

Pathways from parenthood history to later life health: Results from analyses of the English Longitudinal Study of Ageing

Sanna Read and Emily Grundy



<http://pathways.lshtm.ac.uk>



pathways@lshtm.ac.uk



@PathwaysNCRM



UNIVERSITY OF
CAMBRIDGE
Department of Geography

NCRM
National Centre for
Research Methods

LONDON
SCHOOL of
HYGIENE
& TROPICAL
MEDICINE



Associations between parenthood histories and health in later life

- Several, but not all, studies show worse health/higher mortality for nulliparous and high parity women (and men).
- Early parenthood is associated with poorer later health/mortality (women) and poorer later mental health (women and men)
- Late fertility associated better health/lower mortality in both women and men (but some studies the reverse)

These associations may reflect:

- **Selection** and reverse causation
- **Direct effects** e.g. physiological consequences of pregnancy and childbirth.
- **Indirect effects** e.g. costs/benefits of child rearing

Childrearing and health:

Health promoting:

- Incentives towards healthy behaviours and risk avoidance
- More social participation and activity
- Role enhancement
- Social support - in childrearing phases and in later life

Health challenging:

- Physiological demands of pregnancy, childbirth and lactation (although reduced risk breast & some other hormonally related cancers)
- Potential role conflict/role overload
- Stress (and depression)
- Economic strain
- Increased exposure infections
- Disruption of careers/education – especially for young parents

Limitations

Limitations of previous work

- Outcome measures – mortality and disability- may be too far ‘upstream’ – need indicators of sub clinical morbidity observable earlier in life course
- Failure to identify PATHWAYS through which fertility histories influence later life health
- Limited consideration of early life influences on both fertility histories and later health

Addressing these limitations

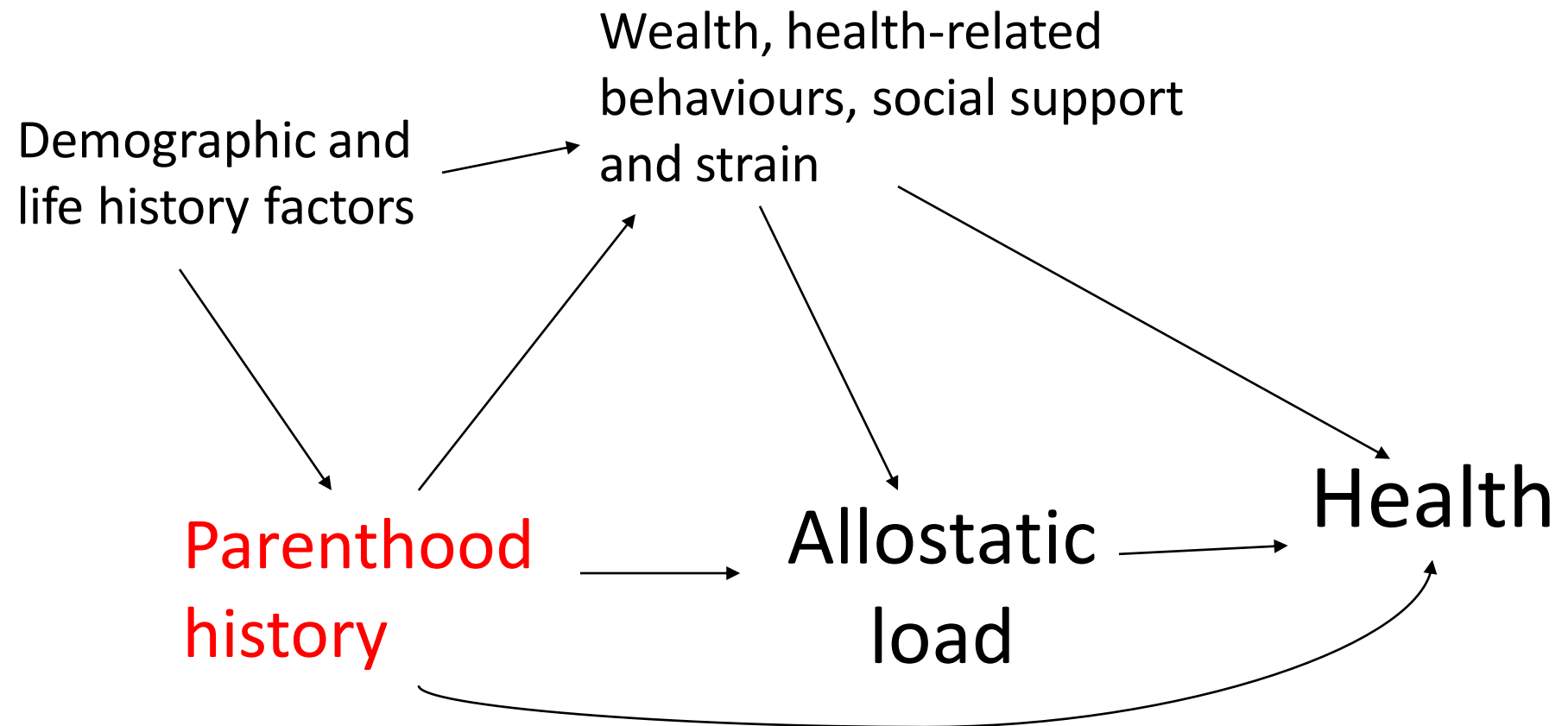
- Measures of allostatic load in mid and later life
- SEM and path analysis to identify pathways
- Modelling including early life indicators

Aim

- Identify pathways from fertility histories to later life health and mediation via wealth, health-related behaviours, social support and strain.

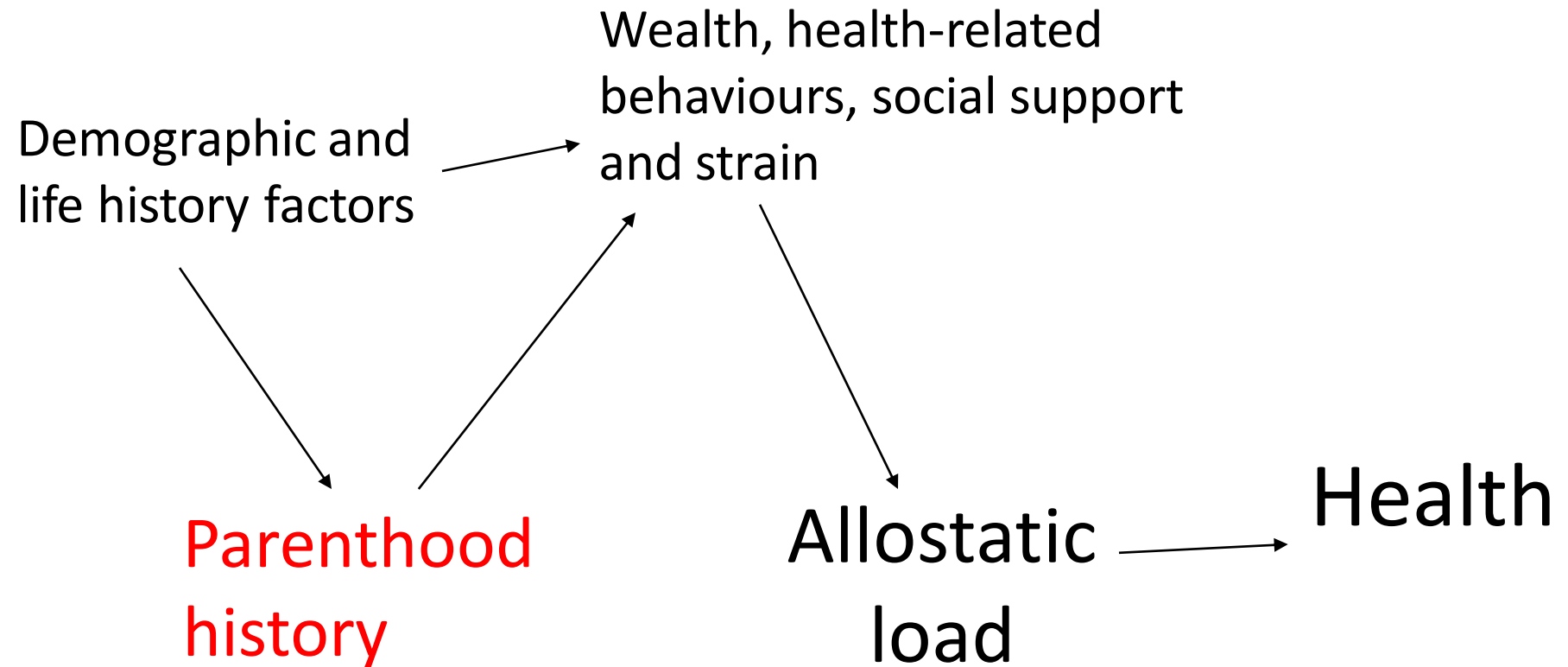
The model to be tested

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Data

- English Longitudinal Study of Ageing (ELSA) waves 1 - 3 (2002-2006) - nationally representative survey of men and women aged 50+ (*mean* = 63, *SD* = 9.2 in wave 1)
- Socio-demographic information and self reported health collected in all waves
- Detailed health data including biomarkers collected in alternate waves –biomarker data used to derive an index of allostatic load
- Retrospective life course data collected in wave 3. Analysis based on those providing life course data (*n* = 6207)

Measures

Demographic & life course:

Age, education, childhood health problem (retrospective), married/not married, and co-residence with child (time varying); ever divorced, ever widowed (wave 3).

Parenthood history:

Number of natural children (0, 1,2,3,4+); any step child; any adopted child; deceased child; for parents: young (<20/23) age first birth; late age last birth (>34/39).

Intermediate measures:

Wealth; smoking; physical activity; social support and strain (Wave 1)

Health outcomes:

Allostatic load (wave 2); health limitation (wave 3).

Allostatic load scores in ELSA

- Allostatic load: multisystem physical dysregulation resulting from long-term exposure to stress
- Grouped allostatic load index: number of biomarkers indicating high risk (upper 25th percentile, except for peak expiratory flow lower 25th percentile) calculated separately for men and women (and age group), weighted by the number of markers per system and adjusted for medication

System	Biomarker
<i>Cardiovascular</i>	Systolic blood pressure
	Diastolic blood pressure
<i>Inflammation</i>	Fibrinogen
	C-reactive protein
<i>Lipid metabolism</i>	Triglycerides
	Glycosylated haemoglobin
	Total/HDL cholesterol ratio
<i>Body fat</i>	Waist/hip ratio
<i>Respiratory</i>	Peak expiratory flow

Associations between fertility & parenthood variables, allostatic load and health limitation among men and women in ELSA

	Allostatic load		Health limitation	
	Men	Women	Men	Women
No. Natural children (ref = 2)				
0	-0.05	0.04	0.10	0.18
1	0.04	-0.14	0.14	0.07
3	0.01	0.18	0.07	-0.01
4+	0.34*	0.29*	0.29*	0.23*
Early child birth ^a	0.51***	0.58***	0.46***	0.43**
Late childbirth ^a	0.10	-0.16	0.29*	-0.23*
Adopted child	-0.15	0.55**	-0.24	0.09
Step child	0.08	0.03	0.30*	-0.09
Child died	0.22	0.03	0.21	0.19

Models include health in childhood; age; education; married/not married; ever widowed; ever divorced; intergenerational contact. Allostatic load adjusted for fasting & inhaler use. ^a parents only.

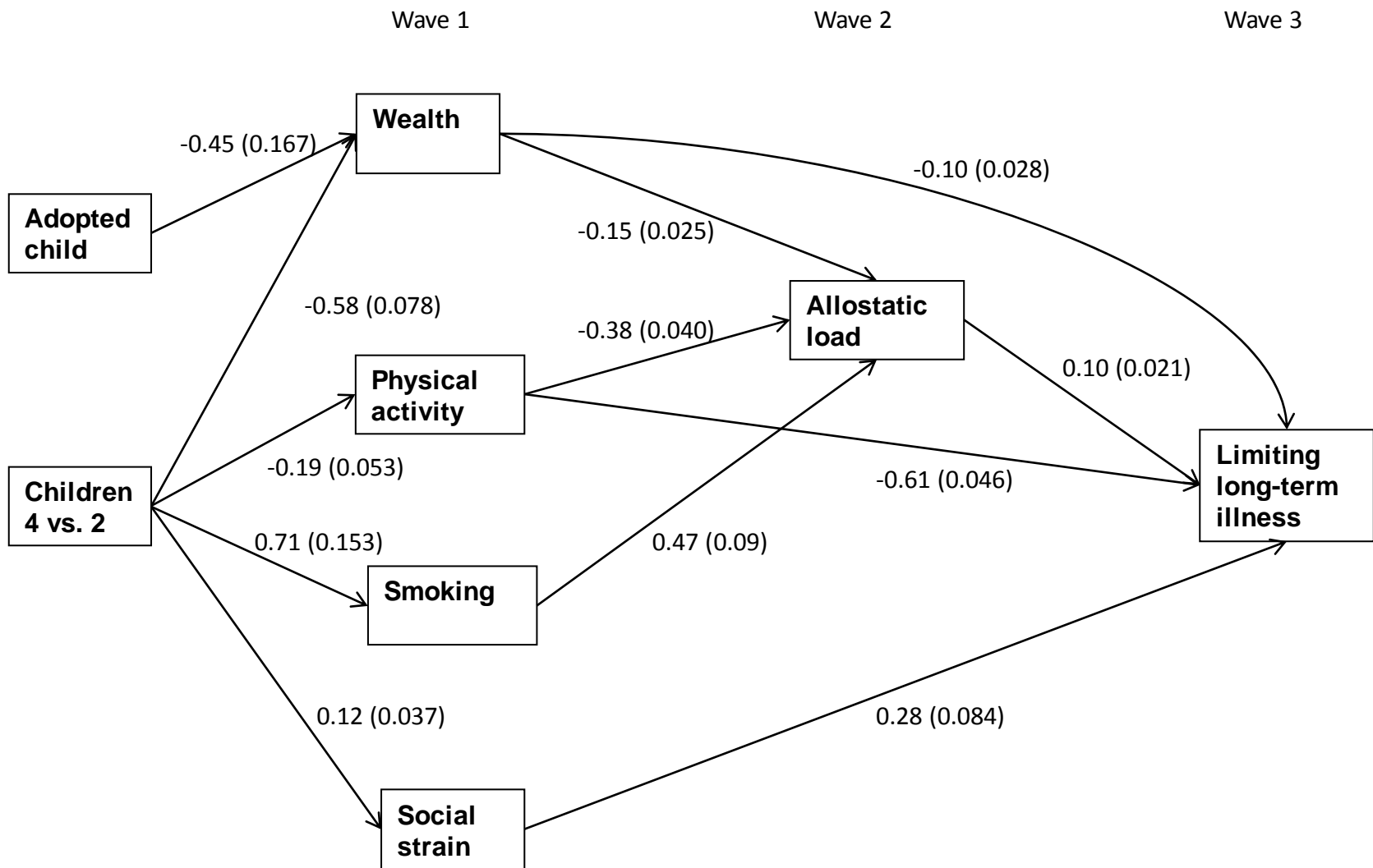


Figure 1. Path model for all women in ELSA. Model adjusted for age, education, being married, ever divorced, ever widowed and childhood health. Significant paths are shown (unstandardized estimate and standard error). Source: Analysis of ELSA waves 1-3.

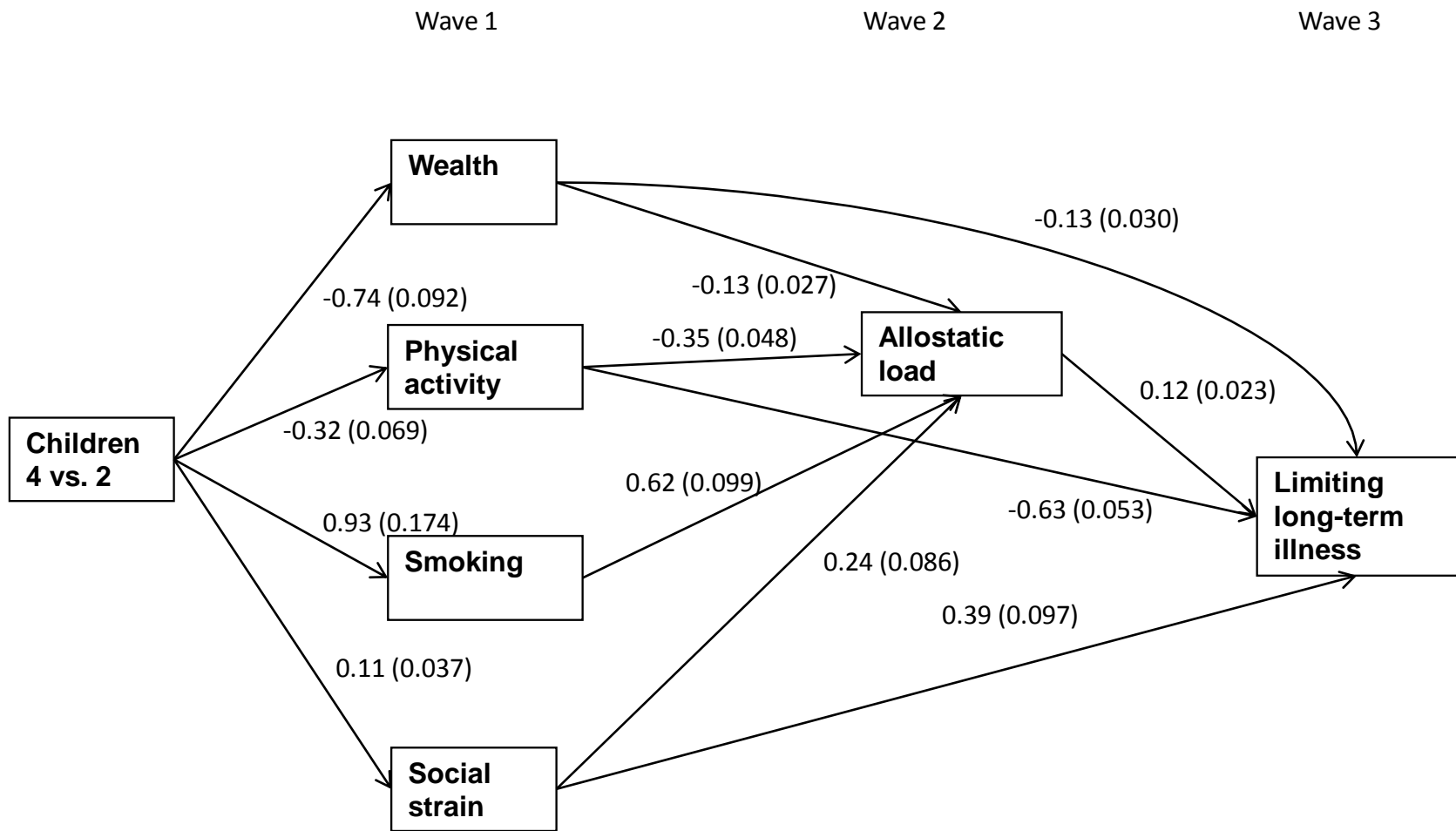


Figure 2. Path model for all men . Model adjusted for age, education, being married, ever divorced, ever widowed and childhood health. Significant paths are shown (unstandardized estimate and standard error). Source: Analysis of ELSA waves 1-3.

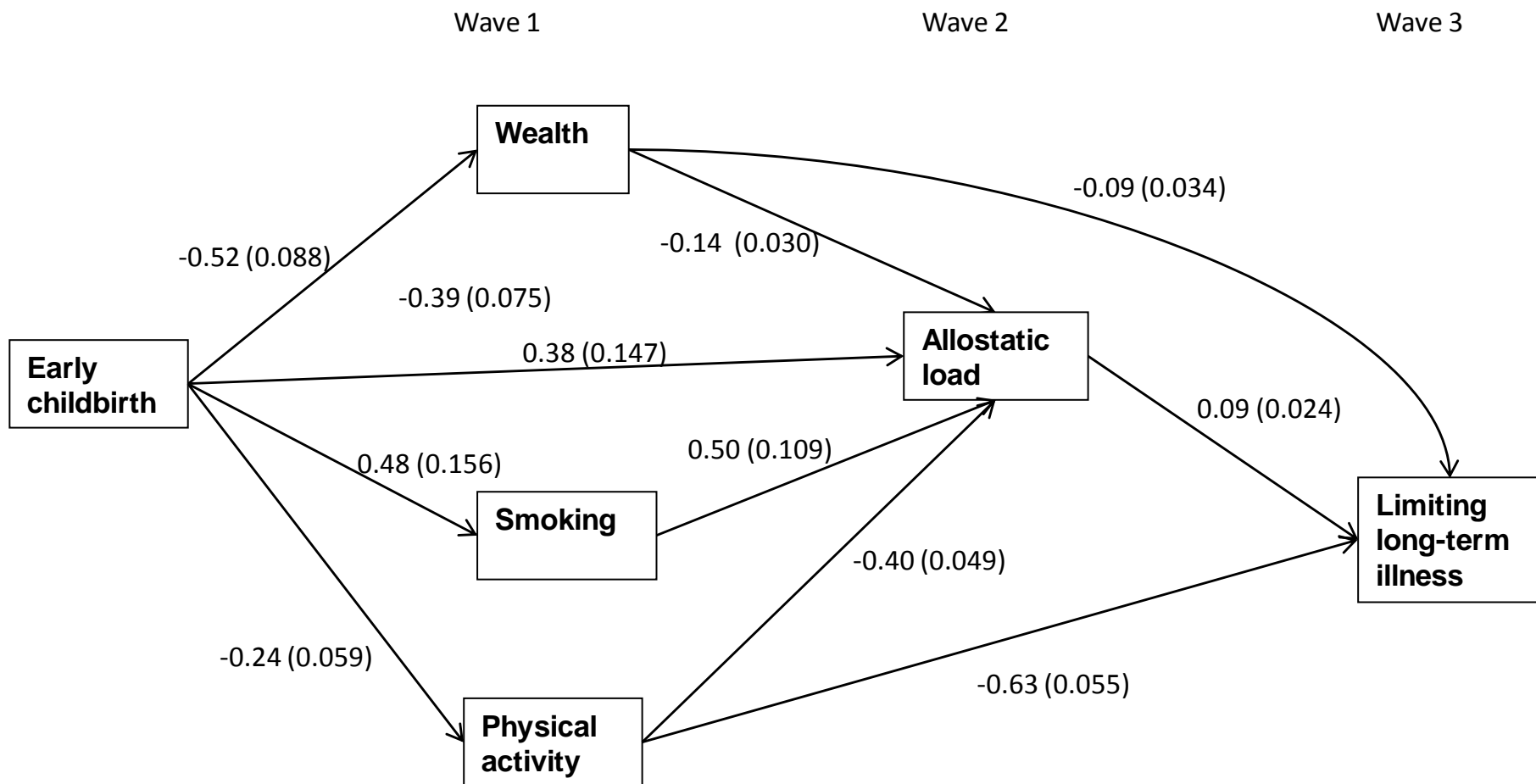


Figure 3. Path model for biological mothers. Model adjusted for age, education, being married, ever divorced, ever widowed, childhood health, and coresidence with child. Significant paths are shown (unstandardized estimate and standard error). Source: Analysis of ELSA waves 1-3.

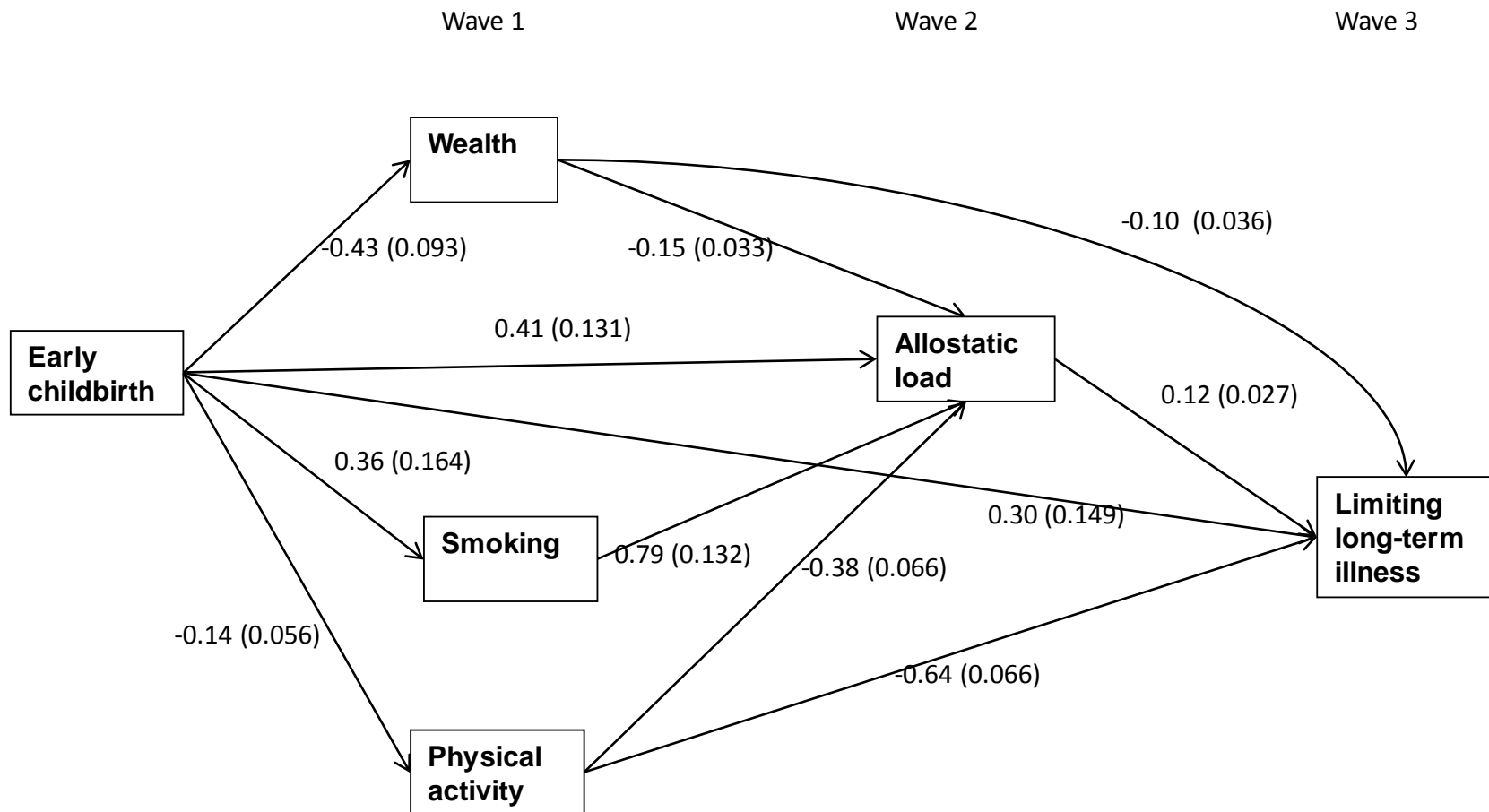


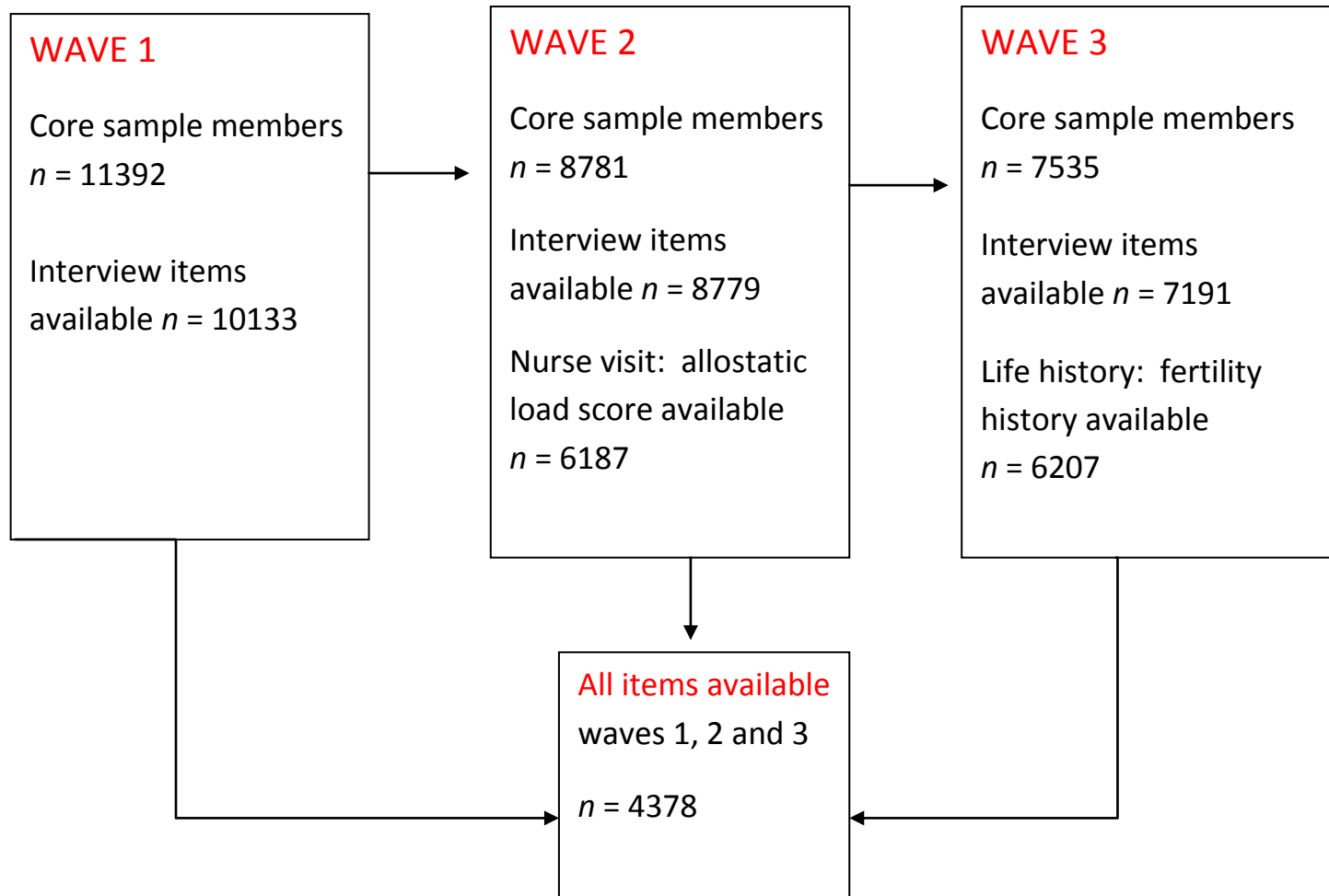
Figure 4. Path model for biological fathers. Model adjusted for age, education, being married, ever divorced, ever widowed, childhood health, and coresidence with child. Significant paths are shown (unstandardized estimate and standard error).

Source: Analysis of ELSA waves 1-3.

Conclusions & Discussion

- Socio-economic position, health-related behaviors and social strain mediate the association between high parity and later life health. They also partially mediate the association between early childbirth and later life health. Of these socio-economic position was the strongest mediator.
- So, as hypothesised, biosocial pathways from parenthood history to health involve economic position, social strain and health related behaviours
 - need now to examine in more detail pathways to particular fertility trajectories- especially childhood SES and broader environmental influences (e.g. support from the state) and other potential mechanisms (e.g. moderation).

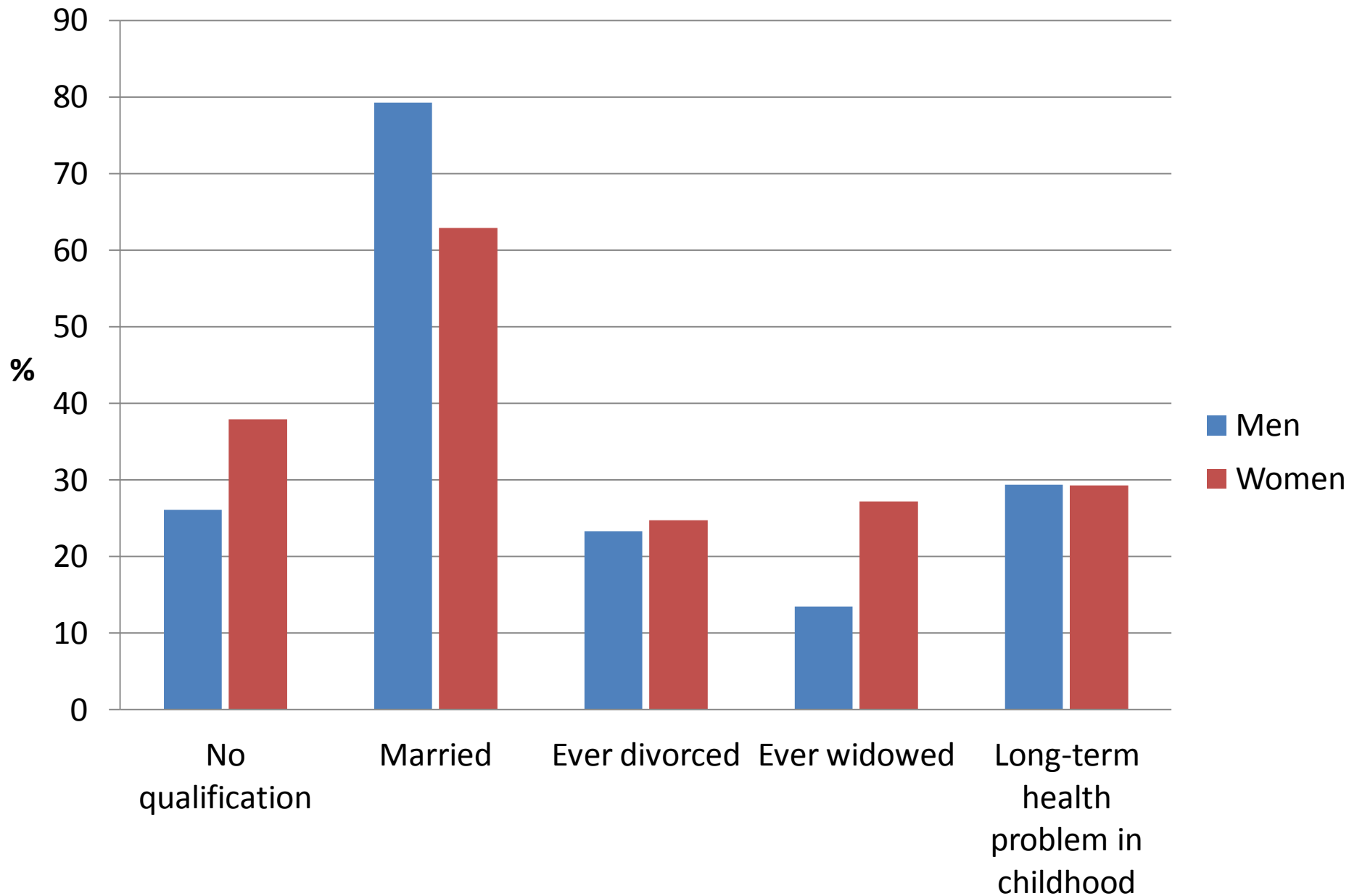
Sample derivation and data availability



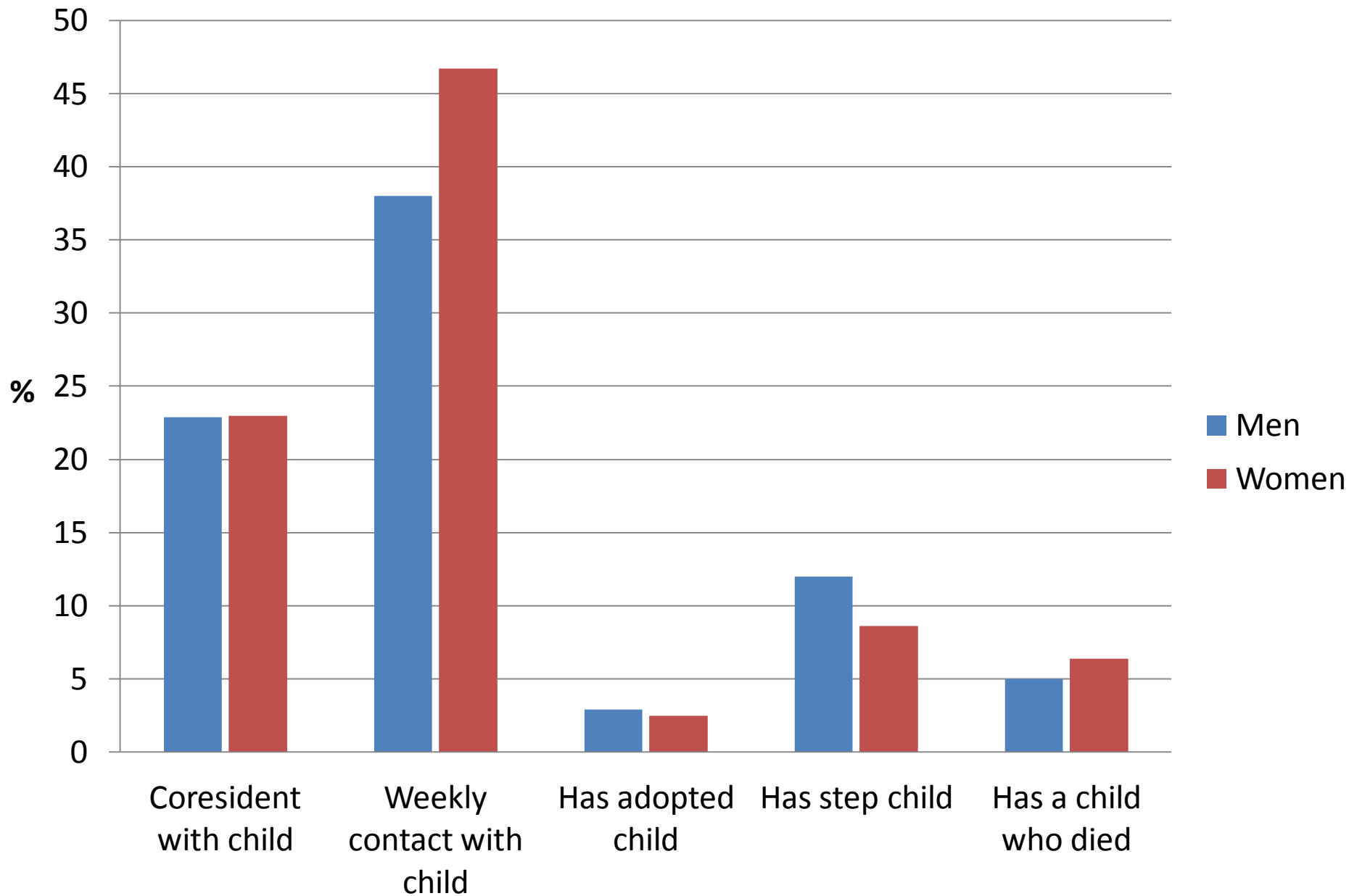
Allostatic load: 25th percentile high risk cut-off points, ELSA in wave 2 (2002).

	Men		Women	
	Aged 51-65	Aged 65+	Aged 51-65	Aged 65+
<i>Inflammation</i>	(n = 1008-1017)	(n = 982-986)	(n = 1219-1232)	(n = 1190-1196)
C-reactive protein	>2.9	>3.4	>3.4	>3.9
Fibrinogen	>3.4	>3.7	>3.5	>3.8
<i>Cardiovascular</i>	(n = 1074)	(n = 1106)	(n = 1319)	(n = 1398)
Systolic blood pressure	>143	>149	>140	>151
Diastolic blood pressure	>85	>80	>83	>79
<i>Lipid metabolism</i>	(n = 1001-1017)	(n = 965-983)	(n = 1219-1233)	(n = 1187-1196)
HDL/Total cholesterol ratio	>5.0	>4.6	>4.4	>4.5
Triglycerides	>2.5	>2.2	>2.1	>2.1
Glycosylated haemoglobin	>5.7	>5.9	>5.6	>5.8
<i>Body fat</i>	(n = 1216)	(n = 1231)	(n = 1486)	(n = 1527)
Waist/hip ratio	>1.00	>1.00	>0.88	>0.89
<i>Respiratory</i>	(n = 1197)	(n = 1190)	(n = 1415)	(n = 1437)
Peak expiratory flow	<506	<406	<344	<265

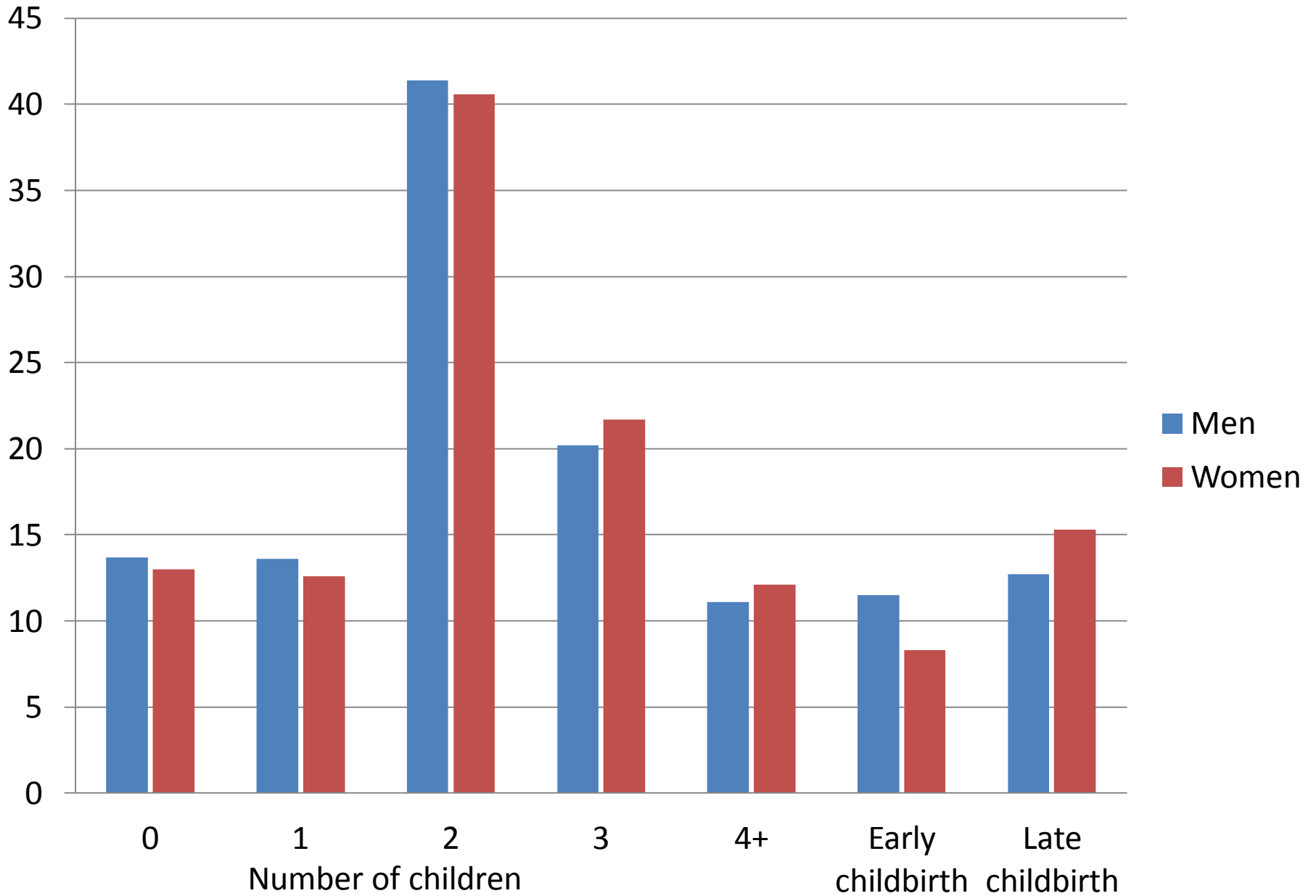
Distribution of the sample by demographic & life history variables



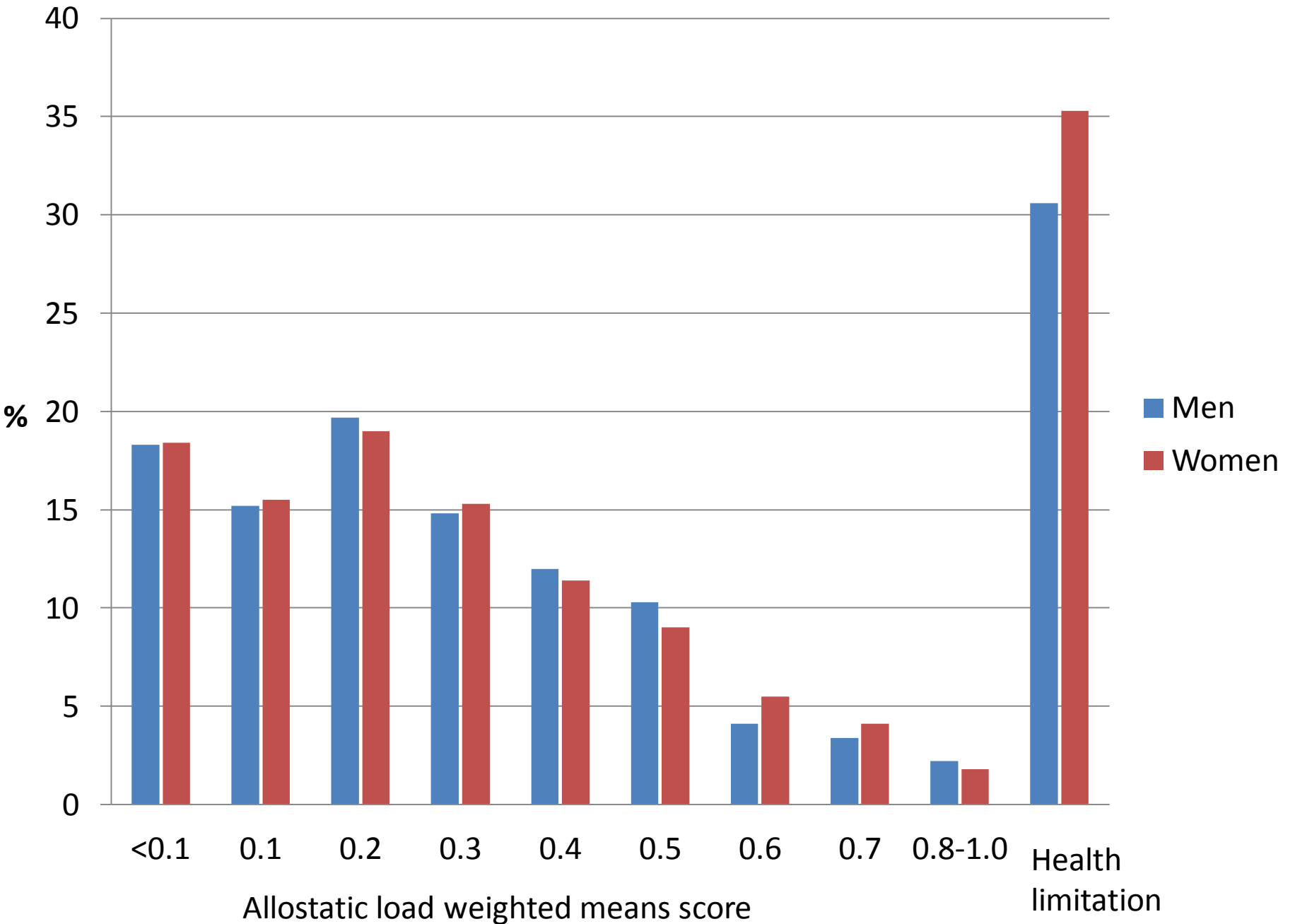
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Distribution of the health outcomes



Distribution of the sample by intermediate variables in wave 1, Mean (*SD*) or %

	Men	Women
Wealth quintile	3.4 (1.38)	3.2 (1.39)
Physical activity	2.2 (0.73)	2.1 (0.78)
Current smoking	13.9	15.5
Perceived social support	4.2 (0.50)	4.3 (0.49)
Perceived social strain	2.7 (0.42)	2.6 (0.45)