of environmental adaptation shared by living beings, a key question is how to measure the physiological reality, the biological expression of embodiment at individual and population levels?

Recently, we showed that psychosocial adversity during childhood (child spent time in care, physical neglect, parental contact with the prison service, parental separation including by death or divorce, family experience of mental illness, family experience of substance abuse) increased twofold the risk of cancer diagnosis and all-cause mortality before 50 years of age, after adjusting for several confounding factors like socioeconomic characteristics at birth, birth weight and breastfeeding. Including mediating factors in the model, like health behaviours or adult socioeconomic position, only slightly decreased the effect of childhood psychosocial adversity (Kelly-Irving et al., 2013a; Kelly-Irving et al., 2013b). Of course, there are a number of possible explanations for these results, such as methodological flaws in design and analysis, or not including an *a priori* confounding or mediating factor. However, one possible explanation is that the childhood psychosocial environment might have resulted in changes to biological systems during development that may alter health over time.

Due to immaturity at birth, humans, as with other altricial mammals, mature in constant interaction with the environment. Our environment

is highly variable requiring the permanent adaptation of physiological systems. This adaption through changes is crucial for survival and refers to allostasis (Sterling & Eyer, 1988). Three main systems, nervous, endocrine and immune, are involved in the allostasis processes, all of which mature during the postnatal period and into adulthood (Adkins, Laclerc and Marshall-Clarke, 2004; Gogtay et al., 2004). Chronic exposures to psychosocial stressors and inter-individual differences in the susceptibility to stress are both associated with a prolonged activation of these allostatic systems. This may lead to an allostatic overload with potentially detrimental health consequences. Allostatic load (AL) is therefore the price paid by the body over time for adapting to challenges. It refers to the concept of biological multisystem wastage, whereby "the strain on the body produced by repeated ups and downs of physiologic response, as well as by the elevated activity of physiologic systems under challenge, and the changes in metabolism and the impact of wear and tear on a number of organs and tissues, can predispose the organism to disease" (McEwen & Stellar, 1993).

An AL score should, by definition, be a composite measure including various physiological systems in order to capture overall physiological wear-and-tear. The MacArthur Study of Successful Aging was the first to propose an AL score (Seeman, Singer, Rowe, Horwitz & McEwen, 1997). Parameters included systolic and diastolic blood pressure (indexes of cardiovascular activity); waist-hip ratio (an index of more long-term levels of metabolism and adipose tissue deposition), thought to be influenced by increased glucocorticoid activity; serum high-density lipoprotein (HDL) and total cholesterol levels (indexes of long-term atherosclerotic risk); blood plasma levels of total glycosylated haemoglobin (an integrated measure of glucose metabolism during a period of several days); serum dehydroepiandrosterone sulphate (DHEA-S) (a functional HPA axis antagonist); 12-hour urinary cortisol excretion (an integrated measure of 12-hour HPA axis activity); 12-hour urinary norepinephrine and epinephrine excretion levels (integrated indexes of 12-hour sympathetic nervous system activity). Some variants of the original items can be found in the literature but the markers most commonly used are associated with cardiovascular and metabolic diseases (blood pressure, heart rate, blood glucose, insulin, blood lipids, body mass index or waist circumference), HPA axis (cortisol, DHEA-S), sympathetic nervous system (epinephrine, norepinephrine, dopamine) and inflammation (C-reactive protein, IL-6) (Seeman, Epel, Gruenewald, Karlamangla & McEwen, 2010). These various scores of AL have been shown to be better predictors of mortality and functional limitations than the metabolic syndrome or any of the individual components used to measure AL when analysed separately (Seeman, McEwen, Rowe & Singer, 2001). AL score is also associated with an increased incidence of cardiovascular disease, and poorer cognitive function (Seeman et al. 1997). Recent research also suggests a link between early environment and AL (Danese & McEwen, 2012; Danese et al., 2009; Shonkoff & Garner, 2012).

As a measure of the global cost of adapting to (and coping with) the environment, AL may be a relevant tool or concept for measuring the way we have embodied our environment. As the way in which human populations embody their environment may partly explain social inequalities in health, we guess that AL may be a relevant and useful tool for measuring and comparing embodiment between population and socioeconomic groups. However, some important issues regarding AL deserve consideration:

Representing multiple biological systems

There is increasing evidence that many chronic diseases are related: this disease interrelatedness, or human disease network, is well established for metabolic diseases like obesity, diabetes and vascular diseases, and more recently for Alzheimer's disease/dementia, and cancer (Barabasi, Gulbahce & Loscalzo, 2011). There is biological plausibility behind the observed associations between these diseases that exemplify health decline and aging processes over the life course. Endocrine physiology and inflammatory processes are shared and many of the same risk factors, such as hyperglycaemia, inflammatory responses or health behaviours, are common to these pathologies. Further progress in understanding therefore requires the development of a measure representing the physiological systems relevant to these diseases. However the AL scores most commonly used are strongly focused on the cardiovascular or metabolic systems. The conceptualisation of AL as a dysregulation across

multiple physiological systems requires that the measure includes a balance of relevant systems, as well as the cardiovascular or metabolic ones. For instance, the inflammatory and immune systems that are involved in various chronic diseases ought to be represented. A main question is therefore how to decide which systems to represent. One of the solutions is to adopt an *a priori* definition of the systems that should be included in the measure of AL by choosing major regulatory systems known to be involved in chronic stress responses. An alternative may be to select major biological systems affecting health (Seeman et al., 2010) with the risk to be limited for studying the link between AL and subsequent health, if health is included in AL score. It may be possible that one single combination of markers do not equally predict different chronic diseases like cardiovascular or metabolic diseases, cancer or Alzheimer's disease, so that our measure of embodiment may need to be adapted according to the health condition under investigation.

Choosing relevant biological markers in each system

After identifying the physiological systems relevant for inclusion in an AL score, it is necessary to define the biological markers within each system that are the most appropriate proxies to summarise the state of that system. Moreover, AL markers could be drawn from several very different physiological 'levels' from epigenetic regulations (DNA methylation, telomere length) to 'health outcomes' (illness, BMI, waist-hip ratio). The cascade of events linked to stress responses, physiological burden and disease thus needs careful consideration. Currently, some markers are presented as primary mediators (cortisol, DHEA-S, catecholamines), some others as secondary mediators (HDL, glucose level and more generally 'biological risk factors') and some others as tertiary mediators (diseases) (McEwen & Seeman, 1999). Furthermore, some mediators are more variable than others. In particular primary mediators, like cortisol, vary according to circadian rhythm and acute environmental challenges whereas secondary mediators, like HDL, are more stable. For primary indicators, multiple measures are required whereas for secondary or tertiary mediators, one measure may suffice. Furthermore, the total hormone level is not necessarily a good index of the active part of the hormone. In this case, transport proteins (such as CBG for cortisol) and salivary or urine assessment (free cortisol) should be measured. This issue raises general methodological considerations regarding AL score construction from various measures. Moreover this issue also raises questions on the feasibility of collecting such biomarkers in accessible samples like blood, saliva or urine.

Building a score

Considering the two previous points, the question of how to go about summarising, in one single score, information contained from a number of biomarkers is fundamental. In practice an AL score is usually built pragmatically from available data. The most widely used method to build an AL score uses a summary measure representing the number of biomarkers within a high risk percentile defined from the biomarkers' distribution in the studied population (Juster, McEwen & Lupien, 2010). Maybe more critical than questions on how to define 'subclinical' thresholds representative in various populations, this approach is empirical and is in large part not based on a theoretical concept of AL. Consequently, some scores are composed of variables that lead to one physiological system being over-represented versus the others. This is often the case with the cardiovascular or metabolic systems that can be measured through several easily-collected variables (HDL, LDL cholesterol total, blood pressure, glucose and insulin level, waist hip ratio, BMI) whereas HPA axis, sympathetic nervous system, inflammatory and immune systems tend to be represented using one or two variables. By simply summing these variables to build a score, it is likely that the score will be well correlated with cardiovascular diseases and less so with other diseases. It may be possible to weight the score according to the outcome measure of interest. The score would then be composed of the same variables weighted differently according to the disease studied. However, using such an approach raises issues about the capability of such a score to 'truly' measure global physiological wear and tear. Additionally, such a method also raises questions related to the fact that these variables are not independent, some of them being linked by physiological pathways. In consequence how best to take the nature of these different relationships into account in the overall score is an important issue. In response to these questions, more sophisticated methods like recursive partitioning or canonical correlation analyses have been used to

manage weighting and interrelation between biomarkers (Juster et al., 2010). More recently new approaches based on confirmatory factor analysis and structural equation modelling have been proposed which could be particularly relevant to 'capture' the concept of AL (Seeman et al. 2010; Booth, Starr & Deary, 2013; McCaffery, Marsland, Strohacker, Muldoon & Manuck, 2012). These methods, based on the covariation of biomarkers, present several advantages including: the possibility of testing an *a priori* hypothesised model or structure linking biomarkers and physiological systems which is relevant to analyse AL; the construction of AL as a latent variable (metafactor) by modelling shared variance among biological systems which is in accordance with the general idea of wear and tear included in the AL concept; testing factorial invariance which could be useful to test the stability of the AL score in various groups of the population (age, gender); the use of continuous variables; the fact that no assumption on weight is required as the weight of each parameter is defined empirically.

Allostatic load across the life course

Taking a life course approach to studying health raises questions regarding how best to measure wear and tear over the life span. AL is by definition the consequence of a cumulative adaptive response to challenges. Thus this is a dynamic process and therefore its measure should be dynamic as well. Moreover, the question of timing is key. The physiological systems identified to measure AL, and how to measure them, are indeed likely to vary considerably according to age. The physiological responses to stress vary by developmental stage in early life, with sensitive periods of brain development and consequent physiological responses occurring well into late adolescence. Sensitive periods of brain change also occur in older age, and are likely to have an impact on physiological stress reactivity (Lupien, McEwen, Gunnar & Heim, 2009). How to measure early stages of physiological wear and tear at different periods of life as well as differences in sex/ gender stress response each deserve further investigation (Bale, 2011).

The mediating role of AL between socioeconomic position and mortality deserves in-depth examination. Though the link between AL and subsequent health is relatively well studied, not many studies analyse the link between adverse environments (physical, chemical, nutritional, psychosocial) and AL, taking a life course approach. Very recent studies using a life course approach have shown very promising results on the link between socioeconomic position over the life course and AL score (Gruenewald et al., 2012; Gustafsson, Janlert, Theorell, Westerlund & Hammarstrom, 2011; Gustafsson, et al. 2012; Merkin, Karlamangla, Roux, Shrager & Seeman, 2014; Robertson, Popham & Benzeval, 2014). These studies justify that in order to identify mechanisms or causal chains linking environmental challenges, AL and subsequent health, a life course approach is required, particularly if interventions are to be implemented. To study such complex mechanisms, implicating direct and indirect effects of adverse exposures over time necessitates rich longitudinal datasets with long follow-ups. Socioeconomic position being a proxy for various exposures, datasets with large panels of variables on socioeconomic and psychosocial environment are particularly precious to disentangle which aspects contained in socioeconomic position influence both health and AL. Another essential ingredient in these datasets is the inclusion of biological samples, repeatedly collected to represent the dynamic nature of AL.

Conclusion

Here, we consider AL as a useful conceptual tool in measuring the biological effect of embodiment that can play a role in the production of the social gradient of many chronic diseases. Measures of the way people cope with their environment, from early life onwards, offer many possibilities regarding public health interventions both at a societal level by investing in childhood or in social environment, and at an individual level by preventing diseases through behavioural or treatment interventions. Before operationalising AL as a measure of embodiment, a number of issues deserve further exploration. To remain loyal to the theory behind AL we highlight that measures used should be constructed, where possible, to represent multiple biological systems. In order to achieve this, good quality stable biological markers of the different physiological systems are needed, as well as data on the psychosocial and socioeconomic environment. All these questions are therefore conditioned by the availability of such markers in human cohorts. The development and maintenance of these cohorts is

essential, including information on socioeconomic and psychosocial environments over the life course, with multiple biological measures, made at various stages across the life span.

Acknowledgements

All authors contributed to the conceptualisation and design of this paper via dedicated meetings and workshops. Cyrille Delpierre and Michelle Kelly-Irving drafted the manuscript. All authors contributed to the writing of subsequent drafts.

This project received funding from INCa-Cancéropôle GSO N°2012-E18 and is part of the IBISS project supported by funds from The French National Research Agency (ANR-12-DSSA-0004). Funding was used for meetings and workshops but there was no role of the study sponsors in the study design or in the collection, analysis and interpretation of the data or in the writing of the report and in the decision to submit the paper for publication.

References

Adkins, B., Leclerc, C., & Marshall-Clarke, S. (2004). Neonatal adaptive immunity comes of age. *Nature Reviews Immunology, 4*(7), 553-564. http://dx.doi.org/10.1038/nri1394

Bale, T. L. (2011). Sex differences in prenatal epigenetic programming of stress pathways. *Stress, 14*(4), 348-356.

Barabasi, A. L., Gulbahce, N., & Loscalzo, J. (2011). Network medicine: a network-based approach to human disease. *Nature Reviews Genetics, 12*(1), 56-68. http://dx.doi.org/10.1038/nrg2918

Barker, D. J. (1990). The fetal and infant origins of adult disease. *British Medical Journal, 301*(6761), 1111. http://dx.doi.org/10.1136/bmj.301.6761.1111

Booth, T., Starr, J. M., & Deary, I. (2013). Modeling multisystem biological risk in later life: allostatic load in the Lothian birth cohort study 1936. *American Journal of Human Biology, 25*(4), 538-543. http://dx.doi.org/10.1002/ajhb.22406

Danese, A., & McEwen, B. S. (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology & Behavior, 106*(1), 29-39. http://dx.doi.org/10.1016/j.physbeh.2011.08.019

Danese, A., Moffitt, T. E., Harrington, H., Milne, B. J., Polanczyk, G., Pariante, C. M., Poulton, R., Caspi, A. (2009). Adverse childhood experiences and adult risk factors for age-related disease: depression, inflammation, and clustering of metabolic risk markers. *Archives of Pediatric & Adolescent Medicine, 163*(12), 1135-1143. http://dx.doi.org/10.1001/archpediatrics.2009.214

Gogtay, N., Giedd, J. N., Lusk, L., Hayashi, K. M., Greenstein, D., Vaituzis, A. C., Nugent, T. F., 3rd Herman, D. H., Clasen, L. S., Toga, A. W., Rapoport, J. L., Thompson, P. M. (2004). Dynamic mapping of human cortical development during childhood through early adulthood. *Proceedings of the National Academy of Sciences USA, 101*(21), 8174-8179. http://dx.doi.org/10.1073/pnas.0402680101

Gruenewald, T. L., Karlamangla, A. S., Hu, P., Stein-Merkin, S., Crandall, C., Koretz, B., & Seeman, T. E. (2012). History of socioeconomic disadvantage and allostatic load in later life. *Social Science & Medicine, 74*(1), 75-83. http://dx.doi.org/10.1016/j.socscimed.2011.09.037

Gustafsson, P. E., Janlert, U., Theorell, T., Westerlund, H., & Hammarstrom, A. (2011). Socioeconomic status over the life course and allostatic load in adulthood: results from the Northern Swedish Cohort. *Journal of Epidemiology & Community Health, 65*(11), 986-992. http://dx.doi.org/10.1136/jech.2010.108332

Gustafsson, P. E., Janlert, U., Theorell, T., Westerlund, H., & Hammarstrom, A. (2012). Social and material adversity from adolescence to adulthood and allostatic load in middle-aged women and men: results from the Northern Swedish Cohort. *Annals of Behavioral Medicine, 43*(1), 117-128. http://dx.doi.org/10.1007/s12160-011-9309-6

Hertzman, C. (1999). The biological embedding of early experience and its effects on health in adulthood. *Annals of the New York Academy of Sciences, 896*, 85-95. http://dx.doi.org/10.1111/j.1749-6632.1999.tb08107.x

Juster, R. P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience & Biobehavioral Reviews, 35*(1), 2-16. http://dx.doi.org/10.1016/j.neubiorev.2009.10.002

Kelly-Irving, M., Lepage, B., Dedieu, D., Bartley, M., Blane, D., Grosclaude, P., Lang, T., Delpierre, C. (2013a). Adverse childhood experiences and premature all-cause mortality. *Euopean Journal of Epidemiology, 28*(9), 721-734. http://dx.doi.org/10.1007/s10654-013-9832-9

Kelly-Irving, M., Lepage, B., Dedieu, D., Lacey, R., Cable, N., Bartley, M., Blane, D., Grosclaude, P., Lang, T., Delpierre, C. (2013b). Childhood adversity as a risk for cancer: findings from the 1958 British birth cohort study. *BMC Public Health, 13*(1), 767. http://dx.doi.org/10.1186/1471-2458-13-767

Krieger, N. (2005). Embodiment: a conceptual glossary for epidemiology. *Journal of Epidemiology & Community Health, 59*(5), 350-355. http://dx.doi.org/10.1136/jech.2004.024562

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience, 10*(6), 434-445. http://dx.doi.org/10.1038/nrn2639

McCaffery, J. M., Marsland, A. L., Strohacker, K., Muldoon, M. F., & Manuck, S. B. (2012). Factor structure underlying components of allostatic load. *PLoS One, 7*(10), e47246. http://dx.doi.org/10.1371/journal.pone.0047246

McEwen, B. S., & Seeman, T. (1999). Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. *Annals of the New York Academy of Sciences, 896*, 30-47. http://dx.doi.org/10.1111/j.1749-6632.1999.tb08103.x

McEwen, B. S., & Stellar, E. (1993). Stress and the individual. Mechanisms leading to disease. *Archives of Internal Medicine, 153*(18), 2093-2101. http://dx.doi.org/10.1001/archinte.1993.00410180039004

Merkin, S. S., Karlamangla, A., Roux, A. V., Shrager, S., & Seeman, T. E. (2014). Life course socioeconomic status and longitudinal accumulation of allostatic load in adulthood: multi-ethnic study of atherosclerosis. *American Journal of Public Health, 104*(4), e48-55. http://dx.doi.org/10.2105/AJPH.2013.301841

Robertson, T., Popham, F., & Benzeval, M. (2014). Socioeconomic position across the lifecourse & allostatic load: data from the West of Scotland Twenty-07 cohort study. *BMC Public Health, 14*, 184. http://dx.doi.org/10.1186/1471-2458-14-184

Seeman, T., Epel, E., Gruenewald, T., Karlamangla, A., & McEwen, B. S. (2010). Socio-economic differentials in peripheral biology: cumulative allostatic load. *Annals of the New York Academy of Sciences, 1186*, 223-239. http://dx.doi.org/10.1111/j.1749-6632.2009.05341.x

Seeman, T., Gruenewald, T., Karlamangla, A., Sidney, S., Liu, K., McEwen, B., & Schwartz, J. (2010). Modeling multisystem biological risk in young adults: The Coronary Artery Risk Development in Young Adults Study. *American Journal of Human Biology, 22*(4), 463-472. http://dx.doi.org/10.1002/ajhb.21018

Seeman, T. E., McEwen, B. S., Rowe, J. W., & Singer, B. H. (2001). Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging. *Proceedings of the National Academy of Sciences USA, 98*(8), 4770-4775. http://dx.doi.org/10.1073/pnas.081072698

Seeman, T. E., Singer, B. H., Rowe, J. W., Horwitz, R. I., & McEwen, B. S. (1997). Price of adaptation--allostatic load and its health consequences. MacArthur studies of successful aging. *Archives of Internal Medicine, 157*(19), 2259-2268. http://dx.doi.org/10.1001/archinte.1997.00440400111013

Shonkoff, J. P., & Garner, A. S. (2012). The lifelong effects of early childhood adversity and toxic stress. *Pediatrics, 129*(1), e232-246. http://dx.doi.org/10.1542/peds.2011-2663

Sterling, P., & Eyer, J. (1988). Allostasis: a new paradigm to explain arousal pathology. In S. Fisher & J. Reason, (Eds.), *Handbook of life stress, cognition and health.* (pp. 629-649). New York: John Wiley & Sons.

Commentary by

Linn Getz Norwegian University of Science and Technology, Norway
linn.getz@ntnu.no
Margret Olafia Tomasdottir University of Iceland, and Primary Health Care of the Capital Area, Iceland

Introduction

The paper by Delpierre and colleagues on 'Allostatic load as a measure of social embodiment' offers an interesting and timely discussion of allostatic load as a mediating mechanism of embodiment, a way to scientifically conceptualise the interrelatedness of life-time experiences and human health. From an epidemiological perspective the authors see a need to operationalise allostatic load in a consensual manner for future application across different populations.

The general practice research group to which we belong has for years taken an interest in allostatic load. The presented connection between allostatic load and the phenomenon embodiment (Krieger, 2005) is highly concordant with our thinking (Getz, Kirkengen, & Ulvestad, 2011; Kirkengen, 2001, 2010; Kirkengen et al., 2015; Kirkengen & Thornquist, 2012; Mjolstad, Kirkengen, Getz, & Hetlevik, 2013; Thornquist & Kirkengen, 2015; Tomasdottir et al., 2014; Tomasdottir et al., 2015; Vogt, Ulvestad, Eriksen, & Getz, 2014). The interrelatedness of human biology and biography has long been evident to experienced doctors in general practice (GPs) who encounter individuals over time across varying circumstances and stages of life (Kirkengen, 2005). Until recently it has been very hard to think and communicate professionally about the topic. We have simply been short of an adequate, non-dualistic terminology. The concepts *embodiment* and *allostatic load* are now gaining momentum as conceptual tools to help establish and consolidate new and relevant medical knowledge. Much work will however be needed before these concepts are likely to influence significantly the mainstream of medical thought and practice.

We support the authors who see a need to further develop 'allostatic load' as an empirical construct. In the initial, tentative phases of allostatic load research (including our own) somewhat differing variables and algorithms have indeed been applied, to a large extent reflecting practical availability of data in each case. Theoretical consensus and empirical rigor are now needed to consolidate and advance this important field.

But we see a lot more to the concept allostatic load than a quantifiable score. We see it as a potential keystone in coherent, integrative (in the sense of non-dualistic) thinking in future medicine (Tomasdottir et al., 2015; McEwen & Getz, 2013). From this perspective, we argue that the concept allostatic load needs more than algorithmic refinement. We must also tend to it as a philosophical concept, and to its associated metaphors.

Before elaborating further on these thoughts we comment on two concrete arguments found in the index paper. Firstly, we will consider the vision of a finite allostatic load score (AL score) in view of ongoing mega-projects in systems biology, captured by the keywords '–omics' and 'big data'. Secondly, we will comment on the existing level of knowledge pertaining to the social gradient in health, and the current implications of this knowledge.

Building an allostatic load score in the age of systems biology

The paper for debate asks which aspects of human physiology ought to be included in the AL score, and whether different algorithms might be useful, depending on the outcome(s) in question. Looking at these questions from a different angle, it seems likely that the search for finite AL algorithm(s) will soon be located in a whirlpool of biological data downstream of techno-scientific megaprojects such as 'the virtual physiological human' (http://www.vph-institute.org/) and 'the 100K wellness project' (www.systemsbiology.org/research/100k-wellness-project/). These prestigious projects aim to mathematically model the human body as a complex system, and are as such in full concordance with the approach of allostasis research. The systems biology projects, however, are not geared towards demarcated, finite algorithms. Their approach is based on high-throughput analysis of 'big data' involving billions of datapoints for each individual attempting to monitor even the faintest

reflections from the individual's norms (Chen et al., 2012; Hood, Lovejoy, & Price, 2015; Hood & Tian, 2012).

The term allostatic load has recently started to appear in association with '-omics' projects (Ghini, Saccenti, Tenori, Assfalg, & Luchinat, 2015) and the idea of applying *systems biology to medicine* has definitely been launched (Boissel, Auffray, Noble, Hood, & Boissel, 2015; Bousquet et al., 2011). In light of this development we wonder how long allostasis research will be based on parameters of the type currently involved in AL scores, e.g. as outlined by McEwen (2015). The new systems biology projects aim to elicit data on all conceivable '–omics levels' of the human organism, from genomics via transcriptomics and metabolomics 'upwards' in the direction of clinical and even behavioural data. From a relative distance we assume that future evaluations of allostatic load will involve '–omics' data/patterns. The optimal way of characterising 'wear and tear' in an organism might in fact evolve as new candidate markers/patterns surface from the hi-throughput analyses. The AL score thereby becomes 'a moving target'.

As we see it the 'billion datapoints' scenario of systems biology represents both an opportunity and a threat to the idea of allostasis as a keystone concept in medical thought and practice. In this state of ambivalence we think that what matters most is to safeguard the philosophical (conceptual) meaning of allostatic load in a way that makes it relatively inert in the face of techno-scientific and political trends and commercial pressure (Diamandis, 2015; James, 2014; Karlsen & Strand, 2009).

Current knowledge – an imperative for action

Our second immediate response to the index paper relates to the existing level of knowledge about the social gradient in health. From the perspective of scientific incompleteness we agree that there is a lot we still do not know and would like to find out. However, we argue that the overall picture is already quite clear, and this fact must not be understated (Forssen, Meland, Hetlevik, & Strand, 2011; Heath, 2010; Marmot, 2010). We have access to hundreds of high quality publications from epidemiology, clinical cohorts, the basic sciences, and neuroimaging, as well as the social sciences and other sources. The term 'the biology of disadvantage' has been used to sum up our existing insight in how social adversity undermines human health (McEwen & Getz, 2013). In the post-genomic era (Hayden, 2010) it has become easier to promote and stimulate knowledge about the impact of social and relational adversity on health across various disciplines. To illustrate the emergence of new and fruitful collaborations we note three publications that emerged independently of each other in 1998. The first introduced the physiological concept allostatic load to a broad medical audience (McEwen, 1998). The second presented the Adverse Childhood Experiences Study, based on clinical-epidemiological data collected by Kaiser Permanente in Southern California (Felitti et al., 1998). The third was a qualitative medical study rooted in phenomenology, later published as *Inscribed bodies - the health impact of childhood sexual abuse* (Kirkengen, 2001). Since then an immense amount of concordant evidence on the detrimental impact of early life adversity has become available (Getz et al., 2011; Kirkengen, 2010). In our research unit – the General Practice Research Unit at the Department of Public Health and General Practice, Norwegian University of Science and Technology – we apply insight from these different perspectives to deal with the conundrum of multimorbidity (Tomasdottir et al., 2014, 2015). So while agreeing that more research would strengthen existing knowledge, we acknowledge that it is possible to pave a good way for public health and primary care with the knowledge we already possess.

Allostatic load and human stories

We observe how the discourse related to allostatic load has started to dismantle walls between traditional "knowledge silos" and unify the perspectives of researchers/clinicians from various areas, including neuroscientists, endocrinologists, immunologists, psychologists, epidemiologists, public health and primary care researchers/practitioners. We believe such "breakthroughs" are facilitated by the fact that allostatic load can be addressed both in everyday metaphorical language ("wear and tear") and as a scientific-empirical construct (Heath, 2013). This seems to draw the individual experts' attention in the same direction, away from fragmented sub-systems in direction of the whole and undividable, living, striving organism . In the context of medicine,

and especially primary health care, the organism in question can best described as *a person*, with reference to physician-philosopher Eric Cassell (E. J. Cassell, 2010).

Already in 1992 Cassell (1992) pointed out that (personal) human agency must necessarily involve the whole human being, all the way down to the mitochondria . Today the basic sciences have reached a point where we can view both Cassell's argument and the mitochondria in terms of allostatic load (Picard, Juster, & McEwen, 2014). This convergence of philosophical and physiological perspectives opens new perspectives on narrative in medicine and the medical relevance of attending to human stories in the clinical encounter (Behforouz, Drain, & Rhatigan, 2014; McEwen & Getz, 2013; Scannell, 2012). It is hardly a coincidence that Nancy Krieger's (2005) erudite discussion of embodiment, the departing point of the index paper, revolves around the term "story", as does anthropologist and systems thinker Gregory Bateson's seminal work *Mind and Nature – a necessary unity* (Bateson, 1979): «But I come with stories – not just a supply of stories to deliver to the analyst but stories built into my very being».

Reflecting on human stories in the light of allostatic load we should keep in mind that such narratives evolve around the past, the present and, not the least, an imagined future. We now possess considerable knowledge about the biological processes by which past and present experiences become embodied. Schulkin (2011) reminds us that yet another essential determinant of a person's allostatic load lies in the person's own view of the future, the anticipation of that which has yet to come.

The metaphors of allostasis: from 'wear and tear' to 'gains and drains'?

Based on the metaphor "wear and tear," the concept *allostatic load* can effectively accommodate knowledge pertaining to the pathogenetic impact of socioeconomic disadvantage and adverse lifetime experiences (Tomasdottir et al., 2014). However, between the lines of the allostasis literature we also encounter considerations pertaining to salutogenetic factors which promote and uphold health. An explicit focus on resilience can be found in recent key publications about allostasis (Ghini et al., 2015; Karatsoreos & McEwen, 2013; McEwen, Gray, & Nasca, 2015). Consequently, we suggest that a metaphorical expression of the fundamental idea of *allostasis* should involve both detrimental (”draining”) and health promoting (”gaining”) phenomena (Kirkengen, 2010; Tomasdottir et al., 2014). Depiction of an existential balance between drains (adversity) and gains (buffering support) is in fact needed to grasp the very essence of the terms ”positive,” ”tolerable” and ”toxic stress” which have become tightly connected to the concept of allostatic load (Shonkoff, Boyce & McEwen, 2009. See also http://developingchild.harvard.edu/). In order to further refine the metaphors of allostasis it is also important to keep underlining the fundamental difference between an *exposure* (objectively categorized) and an *experience* (subjectively lived) (Kirkengen & Thornquist, 2012; Seery, 2011; Tomasdottir et al., 2015; Ulvestad, 2012; Vie, Hufthammer, Holmen, Meland, & Breidablik, 2014; Waller, 2015).

Closing remark

Clinical evaluation of allostatic load might obviously involve a quantifiable score. Although not explicitly defined as such, most risk factors currently monitored in primary health care represent allostatic variables (McEwen, 2015), including blood pressure, lipid profile, glucose metabolism and body composition. As we have discussed, it will be interesting to see what happens to the AL score in the era of systems medicine based on big data. But whatever algorithms are used, it takes more than de-contextualised measurements to appreciate the balance between gaining and draining factors in a clinically meaningful and ethically responsible way (Evans, 2003; Juster et al., 2015; Repetti, Robles, & Reynolds, 2011; Upchurch et al., 2015). From the clinical viewpoint we might speak of a capacity for integrative perception that might at some point become conceptually linked to professional empathy (Ferrari, 2014). The word *gestalt* comes to mind in relation to the perception of another person's allostatic balance, in the sense of being-in-the-world as an embodied *person* (Cassell, 2010). We are indeed speaking of "a structure, configuration, or pattern of physical, biological, or psychological phenomena so integrated as to constitute a functional unit with properties not derivable by summation of its parts" (definition of *gestalt* in Merriam-Webster dictionary, acc. June 30, 2015).

References

Bateson, G. (1979). *Mind and nature: a necessary unity* (p. 13 in edition of 2002). Bantam Books.

Behforouz, H. L., Drain, P. K., & Rhatigan, J. J. (2014). Rethinking the social history. *New England Journal of Medicine, 371*(14), 1277-1279. http://dx.doi.org/10.1056/NEJMp1404846

Boissel, J. P., Auffray, C., Noble, D., Hood, L., & Boissel, F. H. (2015). Bridging systems medicine and patient needs. *CPT: Pharmacometrics and Systems Pharmacoogyl, 4*(3), e00026. http://dx.doi.org/10.1002/psp4.26

Bousquet, J., Anto, J. M., Sterk, P. J., Adcock, I. M., Chung, K. F., Roca, J., Agusti, A., Brightling, C., Cambon-Thomsen, A., Cesario, A., Abdelhak, S., Antonarakis, S.E., Avignon, A., Ballabio, A., Baraldi, E., Baranov, A., Bieber ,T., Bockaert, J., Brahmachari, S., Brambilla, C., Bringer, J., Dauzat, M., Ernberg, I., Fabbri, L., Froguel, P., Galas, D., Gojobori, T., Hunter, P., Jorgensen, C., Kauffmann, F., Kourilsky, P., Kowalski, ML., Lancet, D., Pen, CL., Mallet, J., Mayosi, B., Mercier, J., Metspalu, A., Nadeau, J.H., Ninot, G., Noble, D., Oztürk, M., Palkonen, S., Préfaut, C., Rabe, K., Renard, E., Roberts, R.G., Samolinski, B., Schünemann, H.J., Simon, H.U., Soares, M.B., Superti-Furga, G., Tegner, J., Verjovski-Almeida, S., Wellstead, P., Wolkenhauer, O., Wouters, E., Balling, R., Brookes, A.J., Charron, D., Pison, C., Chen, Z., Hood, L., & Auffray, C. (2011). Systems medicine and integrated care to combat chronic noncommunicable diseases. *Genome Medicine, 3*(7), 43. http://dx.doi.org/10.1186/gm259

Cassell, E. J. (1992). The body of the future. In D. Leder (Ed.), *The body in medical thought and practice* (pp. 233-249). New York: Springer. http://dx.doi.org/10.1007/978-94-015-7924-7_14

Cassell, E. J. (2010). The person in medicine. *International Journal of Integrated Care, 10 Suppl*, e019. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/20228916

Chen, R., Mias, G. I., Li-Pook-Than, J., Jiang, L., Lam, H. Y., Chen, R., Miriami, E., Karczewski, K.J., Hariharan, M., Dewey, F.E., Cheng, Y., Clark, M.J., Im, H., Habegger, L., Balasubramanian, S., O'Huallachain, M., Dudley, J.T., Hillenmeyer, S., Haraksingh, R., Sharon, D., Euskirchen, G., Lacroute, P., Bettinger, K., Boyle, A.P., Kasowski, M., Grubert, F., Seki, S., Garcia, M., Whirl-Carrillo, M., Gallardo, M., Blasco, MA., Greenberg P.L., Snyder, P., Klein, T.E., Altman, R.B., Butte, A.J., Ashley, E.A., Gerstein, M., Nadeau, K.C., Tang, H., & Snyder, M. (2012). Personal omics profiling reveals dynamic molecular and medical phenotypes. *Cell, 148*(6), 1293-1307. http://dx.doi.org/10.1016/j.cell.2012.02.009

Diamandis, E. P. (2015). The Hundred Person Wellness Project and Google's Baseline Study: medical revolution or unnecessary and potentially harmful over-testing? *Biomedcentral (BMC) Medicine, 13*, 5. http://dx.doi.org/10.1186/s12916-014-0239-6

Evans, G. W. (2003). A multimethodological analysis of cumulative risk and allostatic load among rural children. *Developmental Psychology, 39*(5), 924-933. http://dx.doi.org/10.1037/0012-1649.39.5.924 Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12952404

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., Koss, M.P., & Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine, 14*(4), 245-258. http://dx.doi.org/10.1016/s0749-3797(98)00017-8 Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9635069

Ferrari, P. F. (2014). The neuroscience of social relations. A comparative-based approach to empathy and to the capacity of evaluating others' action value. *Behaviour, 151*(2-3), 297-313. http://dx.doi.org/10.1163/1568539X-00003152

Forssen, A., Meland, E., Hetlevik, I., & Strand, R. (2011). Rethinking scientific responsibility. *Journal of Medical Ethics, 37*(5), 299-302. http://dx.doi.org/10.1136/jme.2010.038828

Getz, L., Kirkengen, A. L., & Ulvestad, E. (2011). The human biology--saturated with experience. *Tidsskrift for den Norske Laegeforening, 131*(7), 683-687. http://dx.doi.org/10.4045/tidsskr.10.0874

Ghini, V., Saccenti, E., Tenori, L., Assfalg, M., & Luchinat, C. (2015). Allostasis and resilience of the human individual metabolic phenotype. *Journal of Proteome Research, 14*(7), 2951-2962. http://dx.doi.org/10.1021/acs.jproteome.5b00275

Hayden, E. C. (2010). Human genome at ten: life is complicated. *Nature Biotechnology, 464*, 664-667. doi:doi:10.1038/464664a

Heath, I. (2010). Crocodile tears for health inequality. *British Medical Journal, 340*, c2970. http://dx.doi.org/10.1038/464664a

Heath, I. (2013). Complexity, uncertainty and mess as the links between science and the humanities in health care (pp. 19-26). In J. Sturmberg & C. Martin (Eds.), *Handbook of systems and complexity in health*. New York: Springer. http://dx.doi.org/10.1007/978-1-4614-4998-0_2

Hood, L., Lovejoy, J. C., & Price, N. D. (2015). Integrating big data and actionable health coaching to optimize wellness. *Biomedcentral* (*BMC) Medicine, 13*, 4. http://dx.doi.org/10.1186/s12916-014-0238-7

Hood, L., & Tian, Q. (2012). Systems approaches to biology and disease enable translational systems medicine. *Genomics Proteomics Bioinformatics, 10*(4), 181-185. http://dx.doi.org/10.1016/j.gpb.2012.08.004

James, J. E. (2014). Personalised medicine, disease prevention, and the inverse care law: more harm than benefit? *European Journal of Epidemiology, 29*(6), 383-390. http://dx.doi.org/10.1007/s10654-014-9898-z

Juster, R. P., Hatzenbuehler, M. L., Mendrek, A., Pfaus, J. G., Smith, N. G., Johnson, P. J., . . . Pruessner, J. C. (2015). Sexual orientation modulates endocrine stress reactivity. *Biological Psychiatry, 77*(7), 668-676. http://dx.doi.org/10.1016/j.biopsych.2014.08.013

Karatsoreos, I. N., & McEwen, B. S. (2013). Annual Research Review: the neurobiology and physiology of resilience and adaptation across the life course. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 54*(4), 337-347. http://dx.doi.org/10.1111/jcpp.12054

Karlsen, J., & Strand, R. (2009). The annexation of life. The biopolitics of industrial biology. In J. Solbakk, S. Holm, & B. Hofmann (Eds.), *The ethics of research biobanking* (pp. 315-329). Dordrecht: Springer. http://dx.doi.org/10.1007/978-0-387-93872-1_20

Kirkengen, A. L. (2001). *Inscribed bodies. The health impact of child sexual abuse.* Dordrecht: Kluwer. http://dx.doi.org/10.1007/978-94-017-1886-8

Kirkengen, A. L. (2005). Encountering particulars: a life in medicine. *The Permanente Journal, 9*(3), 19-22. http://dx.doi.org/10.7812/TPP/04-157 Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/22811623

Kirkengen, A. L. (2010). *The lived experience of violation: how abused children become unhealthy adults*. Bucharest: Zeta Books. http://dx.doi.org/10.7761/9789731997476

Kirkengen, A. L., Ekeland, T. J., Getz, L., Hetlevik, I., Schei, E., Ulvestad, E., & Vetlesen, A. J. (2015). Medicine's perception of reality - a split picture: critical reflections on apparent anomalies within the biomedical theory of science. *Journal of Evaluation in Clinical Practice*. http://dx.doi.org/10.1111/jep.12369

Kirkengen, A. L., & Thornquist, E. (2012). The lived body as a medical topic: an argument for an ethically informed epistemology. *Journal of Evaluation in Clinical Practice, 18*(5), 1095-1101. http://dx.doi.org/10.1111/j.1365-2753.2012.01925.x

Krieger, N. (2005). Embodiment: a conceptual glossary for epidemiology. *Journal of Epidemiology and Community Health, 59*(5), 350-355. http://dx.doi.org/10.1136/jech.2004.024562

Marmot, M. (2010). *The Marmot review final report: fair society, healthy lives* Retrieved from www.instituteofhealthequity.org/.../fair-society-healthy-lives-the-marmot

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine, 338*(3), 171-179. http://dx.doi.org/10.1056/NEJM199801153380307

McEwen, B. S. (2015). Biomarkers for assessing population and individual health and disease related to stress and adaptation. *Metabolism: Clinical and Experimental, 64*(3 Suppl 1), S2-S10. http://dx.doi.org/10.1016/j.metabol.2014.10.029

McEwen, B. S., & Getz, L. (2013). Lifetime experiences, the brain and personalized medicine: an integrative perspective. *Metabolism: Clinical and Experimental, 62 Suppl 1*, S20-26. http://dx.doi.org/10.1016/j.metabol.2012.08.020

McEwen, B. S., Gray, J., & Nasca, C. (2015). Recognizing resilience:learning from the effects of stress on the brain. *Neurobiology of Stress, 1*, 1-11. http://dx.doi.org/10.1016/j.ynstr.2014.09.001

Mjolstad, B. P., Kirkengen, A. L., Getz, L., & Hetlevik, I. (2013). Standardization meets stories: contrasting perspectives on the needs of frail individuals at a rehabilitation unit. *International Journal of Qualitative Studies on Health and Well-being, 8*, 21498. http://dx.doi.org/10.3402/qhw.v8i0.21498

Picard, M., Juster, R. P., & McEwen, B. S. (2014). Mitochondrial allostatic load puts the 'gluc' back in glucocorticoids. *Nature Reviews: Endocrinology, 10*(5), 303-310. http://dx.doi.org/10.1038/nrendo.2014.22

Repetti, R. L., Robles, T. F., & Reynolds, B. (2011). Allostatic processes in the family. *Development and Psychopathology, 23*(3), 921-938. http://dx.doi.org/10.1017/S095457941100040X

Scannell, K. (2012). A case of baffling fatigue with a spectral twist. *The Permanente Journal, 16*(1), 51-54. http://dx.doi.org/10.7812/TPP/11-107B Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/22529760

Schulkin, J. (2011). Social allostasis: anticipatory regulation of the internal milieu. *Frontiers in Evolutionary Neuroscience, 2*, 111. http://dx.doi.org/10.3389/fnevo.2010.00111

Seery, M. D. (2011). Challenge or threat? Cardiovascular indexes of resilience and vulnerability to potential stress in humans. *Neuroscience and Biobehavioral Reviews, 35*(7), 1603-1610. http://dx.doi.org/10.1016/j.neubiorev.2011.03.003

Shonkoff, J. P., Boyce, W. T., & McEwen, B. S. (2009). Neuroscience, molecular biology, and the childhood roots of health disparities: building a new framework for health promotion and disease prevention. *Journal of the American Medical Association, 301*(21), 2252-2259. http://dx.doi.org/10.1001/jama.2009.754

Thornquist, E., & Kirkengen, A. L. (2015). The quantified self: closing the gap between general knowledge and particular case? *Journal of Evaluation in Clinical Practice, 21*(3), 398-403. http://dx.doi.org/10.1111/jep.12239

Tomasdottir, M. O., Getz, L., Sigurdsson, J. A., Petursson, H., Kirkengen, A. L., Krokstad, S., McEwen, B.& Hetlevik, I. (2014). Co- and multimorbidity patterns in an unselected Norwegian population: cross-sectional analysis based on the HUNT Study and theoretical reflections concerning basic medical models. *European Journal of Person Centered Healthcare, 2*(3), 335-345.

Tomasdottir, M. O., Sigurdsson, J. A., Petursson, H., Kirkengen, A. L., Krokstad, S., McEwen, B., Hetlevik, I& Getz, L. (2015). Self reported childhood difficulties, adult multimorbidity and allostatic load. A cross-sectional analysis of the Norwegian HUNT Study. *PloS One, 10*(6). http://dx.doi.org/10.1371/journal.pone.0130591

Ulvestad, E. (2012). Psychoneuroimmunology: the experiential dimension. *Methods in Molecular Biology, 934*, 21-37. http://dx.doi.org/10.1007/978-1-62703-071-7_2

Upchurch, D. M., Stein, J., Greendale, G. A., Chyu, L., Tseng, C. H., Huang, M. H., Lewis, T.T., Kravitz, H.M., & Seeman, T. (2015). A longitudinal investigation of race, socioeconomic status, and psychosocial mediators of allostatic load in midlife women: findings from the Study of Women's Health Across the Nation. *Psychosomatic Medicine, 77*(4), 402-412. http://dx.doi.org/10.1097/PSY.0000000000000175

Vie, T. L., Hufthammer, K. O., Holmen, T. L., Meland, E., & Breidablik, H. J. (2014). Is self-rated health a stable and predictive factor for allostatic load in early adulthood? Findings from the Nord Trondelag Health Study (HUNT). *Social Science and Medicine, 117*, 1-9. http://dx.doi.org/10.1016/j.socscimed.2014.07.019

Vogt, H., Ulvestad, E., Eriksen, T. E., & Getz, L. (2014). Getting personal: can systems medicine integrate scientific and humanistic conceptions of the patient? *Journal of Evaluation in Clinical Practice, 20*(6), 942-952. http://dx.doi.org/10.1111/jep.12251

Waller, G. (2015). Self-rated health in general practice: a plea for subjectivity. *British Journal of General Practice, 65*(632), 110-111. http://dx.doi.org/10.3399/bjgp15X683833

Commentary by

Tony Robertson University of Stirling
tony.robertson@stir.ac.uk

Introduction

In this edition of the journal Delpierre et al. open a discussion on the use of the allostatic load concept as means to measure the term 'embodiment' (also referred to as 'biological embedding'), essentially how our cultural, social and economic circumstances 'get under the skin' to eventually damage our physiological systems and play a role in disease development (Adler & Ostrove, 1999). As described by Delpierre and colleagues the allostatic load concept has a long history starting in the late 1980s (Sterling & Eyer, 1988), but it truly came into being as a concept and research tool a decade later with the merging of the theory and a practical score (McEwen, 1998; Seeman, McEwen, Rowe, & Singer, 2001; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997). Delpierre and colleagues summarise the concept and operationalisation of allostatic load, including its strengths, weaknesses and some future considerations, eloquently enough to avoid unnecessary repetition here. However, there are three points linked to those issues raised that I would like to discuss further.

Gaining credibility

The use of concepts such as allostatic load to try and better understand how the environments we live in can affect our physiology and health falls under a holistic approach, in contrast to the more reductionist approach often sought in epidemiology. While the reductionist approach has great value, especially in trying to elucidate causal mechanisms underpinned by theory and biological plausibility, this approach can feel somewhat incongruous given the complex milieu in which we live our day-to-day lives. In addition, given the strong evidence for almost all chronic diseases being socially patterned and following a social gradient (those with lower socioeconomic position having poorer health), the concept of common biological pathways, as offered with allostatic load, in helping explain this patterning is enticing (Adams & White, 2004; Robertson, Benzeval, Whitley, & Popham, 2015). However, in the pursuit of a better understanding of the 'black box' that links our socioeconomic circumstances and our health, this embodiment/embedding/common biological pathways approach, as measured by allostatic load, introduces a type of black box itself. Are we simply combining individual biomarkers that are easy to measure and available together with no strong theory for linking them? How do we intervene at social and healthcare levels to reduce damage across multiple physiological pathways? Is measuring a patient's allostatic load any more helpful than the seemingly ill-fated NHS Health Checks (Capewell, McCartney, & Holland, 2015), or simply more of the same?

What is clear, and of greatest value in getting wider support for the concept, is the evidence that supports allostatic load as a better predictor of morbidity and mortality as compared with the individual biomarkers that comprise the score (Borrell & Crawford, 2011; Duru, Harawa, Kermah & Norris, 2012; Gruenewald, Seeman, Ryff, Karlamangla & Singer, 2006; Hwang et al., 2014; Karlamangla, Singer & Seeman, 2006; Seeman et al., 2004). Recent analyses found that allostatic load shows similar socioeconomic patterning to chronic disease outcomes, including across the life course, with childhood and adolescence/early adulthood representing particularly sensitive periods for poorer socioeconomic circumstances impacting on allostatic load (Gruenewald et al., 2012; Gustafsson, Janlert, Theorell, Westerlund & Hammarstrom, 2012; Gustafsson et al., 2014; Robertson, Popham & Benzeval, 2014). Furthermore, the association between socioeconomic position and allostatic load appears to be largely mediated by material factors (e.g. income, ownership of goods), but not behavioural and psychological factors (Robertson et al., 2015). This indicates that policies and programmes targeted at more downstream factors (such as health behaviours) may have minimal returns in reducing health and physiological inequalities, as shown for morbidity and mortality (Acheson, 1998; Adler & Stewart, 2010; Macintyre, 2007; Marmot, 2010; Marmot, Friel, Bell, Houweling & Taylor, 2008; Scott et al., 2013). As Delpierre and colleagues discuss, it is through this type of evidence, supported by more multi-disciplinary, longitudinal and life course research that also

incorporates causal inference, that the allostatic load concept will not only gain support, but will also be challenged further and naturally improved also.

Biological ageing: A competing or complementary concept?

Many of the ideas and theoretical pathways linking allostatic load and embodiment discussed by Delpierre and colleagues and earlier in this commentary can also be represented by another common biological pathway – biological ageing. This is "the incremental, universal, and intrinsic degeneration of physical and cognitive functioning and the ability of the body to meet the physiological demands that occur with increasing chronologic age" (Robertson et al., 2013). However, the rate at which this ageing occurs will differ given the (socioeconomic) circumstances in which we live. Increased exposures to physical and psychological insults, along with more unhealthy behaviours, have the potential to increase cellular and genomic damage, thereby accelerating biological ageing (Adams & White, 2004). People in more disadvantaged circumstances, where these insults are more prevalent (Adler & Stewart, 2010), would therefore be expected to be 'biologically' older than their more affluent counterparts of the same chronological age (Robertson et al., 2012). Like the allostatic load concept, identifying biomarkers of ageing that can completely encompass the theory has proved difficult and there remain several questions over how biomarkers could and should be combined (Der et al., 2012). The most promising marker of biological ageing to date has been white blood cell telomere length. Telomeres are protective structures present at the ends of chromosomes that typically erode over time to protect against irreversible chromosomal damage, so that their length is a potential predictor of biological ageing (de Lange, 2002). Therefore, this represents accumulated damage over time that goes across large parts, if not the whole, of the body and is strongly influenced by social and economic circumstances and particularly the stress response. Sound familiar?

It has been proposed that markers such as telomere length are simply alternatives to the allostatic load model currently used (multiple physiological markers linked to health conditions in middle and later-ages), especially at younger ages (Theall, Brett, Shirtcliff, Dunn & Drury, 2013). Biomarkers of ageing have been defined as biological measures that "either alone or in some multivariate composite will, in the absence of disease, better predict functional capacity at some late age than will chronological age" (Baker & Sprott, 1988). Allostatic load could claim to be such a marker, although it has not yet been tested in such a fashion as telomere length (Der et al., 2012). Alternatively, would adding measures such as telomere length to the allostatic load construct add some predictive power over the current operationalisation? Again, this is a feature which has not been explored. Finally, is biological ageing/telomere length more of an outcome of allostatic load and somewhat further down the causal chain? In my opinion, this is difficult to answer given current data (see below), but both allostatic load and biological ageing incorporate what can be considered primary (e.g. cortisol vs. oxidative stress) and secondary (e.g. blood pressure vs. telomere length) physiological markers. In addition, our biological systems are active and dynamic, potentially being responsive to changes in our environments and repairing themselves to some degree (Epel, 2012). Hence, there is not really an end-point where one could say someone has reached, for example, allostatic *overload* and that could be considered a true outcome. So, where do we go from here?

Bio-social collaborations

The emergence of this field linking the biological and the social has grown over the last twenty years, but especially over the last decade, with the increasing inclusion of biomarkers in many large, population-based health and social surveys. This growth in collecting simultaneous biological and social data, longitudinally and across the life course, is key if we are to continue to advance our knowledge of the biological impacts of our environments and society. So far, much of the evidence is based on cross-sectional data or comes from biomarkers measured once, but with longitudinal social data for the same individuals. These emerging longitudinal measures will help us to better understand how our physiologies change over time and at different stages in life, exploring the importance of relative change within individuals (i.e. is it a high allostatic load that matters or the change in allostatic load score over time?). We must also begin to embrace theories and methods from

other fields, such as 'system dynamics' (Ford, 1998) and 'complexity theory' (Byrne, 1998). The increase in data linkage to routinely collected data records (e.g. health surveys and hospital admissions) is allowing us to research the long-term health consequences of socioeconomic circumstances, even after studies and surveys have ceased. It may also be possible in the future to link into biomarker data that are collected as is now done with hospital admissions and death records. There are obviously challenges and negatives linked to these ideas, but they offer possibilities to broaden our knowledge of the social determinants of health and to help design better policies and programmes for reducing inequalities and improving health.

References

Acheson, D. (1998). *Inequalities in health: report of an independent inquiry*. HMSO, London, UK. http://dx.doi.org/10.1136/bmj.317.7173.1659

Adams, J. M., & White, M. (2004). Biological ageing: a fundamental, biological link between socio-economic status and health? *European Journal of Public Health, 14*(3), 331-334. http://dx.doi.org/10.1093/eurpub/14.3.331

Adler, N. E., & Ostrove, J. M. (1999). Socioeconomic status and health: what we know and what we don't. *Annals of the New York Academy of Sciences, 896*, 3-15. http://dx.doi.org/10.1111/j.1749-6632.1999.tb08101.x

Adler, N. E., & Stewart, J. (2010). Health disparities across the lifespan: meaning, methods, and mechanisms. *Annals of the New York Academy of Sciences, 1186*, 5-23. http://dx.doi.org/10.1111/j.1749-6632.2009.05337.x

Baker, G., & Sprott, R. (1988). Biomarkers of aging. *Experimental Gerontology, 23*(4-5), 223-239. Baker, G., & Sprott, R. (1988). Biomarkers of aging. Experimental Gerontology, 23(4-5), 223-239. http://dx.doi.org/10.1016/0531-5565(88)90025-3

Borrell, L. N., & Crawford, N. D. (2011). Social disparities in periodontitis among US adults: the effect of allostatic load. *Journal of Epidemiology and Community Health, 65*(2), 144-149. http://dx.doi.org/10.1136/jech.2009.098269

Byrne, D. (1998). *Complexity Theory and the Social Sciences*. London: Routledge.

Capewell, S., McCartney, M., & Holland, W. (2015). Invited debate: NHS Health Checks-a naked emperor? *Journal of Public Health, 37*(2), 187-192. http://dx.doi.org/10.1093/pubmed/fdv063

de Lange, T. (2002). Protection of mammalian telomeres. *Oncogene, 21*(4), 532-540. http://dx.doi.org/10.1038/sj.onc.1205080

Der, G., Batty, G. D., Benzeval, M., Deary, I. J., Green, M. J., McGlynn, L., McIntyre, A., Robertson, T., & Shiels, P. G. (2012). Is telomere length a biomarker for aging: cross-sectional evidence from the west of Scotland? *PLoS One, 7*(9), e45166. http://dx.doi.org/10.1371/journal.pone.0045166

Duru, O. K., Harawa, N. T., Kermah, D., & Norris, K. C. (2012). Allostatic load burden and racial disparities in mortality. *Journal of the National Medical Association, 104*(1-2), 89-95. http://dx.doi.org/10.1016/S0027-9684(15)30120-6

Epel, E. (2012). How Reversible Is Telomeric Aging. *Cancer Prevention Research, 5*, 1163. http://dx.doi.org/10.1158/1940-6207.CAPR-12-0370

Ford, F. (1998). *Modeling the Environment: An Introduction to System Dynamics Modeling of Environmental Systems*. Washington DC: Island Press.

Gruenewald, T. L., Karlamangla, A. S., Hu, P., Stein-Merkin, S., Crandall, C., Koretz, B., & Seeman, T. E. (2012). History of socioeconomic disadvantage and allostatic load in later life. *Social Science & Medicine, 74*(1), 75-83. http://dx.doi.org/10.1016/j.socscimed.2011.09.037

Gruenewald, T. L., Seeman, T. E., Ryff, C. D., Karlamangla, A. S., & Singer, B. H. (2006). Combinations of biomarkers predictive of later life mortality. *Proceedings of the National Academy of Sciences of the United States of America, 103*(38), 14158-14163. http://dx.doi.org/10.1073/pnas.0606215103

Gustafsson, P. E., Janlert, U., Theorell, T., Westerlund, H., & Hammarstrom, A. (2012). Social and material adversity from adolescence to adulthood and allostatic load in middle-aged women and men: results

from the Northern Swedish Cohort. *Annals of Behavioral Medicine, 43*(1), 117-128. http://dx.doi.org/10.1007/s12160-011-9309-6

Gustafsson, P. E., San Sebastian, M., Janlert, U., Theorell, T., Westerlund, H., & Hammarstrom, A. (2014). Life-course accumulation of neighborhood disadvantage and allostatic load: empirical integration of three social determinants of health frameworks. *American Journal of Public Health, 104*(5), 904-910. http://dx.doi.org/10.2105/AJPH.2013.301707

Hwang, A. C., Peng, L. N., Wen, Y. W., Tsai, Y. W., Chang, L. C., Chiou, S. T., & Chen, L. K. (2014). Predicting all-cause and cause-specific mortality by static and dynamic measurements of allostatic load: a 10-year population-based cohort study in Taiwan. *Journal of the American Medical Directors Association, 15*(7), 490-496. http://dx.doi.org/10.1016/j.jamda.2014.02.001

Karlamangla, A. S., Singer, B. H., & Seeman, T. E. (2006). Reduction in allostatic load in older adults is associated with lower all-cause mortality risk: MacArthur studies of successful aging. *Psychosomatic Medicine, 68*(3), 500-507. http://dx.doi.org/10.1097/01.psy.0000221270.93985.82

Macintyre, S. (2007). *Inequalities in health in Scotland: what are they and what can we do about them? Occasional Paper 17*. MRC/CSO Social and Public Health Sciences Unit, Glasgow.

Marmot, M. (2010). *Fair society, healthy lives: strategic review of health inequalities in England post 2010*. Institute of Health Equity, London.

Marmot, M., Friel, S., Bell, R., Houweling, T. A. J., & Taylor, S. (2008). Closing the gap in a generation: health equity through action on the social determinants of health. *The Lancet, 372*, 1661-1669. http://dx.doi.org/10.1016/S0140-6736(08)61690-6

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine, 338*(3), 171-179. http://dx.doi.org/10.1056/NEJM199801153380307

Robertson, T., Batty, G. D., Der, G., Fenton, C., Shiels, P. G., & Benzeval, M. (2013). Is socioeconomic status associated with biological aging as measured by telomere length? *Epidemiologic Reviews, 35*, 98-111. http://dx.doi.org/10.1093/epirev/mxs001

Robertson, T., Batty, G. D., Der, G., Green, M. J., McGlynn, L. M., McIntyre, A., Shiels, P. G., & Benzeval, M. (2012). Is telomere length socially patterned? Evidence from the West of Scotland Twenty-07 Study. *PLoS One, 7*(7), e41805. http://dx.doi.org/10.1371/journal.pone.0041805

Robertson, T., Benzeval, M., Whitley, E., & Popham, F. (2015). The role of material, psychosocial and behavioral factors in mediating the association between socioeconomic position and allostatic load (measured by cardiovascular, metabolic and inflammatory markers). *Brain, Behavior, and Immunity, 45*, 41-49. http://dx.doi.org/10.1016/j.bbi.2014.10.005

Robertson, T., Popham, F., & Benzeval, M. (2014). Socioeconomic position across the lifecourse & allostatic load: data from the West of Scotland Twenty-07 cohort study. *BMC Public Health, 14*, 184. http://dx.doi.org/10.1186/1471-2458-14-184

Scott, S., Curnock, E., Mitchell, R., Robinson, M., Taulbut, M., Tod, E., & McCartney, G. (2013). *What would it take to eradicate health inequalities? Testing the fundamental causes theory of health inequalities in Scotland*. NHS Health Scotland, Glasgow, UK.

Seeman, T., Glei, D., Goldman, N., Weinstein, M., Singer, B., & Lin, Y. H. (2004). Social relationships and allostatic load in Taiwanese elderly and near elderly. *Social Science & Medicine, 59*(11), 2245-2257. http://dx.doi.org/10.1016/j.socscimed.2004.03.027

Seeman, T., McEwen, B. S., Rowe, J. W., & Singer, B. H. (2001). Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging. *Proceedings of the National Academy of Sciences of the United States of America, 98*(8), 4770-4775. http://dx.doi.org/10.1073/pnas.081072698

Seeman, T., Singer, B. H., Rowe, J. W., Horwitz, R. I., & McEwen, B. S. (1997). Price of adaptation--allostatic load and its health consequences. MacArthur studies of successful aging. *Archives of Internal Medicine, 157*(19), 2259-2268. http://dx.doi.org/10.1001/archinte.1997.00440400111013

Sterling, P., & Eyer, J. (1988). Allostasis: A new paradigm to exlain arousal pathology. In S. Fisher & R. Reason (Eds.), *Handbook of Life Stress, Cognition and Health* (pp. 629-649). New York: John Wiley & Sons.

Theall, K. P., Brett, Z. H., Shirtcliff, E. A., Dunn, E. C., & Drury, S. S. (2013). Neighborhood disorder and telomeres: connecting children's exposure to community level stress and cellular response. *Social Science & Medicine, 85*, 50-58. http://dx.doi.org/10.1016/j.socscimed.2013.02.030

Commentary by

Per E Gustafsson Umeå University, Sweden
Per.E.Gustafsson@umu.se

Introduction

The authors give a thoughtful and incisive outlook on the theory and study of allostatic load (AL). In addition to a number of specific and concrete contributions I particularly welcome the general attention the authors pay to conceptual clarity, both with regard to the conceptual framing of the AL, and to its operationalisation. I believe that the conceptual ideas suggested by the authors have merit, and also that there are several details that invite further thought and discussion. Therefore, in this commentary I aim to highlight certain conceptual issues relevant for two links the authors explore; first, the one between the concept of AL and its operationalisation; and second, the link between the concepts of AL on one hand and embodiment on the other. I hope that my reflections will be helpful in furthering the endeavors the authors have initiated.

In the article, the authors mention the need to remain *loyal,* more precisely loyal to the theory of AL in the process of operationalising the concept. I think that describing it as a matter of (conceptual) loyalty is a very apt expression for situations where you aim to keep in concordance to an underlying theory (or belief system or ideology). I also think it may be a heuristic term to illustrate some of the complexities that may arise when dealing with concepts. My commentary can be viewed as an exploration of some conceptual loyalties, disloyalties and conflicts of loyalties in play in the operationalisation and conceptual framing of AL discussed by the authors.

Loyalty in operationalisation

I will start by commenting on loyalty in the sense the authors use it; that is, as staying true to the concept in the process of operationalisation. The authors make several constructive points here that if followed, would promote conceptual loyalty. For example, the observations that the definition of thresholds deviates from the concept of AL, the interesting possibility of constructing different AL scores for different manifest diseases, and the consequences of the heterogeneous stability of AL components. I also appreciate that they revitalise the idea of the causal ordering of the mediators and effects, which I regard as an important part of AL theory, but which unfortunately has received comparatively little empirical attention.

I would also like to comment on a specific issue where I do not seem to agree with the authors; or more specifically, where I do not see how their reasoning promotes loyalty to the concept of AL. The issue concerns the authors' discussion on selection of the multiple biological systems. Here, the authors seem to frame loyalty to the theory of AL only with respect to the degree AL operationalisations (or the set biological systems) predict manifest disease. Yet AL was developed as a concept and a measure designed to connect the social and the biological worlds or realities, with AL acting as a link between stressful experiences and the pathogenesis of manifest disease (McEwen, 1998). The theory of AL thus makes assumptions on both the predictor and outcome side of AL, and AL could be said to have the putative causal status of a mediator or intervening variable between environmental exposures on one hand, and manifest disease outcomes on the other. While the 'disease criterion' (AL as a predictor of manifest disease) is commonly considered in discussions and empirical examinations of AL operationalisations, the 'environmental criterion' (AL as an outcome of environmental exposures) has been given less consideration in operationalisations of AL, but instead is left as an empirical question to be examined subsequent to and independently from the operationalisation of AL. This emphasis is also reflected in the present article.

My question is then; should not a conceptually loyal AL measure need to reflect accurately the biological impact of the (social, physical) environment to the same degree as it accurately predicts manifest disease? If no, why not; what in the theory of AL suggests that the environmental criterion is secondary to the disease criterion?

To me, this emphasis of the disease criterion in the operationalisation of AL reflects a disloyalty to parts of AL theory. I also believe that this disloyalty may have unfortunate consequences for our understanding of the role of AL. An approach considering only, or mostly, the disease criterion in

the operationalisation of AL can be expected to result in AL measures which indeed are good predictors of manifest disease, but which do not necessarily play an important role in explaining social causes of disease. This kind of approach will therefore yield poor AL measures for the purpose the authors state; AL playing an integral role in explaining social gradients in health. Ultimately, we risk ending up with the empirical results and conclusions suggesting that AL does not play a role in explaining social health differentials. However, such inferences would be laden with the repercussions of bias we introduced in our operational approach – our initial disloyalty to the theory of AL.

To the degree that such empirical considerations should influence the operationalisation of AL, I wonder if a more loyal approach should give equal consideration to both criteria; to both sides of the causal chain in which AL is supposedly a link. This would mean choosing the physiological markers most accurately reflecting environmental conditions, in addition to those that most accurately predict disease. As a statistical representation (or simply a heuristic illustration) of this dual consideration, estimates such as the 'indirect effect' used in classic regression-based mediation analysis (Baron & Kenny, 1986) could be used, as it takes the mediator's associations to both the exposure and outcome equally into account. Selecting biological systems and also individual markers guided by such a (data-driven or theory-based) approach would result in conceptually loyal AL measures, which also are given a fair chance to empirically explain social inequalities in health.

Loyalty in conceptualisation

Conceptual loyalty becomes even more intricate under situations of dual loyalty, which is the case when we seek to integrate different concepts or frameworks. Conceptual integration can of course be straightforward. Maybe the entities to be integrated have been developed in the same scientific-historical context, maybe they share a phenomenon under study, and maybe they have similar conceptual goals and terminology. But conceptual integration of two or more concepts may also be trickier than first anticipated. We might be caught in conflicts of loyalty.

In their article, the authors propose integration of two concepts. First, the concept of *allostatic load*, which was born in the scientific context of physiology and stress research, based on the writing of Sterling and Eyer (1988) on allostasis and developed by McEwen and Stellar (McEwen, 1998; McEwen & Stellar, 1993). Second, the concept of *embodiment,* which has a more diverse history and which has been used (often implicitly) with widely different meanings in the health sciences literature (Hammarstrom, et al., 2014), e.g. by the sociologist Raewyn Connell (Connell, 2011), or within the phenomenological tradition (Bullington, 2009). In the present paper the authors use the embodiment concept of Nancy Krieger, who developed her own formulation of embodiment within a distinctly social epidemiological context during the 1990s and 2000s, as one central concept within the larger theoretical framework of *ecosocial theory* (Krieger, 1994, 2005, 2011).

At a glance, the purposes of the AL and embodiment concepts may seem readily commensurable. Both concepts are dealing with the same general phenomenon of environment and biology, and could be viewed as existing, at least partly, within the family of theories relevant to social determinants of health. Both focus on different areas of this phenomenon, but also encompass the area of the other, and both mention life course perspectives as one central tenet (but without delving into the details) (Krieger, 1994; McEwen, 1998). But what would the point be in doing such an integration? What do the two perspectives have to offer each other?

For AL theory, I would say that framing allostatic load under embodiment does have the potential to put the theory of AL into a well-developed theory of societal structure and population patterns of health and disease, in this case ecosocial theory. This I see as a substantial and much-needed conceptual gain for the theory of AL. Sure, references to society and social inequalities have always been present within AL theory, but they have generally taken the form of vague hints to frameworks without a detailed conceptual integration (Juster, McEwen, & Lupien, 2010; McEwen, 1998), or empirical examinations (Dowd, Simanek & Aiello, 2009; Seeman, Epel, Gruenewald, Karlamangla & McEwen, 2010; Szanton, Gill & Allen, 2005). Moreover, in the same way that societal structure has not been the main focus of the theory of AL, formulating specific intermediate links or health outcomes has not been a high priority within ecosocial theory. From

ecosocial theory's point of view, allostatic load could therefore contribute with a specific, concrete and operationalisable summary construct capturing a range of structural exposures that are relevant for the process of embodiment.

So, in such an integration, what do we need to pay attention to? Here, I think that we do need to clarify where our conceptual loyalties are, and also where they should be. With regard to the latter, from my point of view, conceptual loyalty should be mutual and equal towards each of the concepts or frameworks that are to be integrated. With regard to the former, in reading the article, I notice a strong conceptual loyalty towards the concept of AL, but a more tenuous one towards embodiment. This I interpret as a conflict of loyalty.

To exemplify my point, the authors title their paper, 'Allostatic load as a measure of social embodiment'. This view, where embodiment seems to be construed as something that can be captured by AL, is also expressed in parts of the paper ('AL may be a relevant and useful tool for measuring and comparing embodiment'). In other places in the article, however, the relationship between the two concepts is described as something which appears to be substantively different from in the first view; AL is described as the *biological expression* or *effect* of embodiment ('the 'physiological reality', the 'biological expression of embodiment', 'measuring the biological effect of embodiment'). My interpretation here is that AL is construed as something other than, causally subsequent to, or part of, embodiment. Thus, it seems to me that the article comprise two different conceptualisations of embodiment in relation to AL; one where the latter is an example of the former, and one where the latter is a result of the former. Here, I reminisce about the oft-cited quote referencing Hans Selye's stress theory: "Stress in addition to being itself, was also the cause of itself, and the result of itself." (Rosch, 1998).

So, which of the conceptualisations is more loyal to the concept of embodiment? With regard to the first conceptualisation I wonder whether embodiment really can be reduced to a physiological measurement. In what way are we, by summarising a number of cardiovascular risk factors and neuroendocrine markers, capturing 'how we literally incorporate, biologically, in societal and ecological context, the material and social world in which we live'? In relation to this question it is worth noting that Krieger emphasises that embodiment is not equivalent to, but encompasses more than, 'how society gets under the skin' or 'biological embedding' (Krieger, 2011, p. 222). Specifically, I interpret embodiment as not primarily reflecting how the proximal environment becomes embodied (as is the case in stress frameworks such as AL), but more how societal structure and dynamics become embodied and thereby create population patterns of disease. Here, embodiment is an alternating macro-micro-macro process, and as such by necessity a multilevel phenomenon. This does not seem to correspond well to the first conceptualisation of embodiment in the article, where embodiment is reduced to a much more limited construct, which seems to be guided more by loyalties to the theory of AL than to loyalties to the theory of embodiment. This expresses the loyalty conflict, and I would say that in this restricted sense the concept of embodiment adds little to the theory of allostatic load which was not already contained in the theory of AL. Consequently, I would advise against this conceptualisation of embodiment.

In the second conceptualisation of embodiment in the article, AL is instead construed as an effect of embodiment. Here, there are no restraints put on the concept of embodiment and what it represents; it just positions AL as one (possibly of many) biological effects of the (possibly complex and multilevel) phenomenon that is embodiment. Therefore, I regard this view of embodiment as more loyal to the concept of embodiment, and a more fruitful starting point for a conceptual integration of the two concepts.

Still, as noted above, embodiment is one concept within the larger theoretical framework of ecosocial theory, where ecosocial theory cannot be reduced to embodiment, and embodiment does not capture the entirety of ecosocial theory. To stay loyal towards the concept of embodiment I therefore think it should not be picked out as a single concept, disentangled from its theoretical context. Instead, I would rather approach the integration by framing AL within the complete ecosocial theory. This would for example mean construing AL as a phenomenon which is part of the societal arrangements of power and property; of current and changing societal patterns of disease; and for which we as researchers who study social inequalities in health, as well as those in power, are

explicitly held to account (Krieger, 2011). By using the entirety of ecosocial theory we could stay loyal to both the theories of embodiment and AL. I also believe this has a greater potential to lift the theory of AL from its individual and micro-focus, to include the grander macro-level narrative of society and its flourishing inequities. Such a perspective is offered by ecosocial theory and I believe is necessary for the theory of AL to be able to play an important role, not only in empirically explaining social gradients in health, but also in the theoretical context of equity in health.

While I hold that no theory or framework is holy or deserves our loyalty simply by its existence, I do believe that the ideas (e.g. theories, frameworks or concepts) on which we base our research are particularly important for us to articulate and scrutinise, or else our research, and the understanding we believe that we gain from it, runs the risk of simply reflecting our initial errors in thought. In this commentary I have sought to keep in line with the attention to conceptual detail of the original article, by highlighting a few problems of conceptual loyalty I perceived, as well as giving my thoughts on how to possibly solve or avoid them. Together, I hope these small reflections can contribute to further thought and discussion on the theory of AL, and I am sincerely looking forward to the authors' response.

References

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*(6), 1173-1182. http://dx.doi.org/10.1037/0022-3514.51.6.1173

Bullington, J. (2009). Embodiment and chronic pain: implications for rehabilitation practice. *Health Care Analysis, 17*(2), 100-109. http://dx.doi.org/10.1007/s10728-008-0109-5

Connell, R. (2011). Gender, health and theory: conceptualizing the issue, in local and world perspective. *Social Science and Medicine*, *74*(11), 1675-1683. http://dx.doi.org/10.1016/j.socscimed.2011.06.006

Dowd, J. B., Simanek, A. M., & Aiello, A. E. (2009). Socio-economic status, cortisol and allostatic load: a review of the literature. *International Journal of Epidemiology, 38*(5), 1297-1309. http://dx.doi.org/10.1093/ije/dyp277

Hammarstrom, A., Johansson, K., Annandale, E., Ahlgren, C., Alex, L., Christianson, M., Elwer, S., Eriksson, C., Fjellman-Wiklund, A., Gilenstam, K., Gustafsson, P. E., Harryson, L., Lehti, A., Stenberg, G., & Verdonk, P. (2014). Central gender theoretical concepts in health research: the state of the art. *Journal of Epidemiology and Community Health, 68*(2), 185-190. http://dx.doi.org/10.1136/jech-2013-202572

Juster, R. P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience and Biobehavioral Reviews, 35*(1), 2-16. http://dx.doi.org/10.1016/j.neubiorev.2009.10.002

Krieger, N. (1994). Epidemiology and the web of causation: has anyone seen the spider? *Social Science and Medicine, 39*(7), 887-903. http://dx.doi.org/10.1016/0277-9536(94)90202-X

Krieger, N. (2005). Embodiment: a conceptual glossary for epidemiology. *Journal of Epidemiology and Community Health, 59*(5), 350-355. http://dx.doi.org/10.1136/jech.2004.024562

Krieger, N. (2011). *Epidemiology and the people's health: theory and context*. Oxford: Oxford University Press. http://dx.doi.org/10.1093/acprof:oso/9780195383874.001.0001

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine, 338*(3), 171-179. http://dx.doi.org/10.1056/NEJM199801153380307

McEwen, B. S., & Stellar, E. (1993). Stress and the individual. Mechanisms leading to disease. *Archives of Internal Medicine, 153*(18), 2093-2101. http://dx.doi.org/10.1001/archinte.1993.00410180039004

Rosch, P.J. (1998) Reminiscences of Hans Selye and the birth of 'stress'. *Stress Medicine 14*(1), 1-6. http://dx.doi.org/10.1002/(SICI)1099-1700(199801)14:1<1::AID-SMI777>3.0.CO;2-W

Seeman, T., Epel, E., Gruenewald, T., Karlamangla, A., & McEwen, B. S. (2010). Socio-economic differentials in peripheral biology: cumulative allostatic load. *Annals of the New York Academy of Sciences, 1186*, 223-239. http://dx.doi.org/10.1111/j.1749-6632.2009.05341.x

Sterling, P., & Eyer, J. (1988). Allostasis: a new paradigm to explain arousal pathology. In J. Reason & S. Fisher (Eds.), *Handbook of life stress, cognition and health* (pp. 629–649). Chichester: Wiley.

Szanton, S. L., Gill, J. M., & Allen, J. K. (2005). Allostatic load: a mechanism of socioeconomic health disparities? *Biological Research for Nursing, 7*(1), 7-15. http://dx.doi.org/10.1177/1099800405278216

Response by

Cyrille Delpierre Université of Toulouse III Paul Sabatier, France
cyrille.delpierre@inserm.fr
Cristina Barbosa-Solis Université of Toulouse III Paul Sabatier, France
Jerome Torrisani Cancer Research Center of Toulouse, France
Muriel Darnaudery University of Bordeaux, France
Melanie Bartley University College London, UK
David Blane Imperial College London, UK
Michelle Kelly-Irving Université of Toulouse III Paul Sabatier, France

We thank the authors who commented on our paper and discuss some of the most salient points they raised.

Firstly, regarding the many methodological considerations we mentioned, Getz and Tomasdottir in their comment point towards the burgeoning fields of biological systems research for potential answers. We agree that this area is promising in terms of understanding further the complexities of our biological systems and finding the most suitable way of measuring them. Indeed, the combined forces of methodological developments in the areas of bioinformatics, 'omics' research and biological systems will probably render redundant a simplified cumulative score, such as the ones typically used to measure allostatic load (AL). A more optimal method of measuring multi-system wear and tear due to stress may well emerge from these fields allowing the identification of biomarkers with a predictive or diagnostic value. A possible caveat of the increasingly accessible technologies around biological data and methodological developments within bioinformatics, is the risk of becoming overly focused on molecular-level details. Though a measure of cumulative wear-and-tear may benefit from such developments, we must not be tempted to stray too far into the attractive rabbit hole of detailed biological data and away from the original intent of the AL concept. The purpose of the measurement developed by McEwen et al (McEwen & Stellar, 1993; Seeman et al., 1997) was to capture, in one summary score, the physiological consequences of adaptations to the environment via the stress response pathways. Our aim should be to describe and capture these adequately enough to demonstrate the modifiable factors within the environment – in its broadest sense – that may be used to alter processes affecting socially structured groups of the population and leading to health inequalities. There is a risk of forgetting these important facets when faced with new and attractive methodologies.

Secondly, as mentioned by Robertson, it is relevant to question whether by attempting to understand mechanisms producing health inequalities and opening a black box, have we not formulated a new one with the concept of AL. We would argue that unlike many 'black boxes' AL has a well formulated conceptual foundation linking the environment to physiological processes via the stress response systems. Telomere length may be an interesting component of these processes, possibly to be included within an AL measure with its multi-system specificity. The links between AL and biological ageing are indeed clear. We would maintain that physiological wear-and-tear captures one set of processes potentially implicated in a wider notion of biological ageing wherein the link with stress is fundamental. Both the concepts of AL and biological ageing may deserve to be explored together, where AL is one among other potential mechanisms of biological ageing. Both concepts also deserve to be disentangled relative to the wider notions of embodiment and the framework of ecosocial theory, as pointed out by Gustafsson.

Indeed Gustafsson highlights that we were ambiguous regarding the position of the concept of AL relative to that of embodiment within the theoretical framework of 'ecosocial theory' (Krieger, 2001). We define embodiment as a dynamic concept, consisting of: i) responses to past environments and ii) an ongoing response to the present environment. The elements and mechanisms leading to the responses may vary in their nature, intensity and cadence over the life course. We suggest that AL captures one process of embodiment linking the environment, stress responses, and possible chronic damage to physiological systems, and as such this fits wholly

into the framework of ecosocial theory. Of course many other mechanisms of embodiment deserve further exploration in terms of environmental conditions across the life course, such as behavioural and psychological factors, socioemotional changes or cognitive function.

We agree with Gustasfsson that our desire to maintain an AL measure that is 'loyal' to a balance of physiological systems should be applied equally to the environmental factors that the measure attempts to capture. Now that a number of openly accessible longitudinal datasets collecting a large array of environmental and biological variables are available, it has become possible to specify plausible hypotheses to test and unpick many of the concepts raised here (Kelly-Irving, Tophoven & Blane, 2015). With this in mind, the ecosocial determinants of AL deserve to be deliberately defined and explored across contexts. Specific hypotheses that may link ecosocial factors at different environmental strata to AL need to be defined and tested using comparable data within different populations and at different stages of the life course.

References

Kelly-Irving, M., Tophoven, S. & Blane, D. (2015). Life course research: new opportunities for establishing social and biological plausibility. *International Journal of Public Health 60*,(6), 629-630. http://dx.doi.org/10.1007/s00038-015-0688-5

Krieger, N. (2001). Theories for social epidemiology in the 21st century: an ecosocial perspective. *International Journal of Epidemiology 30*, 668-677. http://dx.doi.org/10.1093/ije/30.4.668

McEwen, B. S. & Stellar, E. (1993). Stress and the individual - mechanisms leading to disease. *Archives of Internal Medicine 153*(18), 2093-2101. http://dx.doi.org/10.1093/ije/30.4.668

Seeman, T. E., Singer, B. H., Rowe, J. W., Horwitz, R. I. & McEwen, B. S. (1997). Price of adaptation - allostatic load and its health consequences - MacArthur studies of successful aging. *Archives of Internal Medicine 157*(19), 2259-2268. http://dx.doi.org/10.1001/archinte.1997.00440400111013

Referencing

The debate should be referenced as:

Delpierre, C., Barbosa-Solis, C., Torrisani, J., Darnaudery, M., Bartley, M., Blane, D., Kelly-Irving, M., Getz, L. & Tomasdottir, M.O., Roberston, T. & Gustafsson, P. (2016). Origins of heath inequalities: the case for Allostatic Load. *Longitudinal and Life Course Studies, 7, 79 – 103.* http://dx.doi.org/10.14301/llcs.v7i1.325

AUTHOR GUIDELINES SUMMARY

Submission of Papers

All papers, written in the English language, should be submitted via the *LLCS* website as a Microsoft Word file. If there is a good reason why this is not possible, authors are requested to contact crandall@slls.org.uk **before** submitting the paper. All subsequent processes involving the author are carried out electronically via the website.

Preparation of Texts

Length. The paper should normally be **approximately 5,000 words**, with longer papers also accepted up to a maximum of 7,000 words. This word count excludes tables, figures and bibliography.

Font and line spacing. Please use Calibri (or similar sans serif) font, 12pt, with 1.5 line spacing in all main text, single space in figure or table captions.

Page layout. All text should be justified to the left hand margin (all margins of at least 2.5cm) with no indents at the start of paragraphs and a line space separating paragraphs. All headings and sub-headings used within the body of the paper should also be left justified. Please do **NOT** use automatic text formatting.

Weblinks. To help our readers to look up cited references or other information available on the web, authors should ensure that all such references are activated.

DOIs. Do NOT include DOIs in the bibliography – they are added during editing as resolvable URLs.

Ensuring an anonymous (blind) review. Please submit papers with a full detailed title page. Once a paper has been submitted to *LLCS* via the website, it will be 'anonymised' by the removal of all author name(s) and institution names on the title page, and any identifying electronic document properties will also be removed. Authors do not need to remove their name(s) from the main text or references but any reference to their work or themselves should be expressed in the third person.

Abstract. The abstract (no subheads or paragraphs) should be no more than 250 words (not part of the main word count).

Keywords. These should be included just below the author list (minimum 3 maximum 10).

Abbreviations. Words to be abbreviated should be spelt out in full the first time they appear in the text with the abbreviations in brackets. Thereafter the abbreviation should be used.

References. Please use the APA 6th edition and refer to examples in the full Guidelines.

Authors not complying with these reference guidelines will be asked to make all the necessary alterations themselves, if their paper is accepted for publication.

Notes. As a general rule, supplementary notes should be avoided, but if thought to be essential, they should *not* appear on the page as Footnotes, but instead, be included as Endnotes.

Supplementary material. Supplementary material may be uploaded with the submission, and if the paper is published, will be visible to the reader via a link on the RHS of the screen.

Submissions containing graphs, tables, illustrations or mathematics. All graphs, tables and illustrations should be embedded in the submitted text, and have clear, self-explanatory titles and captions. Mathematical expressions should be created in Word 2003 (or a compatible package) with equation editor 3.0, unless the author has good reason to use other software, in which case please contact crandall@slls.org.uk. All biological measures should be reported in SI units, as appropriate, followed, in the text, by traditional units in parentheses.

Author citation. If the paper is accepted for publication, a version of the paper with all authors cited in full on the title page will be used. Only individuals who have contributed substantially to the production of the paper should be included.

Copy editing. All accepted manuscripts are subject to copy editing, with reference back to author with suggested edits and queries for response.

Proofs. The corresponding author will be asked to view a layout proof of the article on the website and respond with final amendments within three days.

(Full Author Guidelines at: http://www.llcsjournal.org/index.php/llcs/about/submissions#authorGuidelines**)**

Open Journal System

The ***LLCS*** journal is produced using the Open Journal System, which is part of the Public Knowledge Project. OJS is open source software made freely available to journals worldwide, for the purpose of making open access publishing a viable option for more journals. Already, more than 8,000 journals around the world use this software.

INTRODUCTION

1 – 2 **Editorial**
John Bynner

PAPERS

3 – 24 **Heterogeneity in chronic disease outcomes among women and men in midlife: examining the role of stability and change in childhood economic hardship**
cole Etherington, Andrea Willson, Kim Shuey

25 – 40 **Temporal trends in energy and macronutrient distribution in meals eaten by children from the Avon Longitudinal Study of Parents and Children**
Suzana Almoosawi, Victoria Cribb, Pauline Emmett, Alison Lennox

41 – 52 **Bi-directional relationships between body mass index and height from three to seven years of age: an analysis of children in the United Kingdom Millennium Cohort Study**
Laia Bécares, Yvonne Kelly, Scott Montgomery, Amanda Sacker

53 – 63 **Handling attrition and non-response in longitudinal data with an application to a study of Australian youth**
Joy Cumming, Harvey Goldstein

STUDY PROFILE

64 – 78 **The Swiss Household Panel Study: Observing social change since 1999**
Robin Tillmann, Marieke Voorpostel, Erika Antal, Ursina Kuhn, Florence Lebert, Valérie-Anne Ryser, Oliver Lipps, Boris Wernli

COMMENT AND DEBATE

79 – 103 **Origins of heath inequalities: the case for Allostatic Load**
Cyrille Delpierre, Cristina Barbosa-Solis, Jerome Torrisani, Muriel Darnaudery, Melanie Bartley, David Blane, Michelle Kelly-Irving, Tony Roberston, Per E. Gustafsson, Linn Getz, Margret Olafia Tomasdottir

LLCS Journal can be accessed online at: www.llcsjournal.org

Published by
The Society for Longitudinal and Life Course Studies
Institute of Education, 20 Bedford Way, London, WC1H 0AL

info@slls.org.uk http://www.slls.org.uk