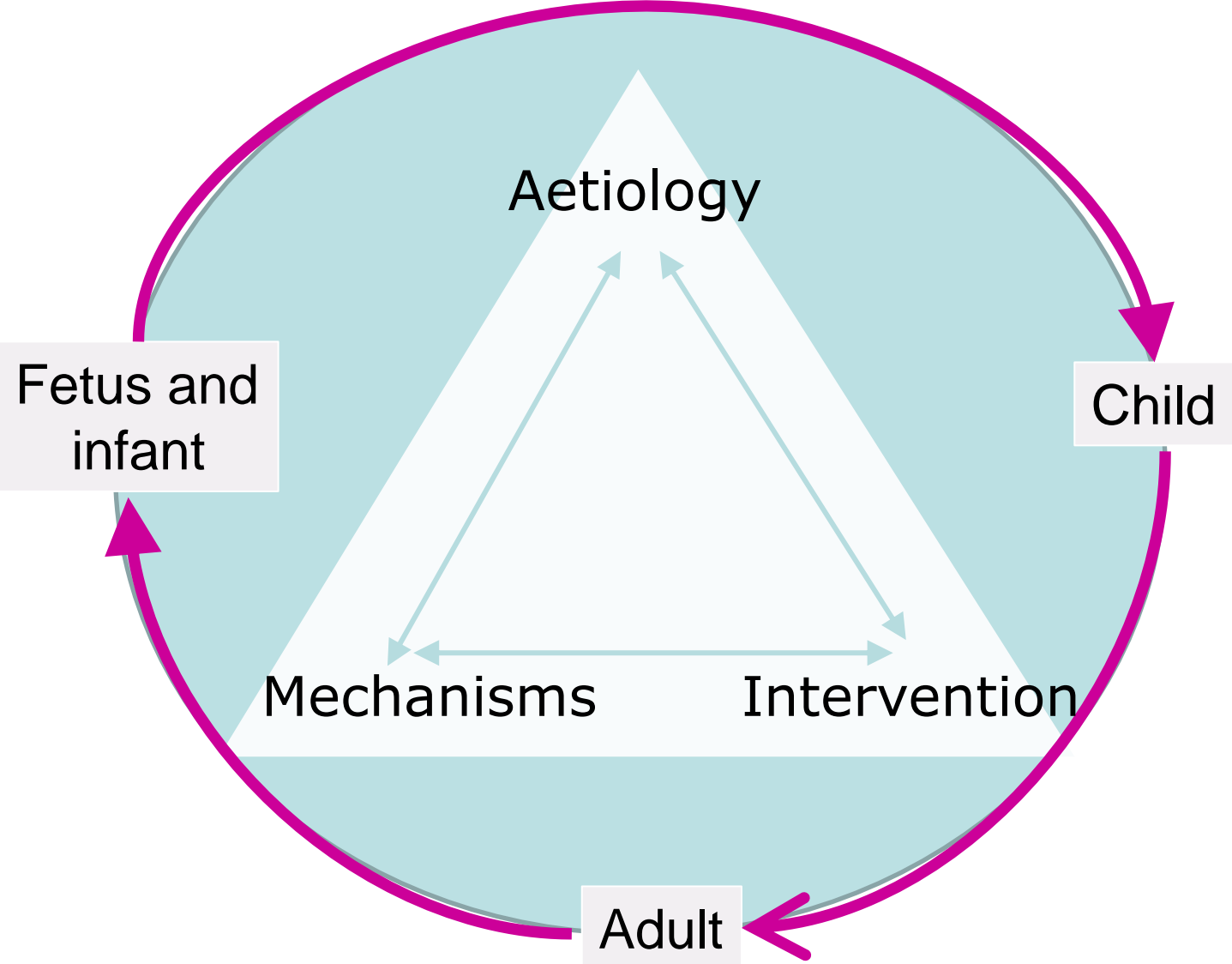


Introduction to the life-course influences on health

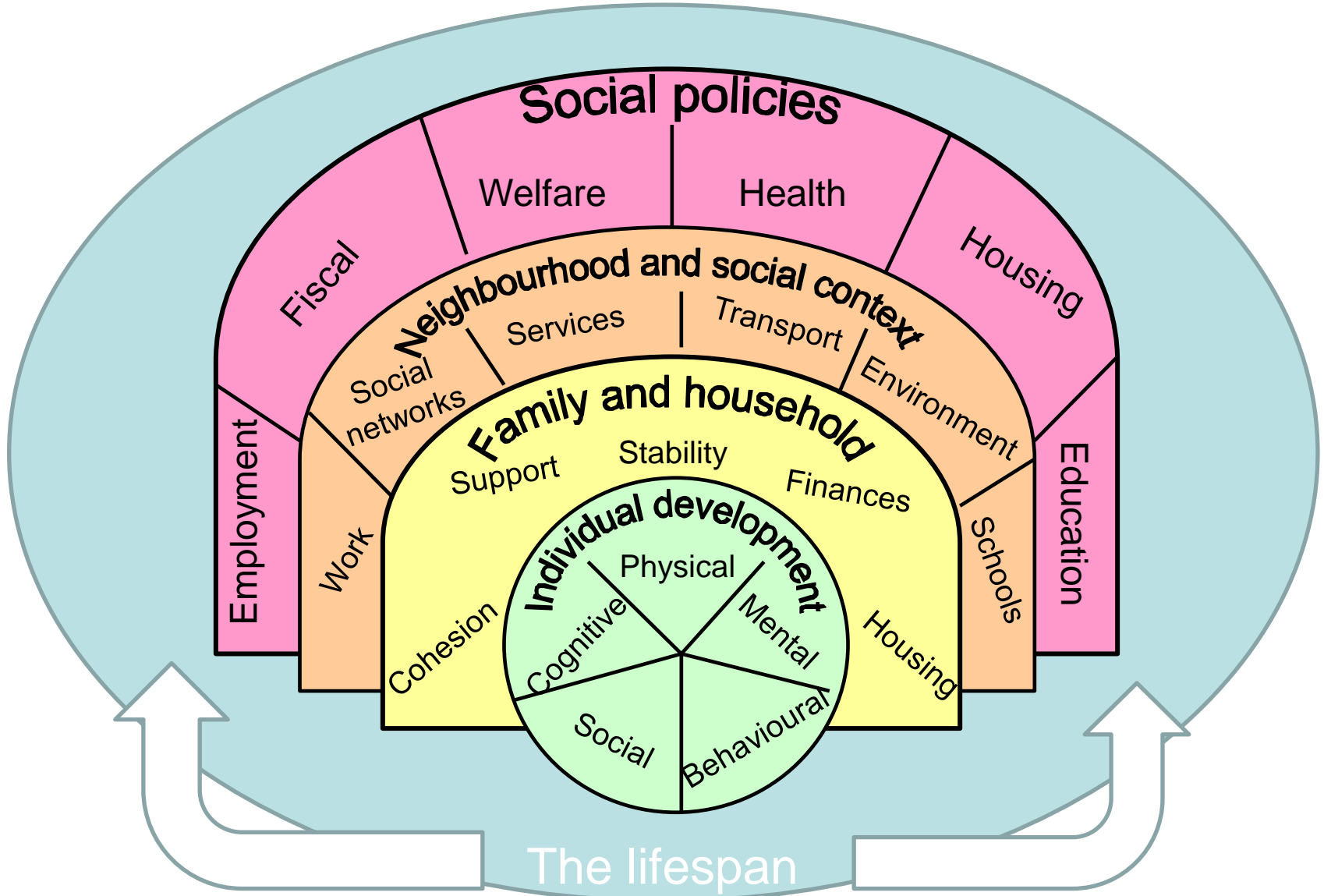
Outline

- Introducing the lifecourse into social epidemiology
- Lifecourse epidemiological models
- How lifecourse thinking can inform policy

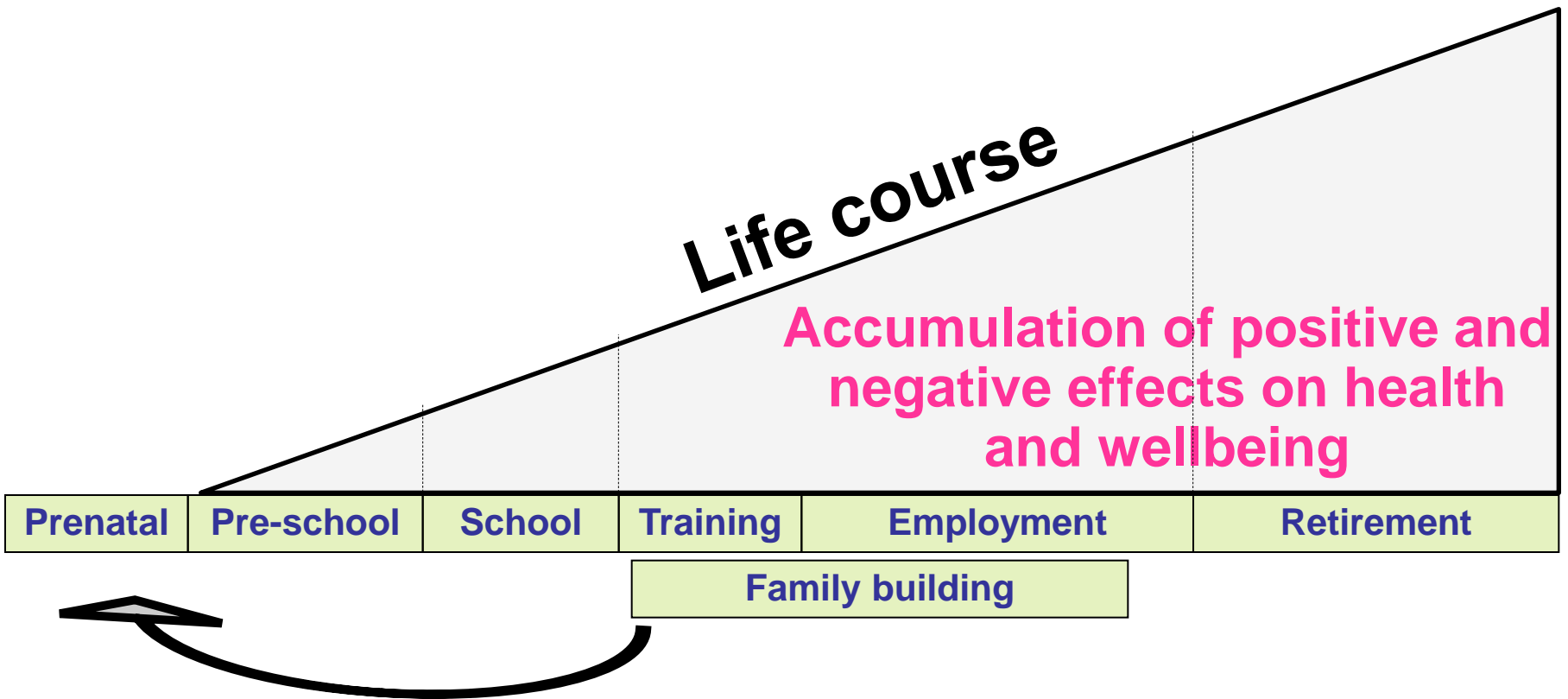
Lifecourse epidemiology



Ecological model of health across the lifecourse



Life course stages



Life course epidemiology

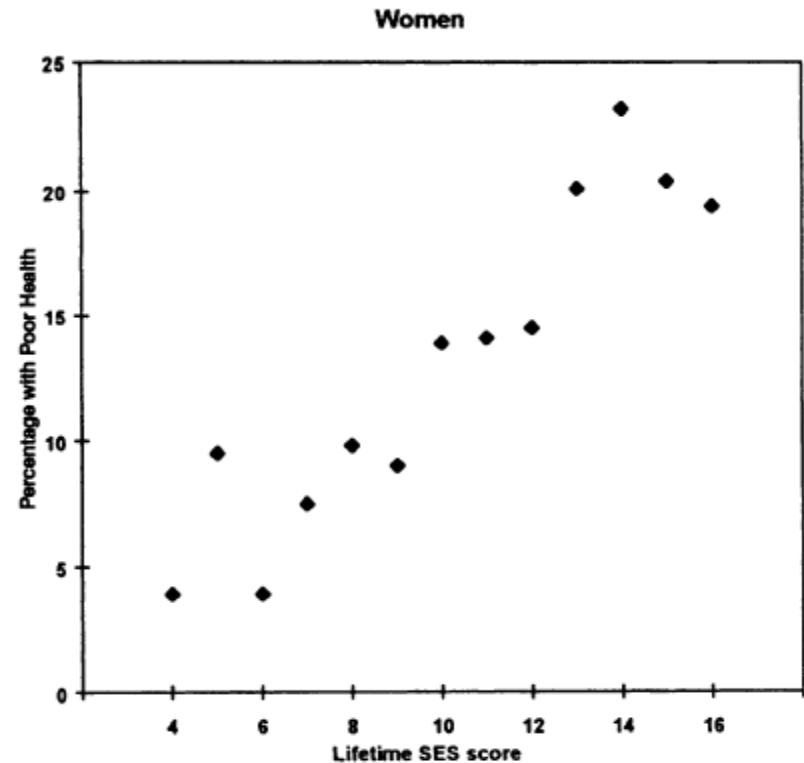
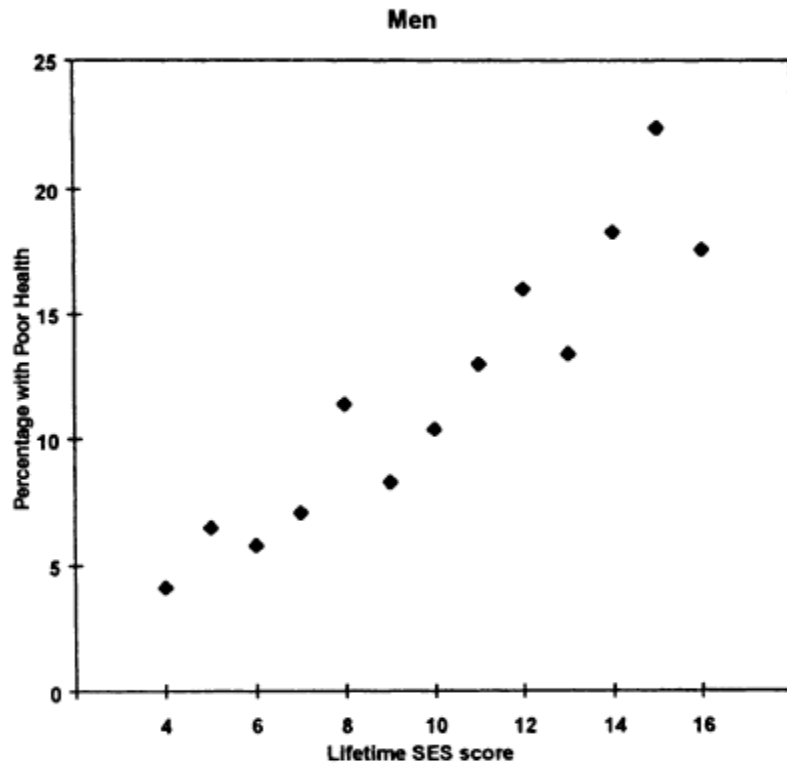
- Causal pathways
 - accumulation
 - chain of risk
 - trajectory
- Timing of causal actions
 - critical and sensitive periods

Causal pathways

- Accumulation
 - exposures (environmental, socioeconomic, behavioural) gradually accumulate to damage health as body systems age and are less able to repair themselves
- Chain of risk
 - a sequence of linked exposures that raise disease risk because one bad experience or exposure tends to lead to another and then another
- Trajectory
 - long term view of one dimension of an individual's life over time

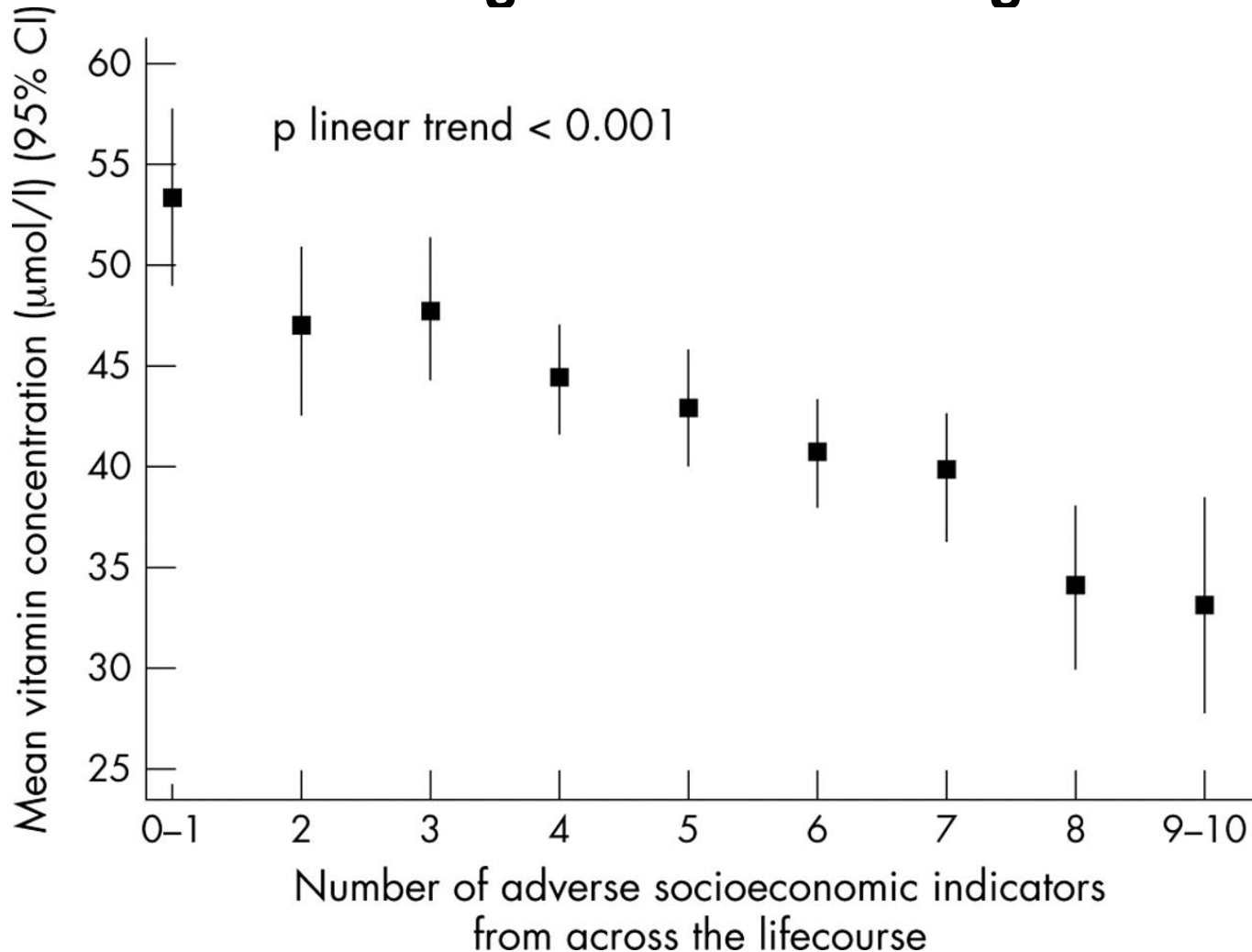
ACCUMULATION OF RISK

Cumulative social circumstances and health at age 33

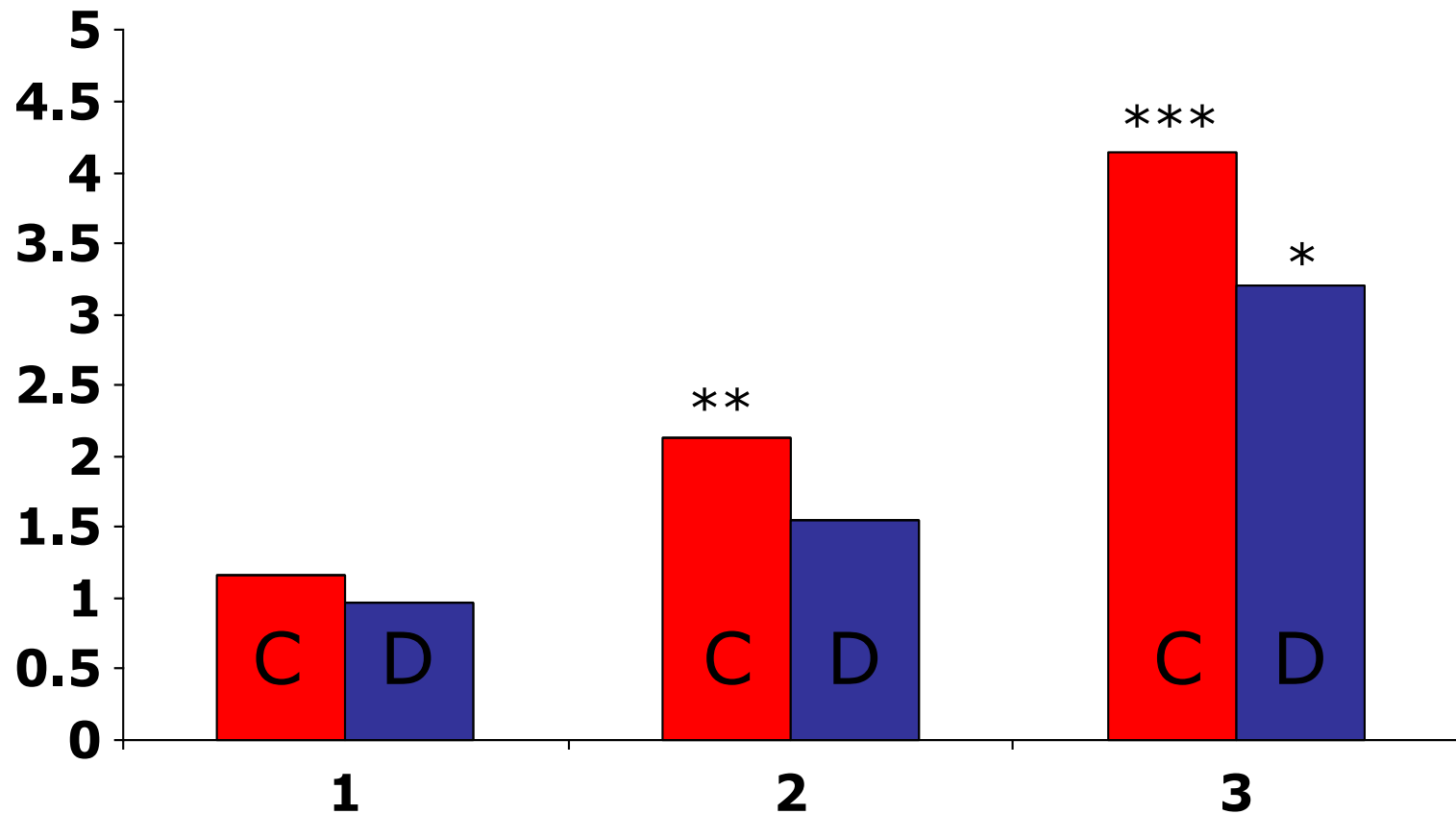


Note. SES = socioeconomic status.

Mean vitamin C concentrations by number of adverse life course indicators among British women aged 60–79 years.



Sustained economic hardship and odds ratio of self-reported cognitive difficulty (C) and depression (D) in late midlife (controlling for age, sex and prevalent diseases)



No. times household income below 200% of USA federal poverty level

Lynch, Kaplan & Shema, NEJM 1997

CHAIN OF RISK MODELS

A causal chain

"Why is Jason in the hospital?"

Because he has a bad infection in his leg.

But why does he have an infection?

He has a cut on his leg and it got infected.

But why does he have a cut on his leg?

He was playing in a junk yard next to his apartment building and fell on some sharp, jagged steel there.

But why was he playing in a junk yard?

His neighbourhood is run down. Kids play there and there is no one to supervise them.

But why does he live in that neighbourhood?

His parents can't afford a nicer place to live.

But why can't his parents afford a nicer place to live?

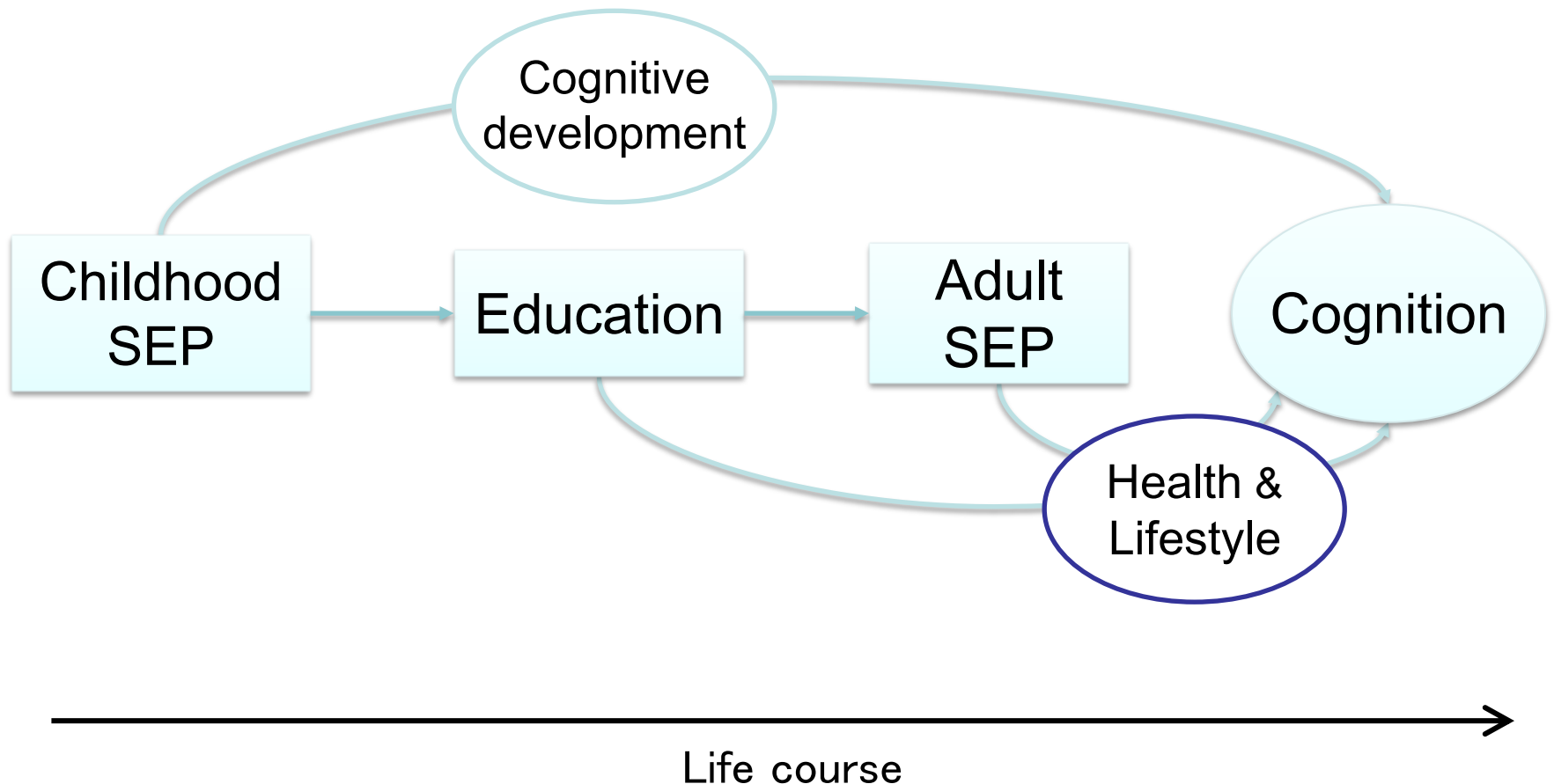
His dad is unemployed and his mom is sick.

But why is his dad unemployed?

Because he doesn't have much education and he can't find a job.

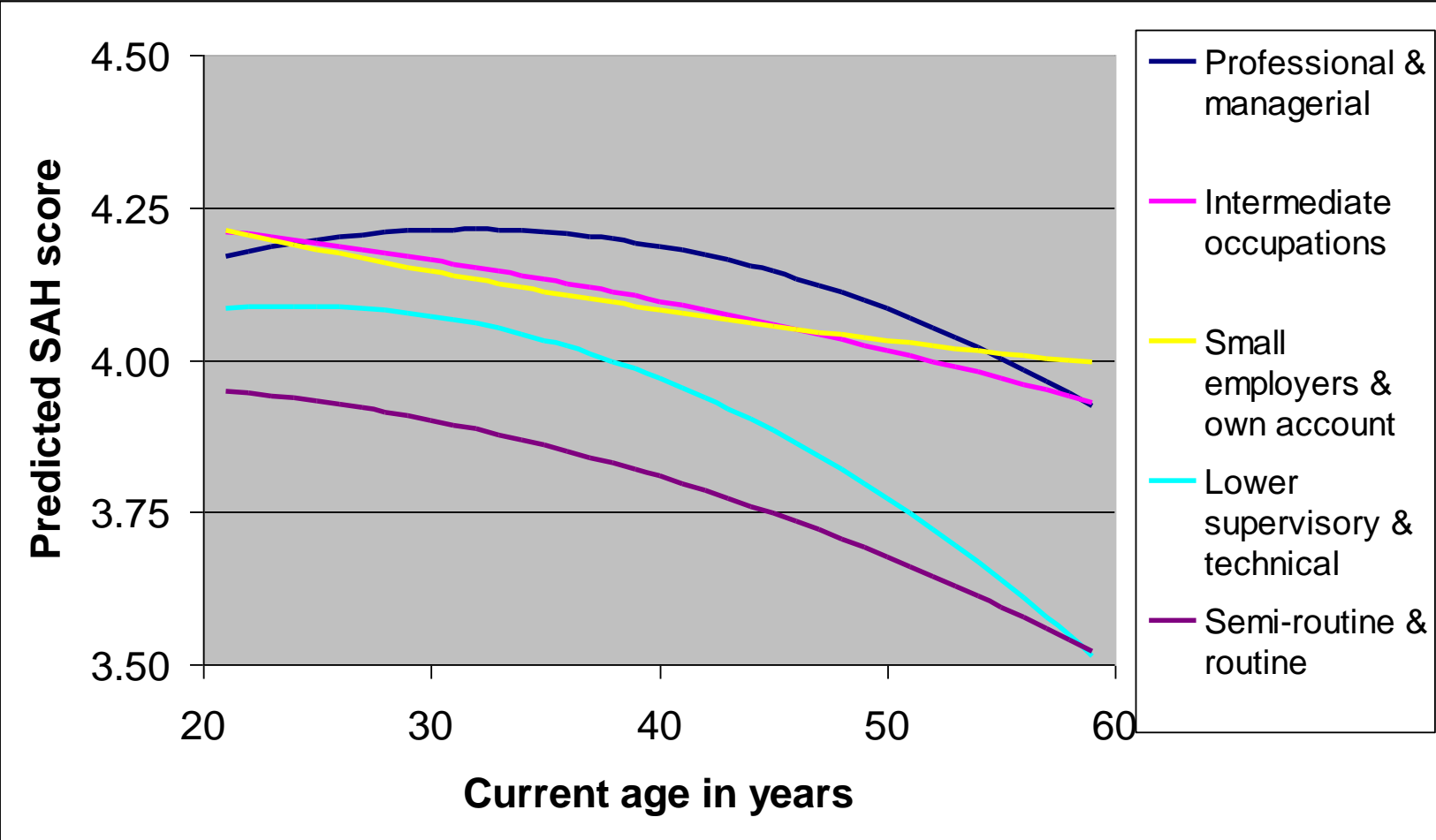
But why ... ?

Chains of causes of the life course: social position and cognition in later life

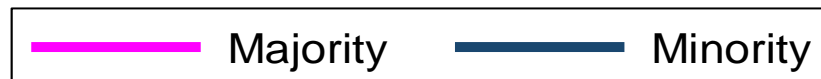
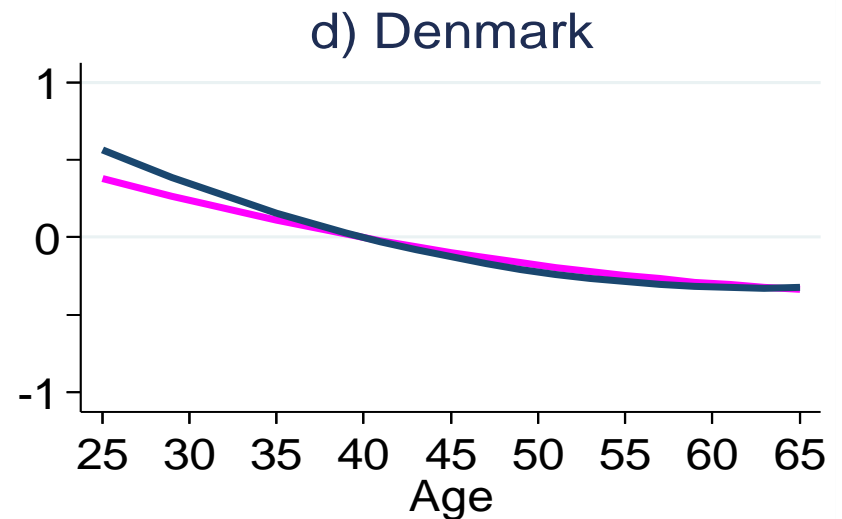
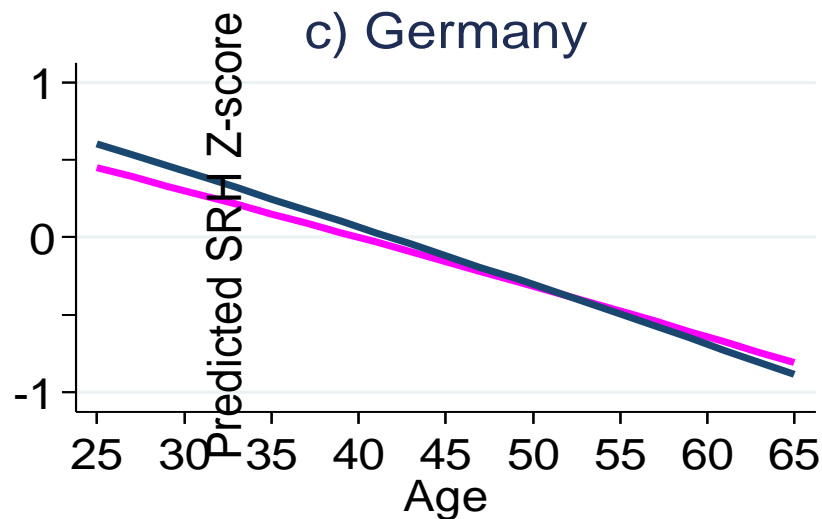
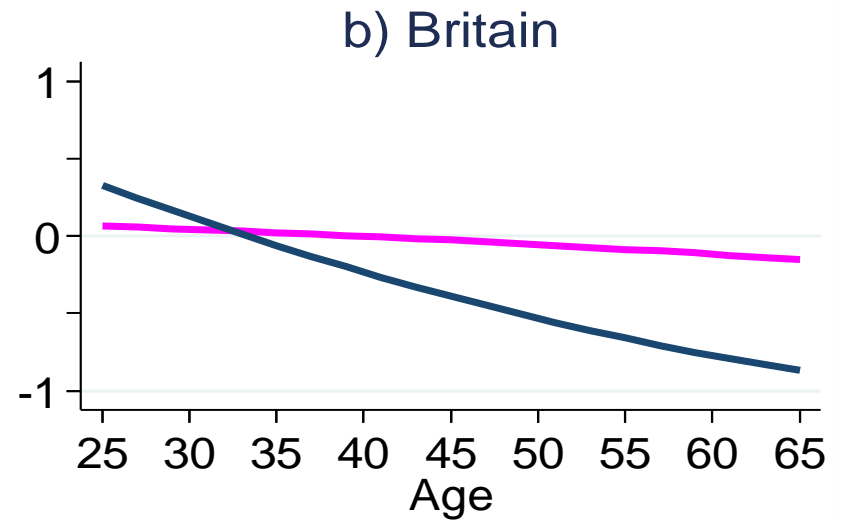
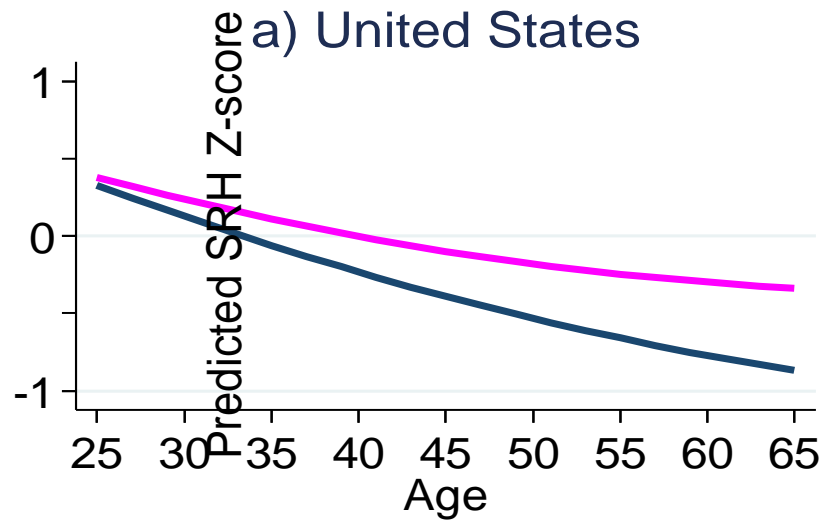


HEALTH TRAJECTORY MODELS

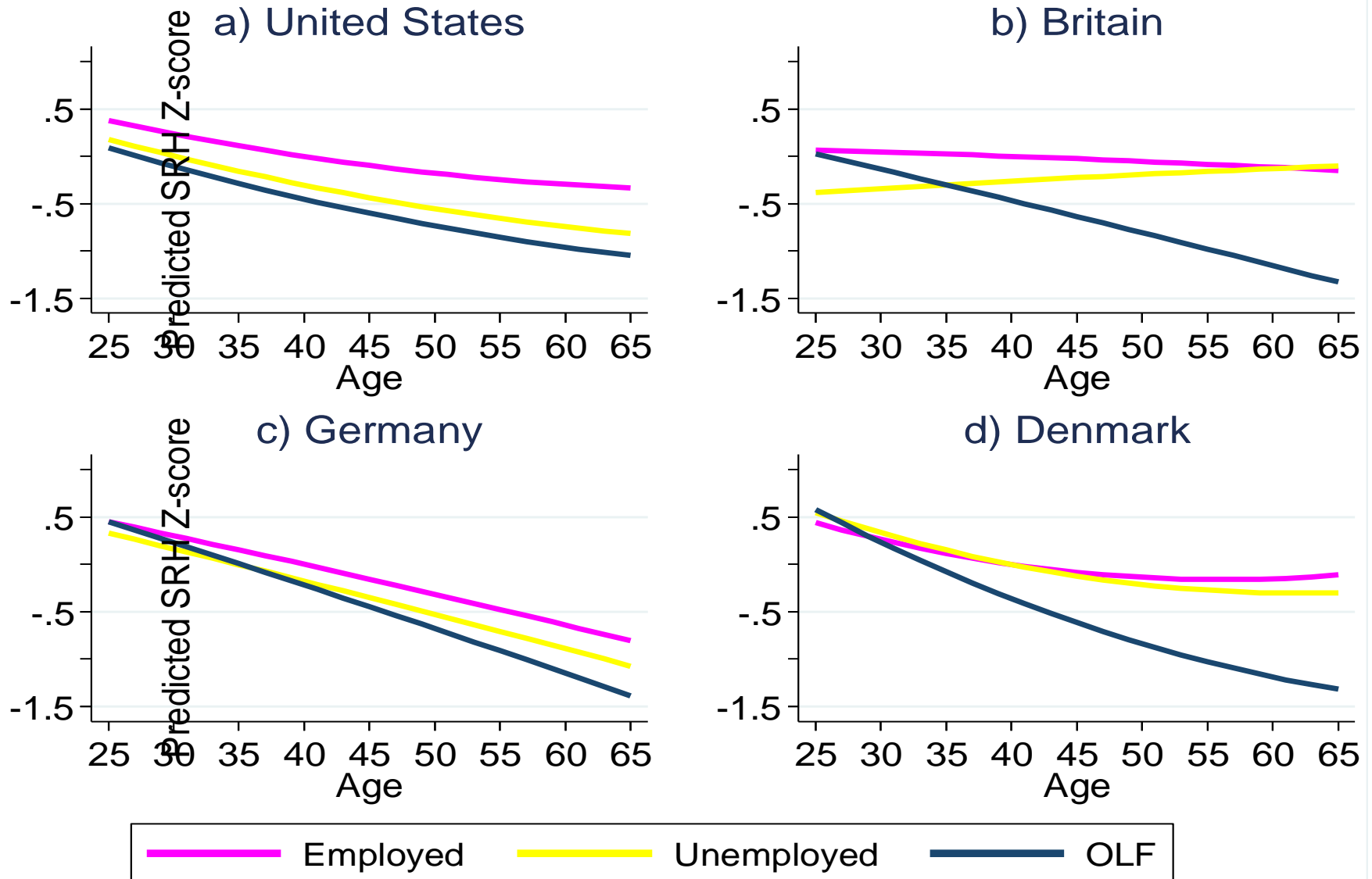
Mean predicted health scores by NS-SEC



Health decline by ethnic minority status



Health decline by employment status

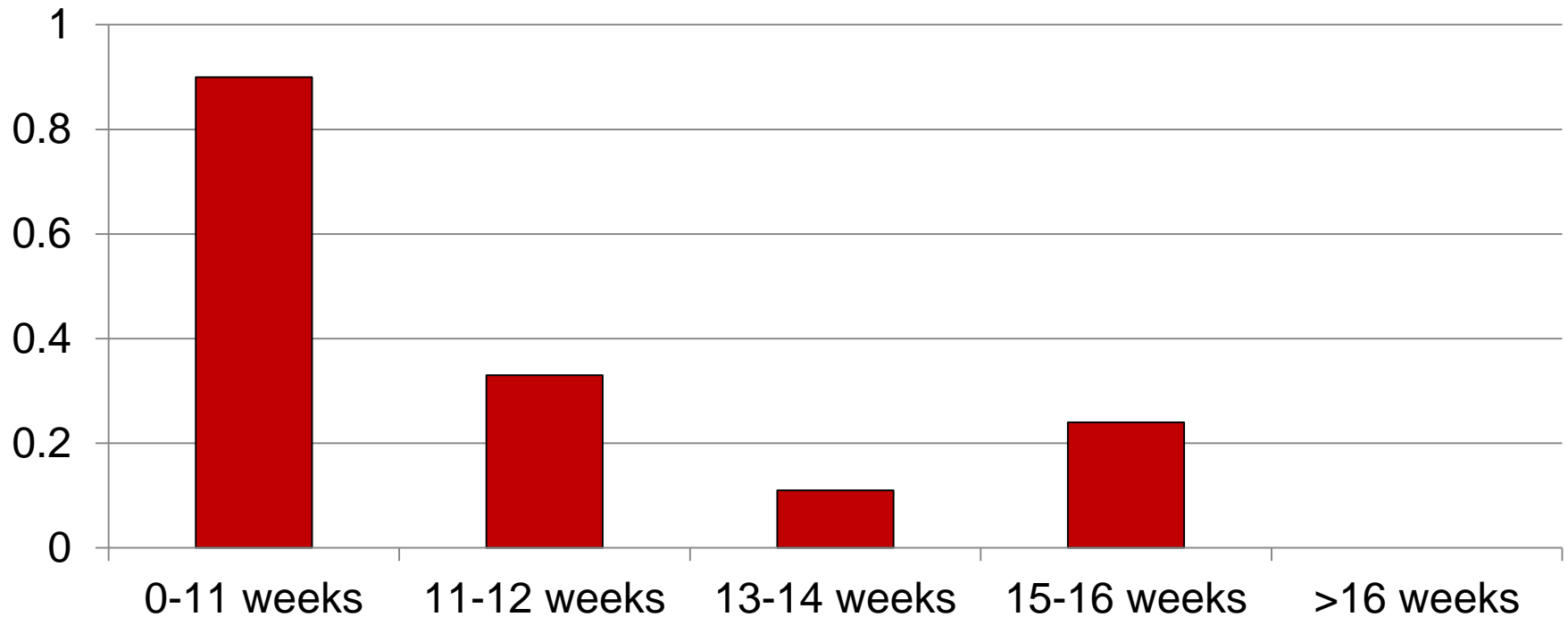


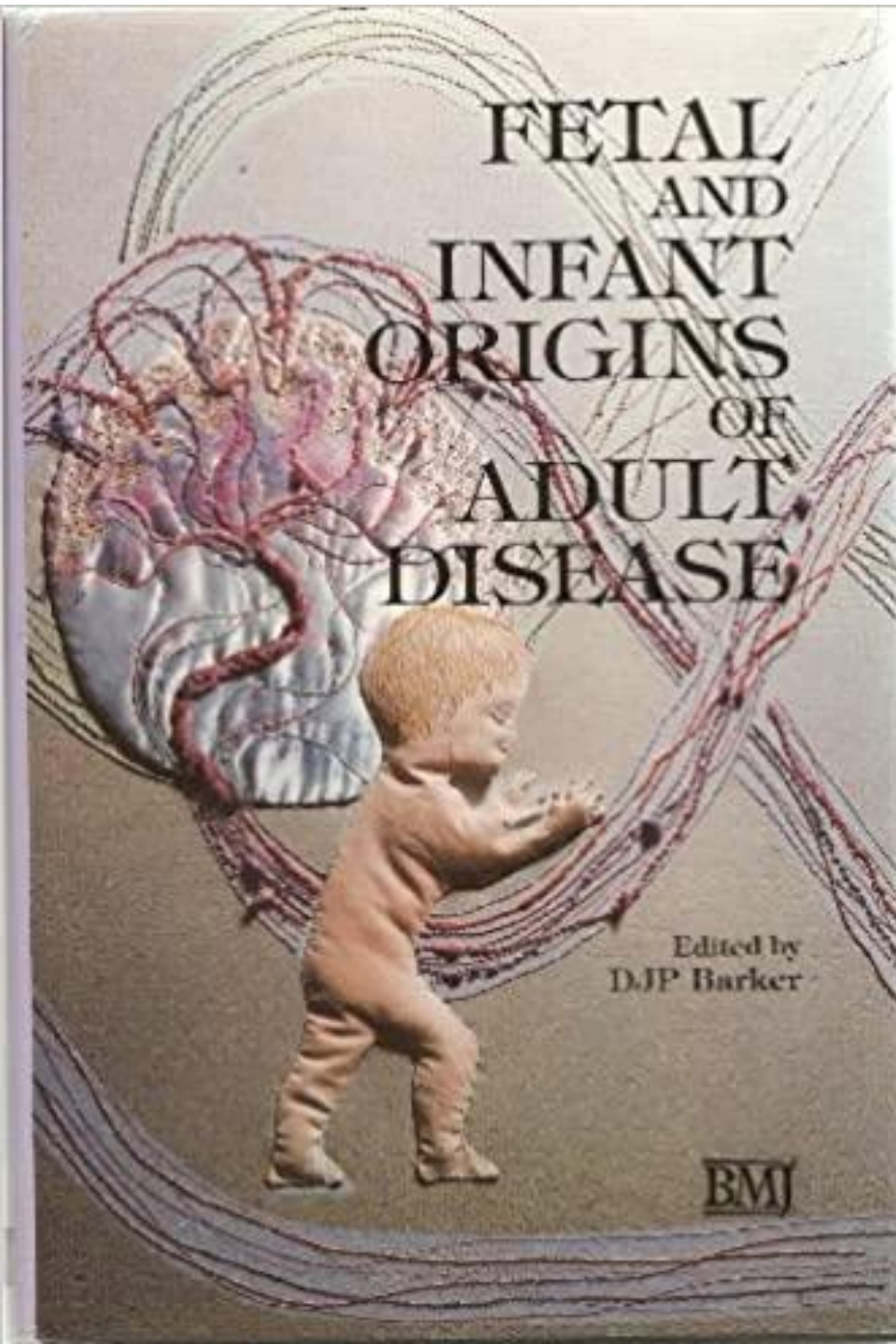
CRITICAL AND SENSITIVE PERIOD MODELS

Timing of causal actions

- Critical period
 - “biological programming” or “latency model” of disease
 - exposure has effects on body systems that cannot be modified in any dramatic way, precipitating disease later in life
- Sensitive period
 - Time when the individual is particularly sensitive to the environment
 - Increases risk but less deterministic than a critical period
 - Probabilistic

Timing of exposure to rubella in pregnancy and risk of congenital malformation

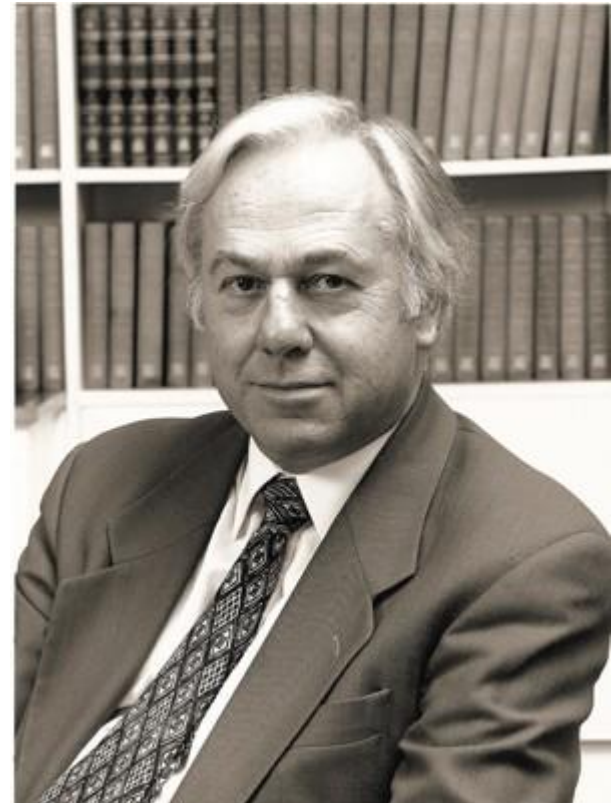


The book cover features a central illustration of a fetus in a blueish-purple hue, with a network of red and purple vessels extending from it. Below the fetus, a toddler in a brown outfit is walking towards the right. The background is a light beige color with faint, swirling lines. The title is printed in a large, black, serif font.

FETAL
AND
INFANT
ORIGINS
OF
ADULT
DISEASE

Edited by
D.J.P. Barker

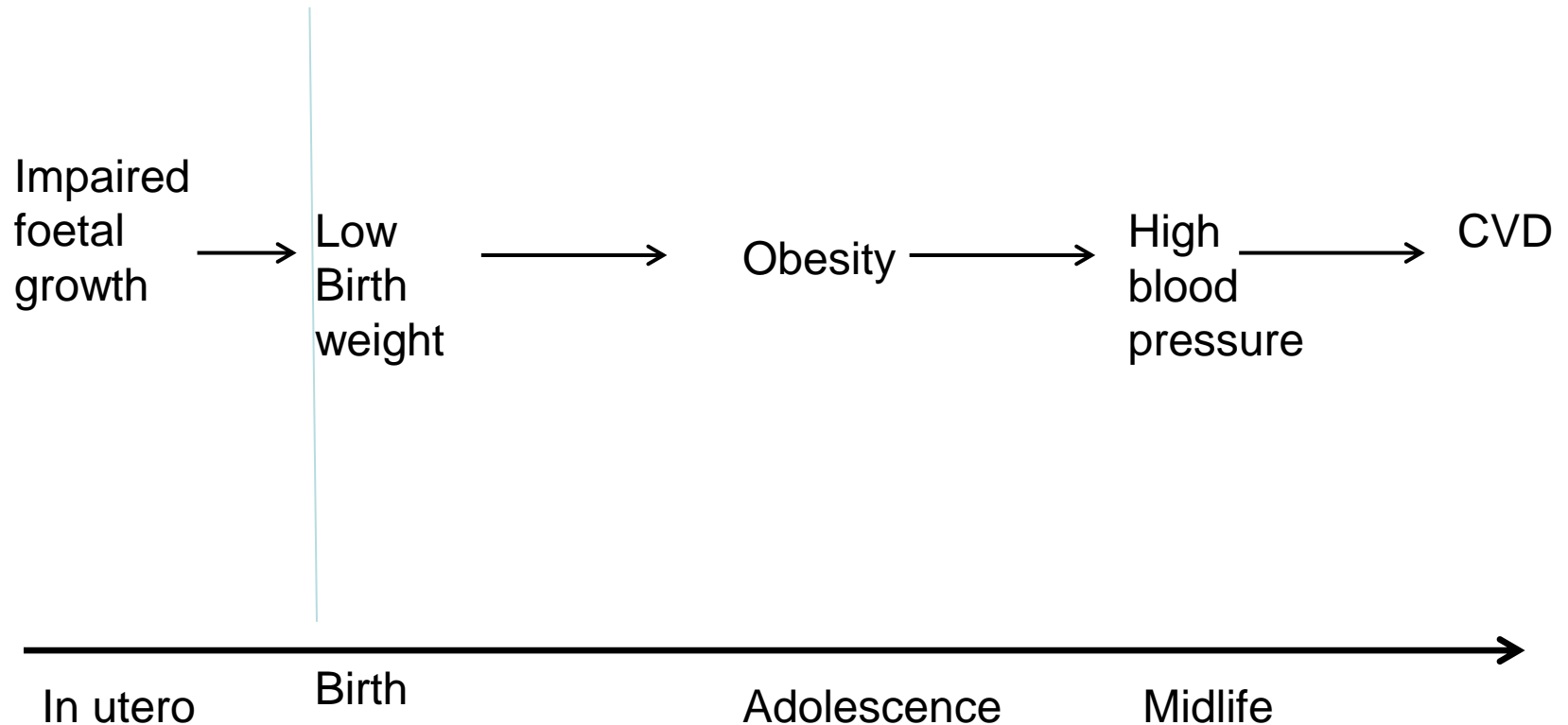
BMJ



Barker hypothesis

- Proposed in 1990 by David Barker
- Intrauterine growth retardation, low birth weight, and premature birth have a causal relationship to the origins of hypertension, coronary heart disease, and non-insulin-dependent diabetes, in middle age.
- The hypothesis was derived from a historical cohort study that revealed a significant association between the occurrence of hypertension and coronary heart disease in middle age and premature birth or low birth weight.
- Evidence remains inconsistent

Critical periods (foetal programming)



BIRTH WEIGHT

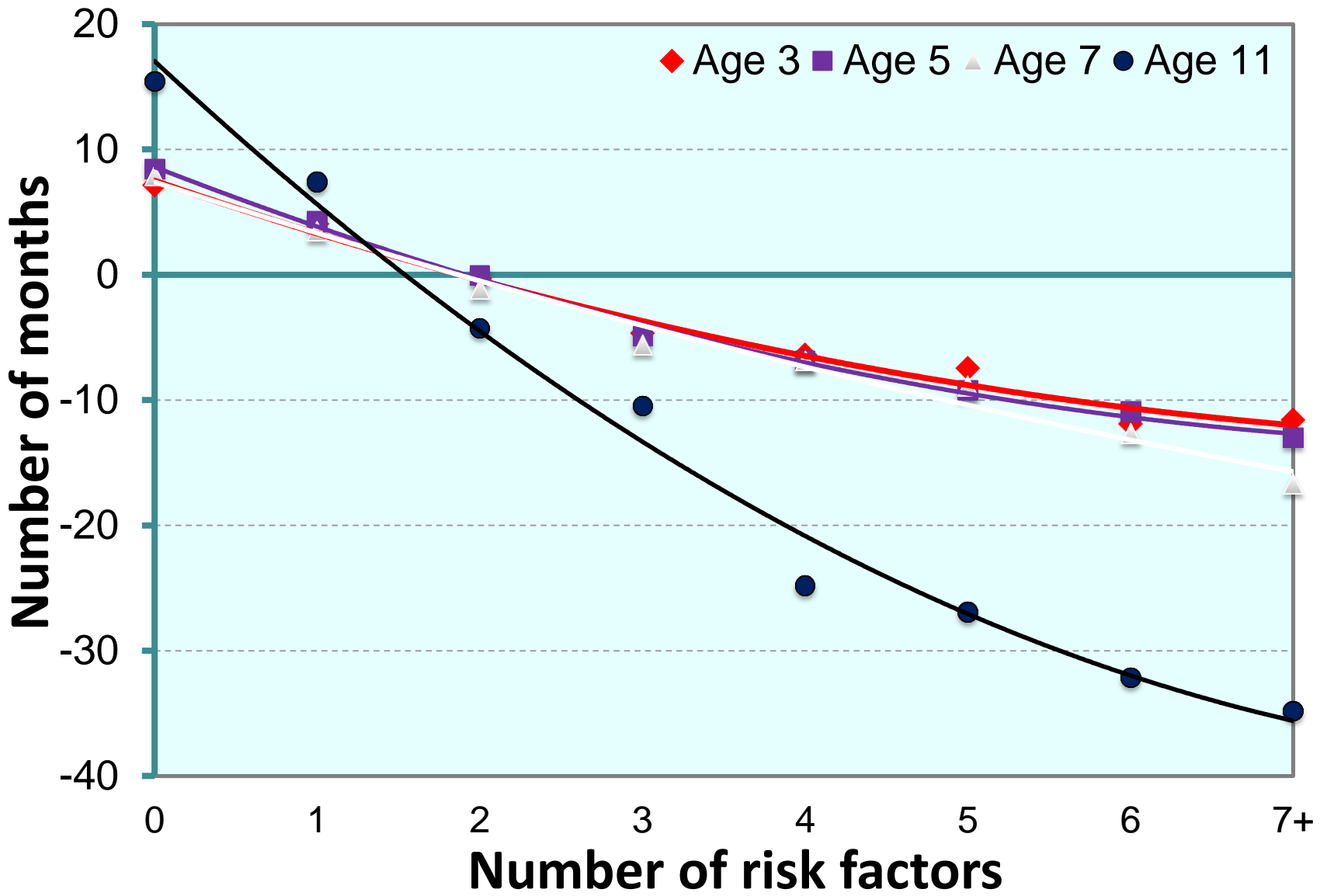
SYSTEMATIC REVIEW: SHENKIN ET AL.

Psychol Bulletin 2004; 130: 989-1013

“Small, consistent, positive association between birth weight and childhood cognitive ability, even when corrected for confounders”

- Record et al. Ann Human Genet 1969; 33: 71-79
- Matte et al. BMJ 2001; 323: 310-314
- Richards et al. BMJ 2001; 322: 199-203
- Shenkin et al. Arch Dis Child 2001; 85: 189-196
- Jefferis et al. BMJ 2002; 325: 305-308.
- Corbett et al. 2004 unpublished

Verbal ability: months ahead or behind by no. of risk factors

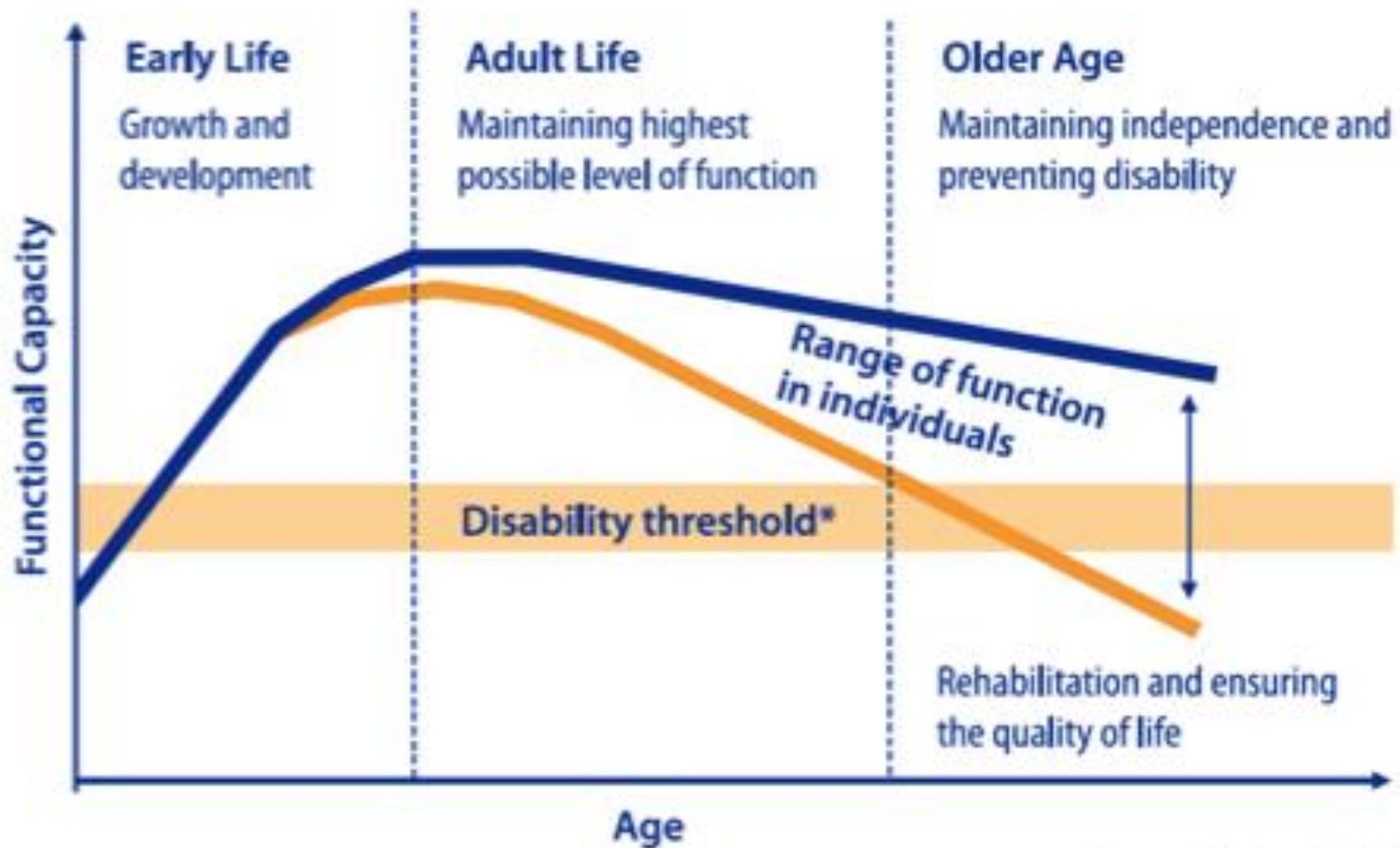


Early cognition and dementia risk

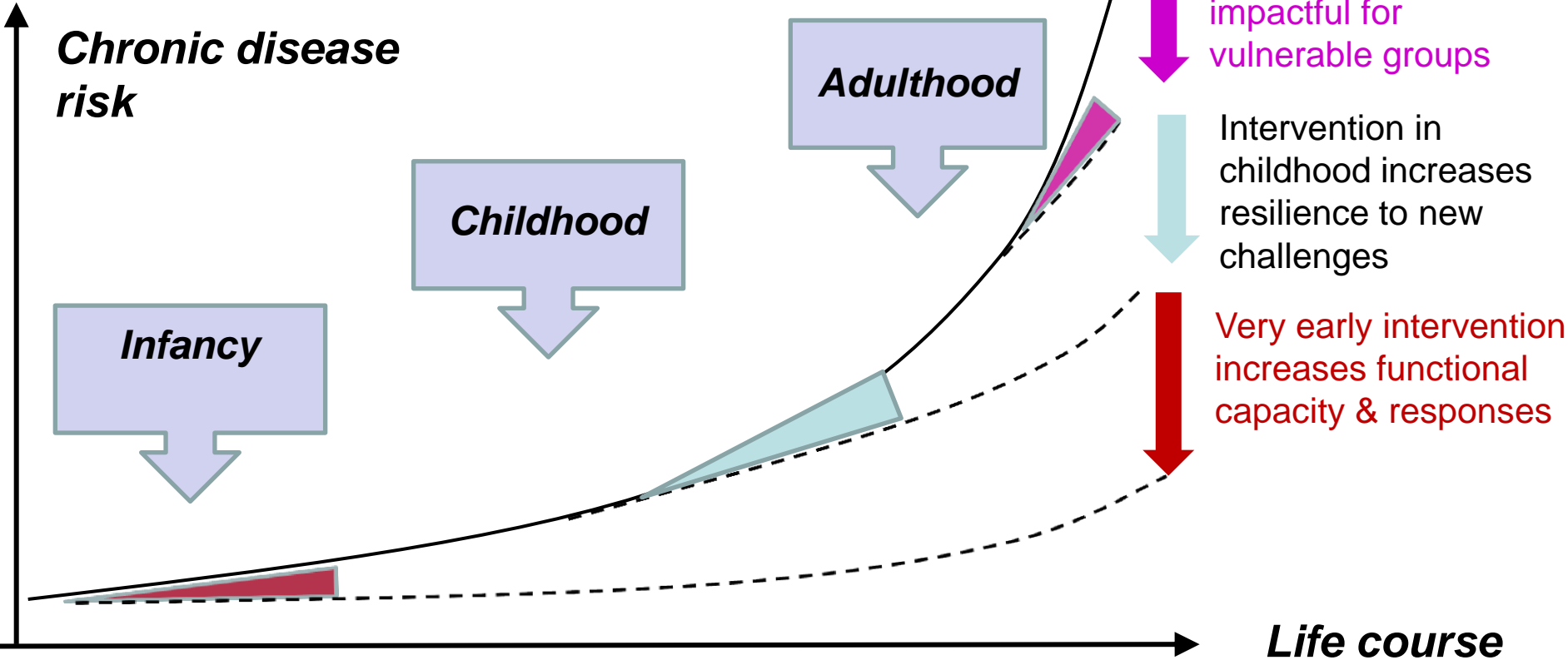
- **93 members of the School Sisters of Notre Dame born before 1917 in the Milwaukee area**
- **Idea density and grammatical complexity, derived from autobiographies written around age 22 years was a strong predictor of cognitive function and Alzheimer's disease risk in later life (Snowdon et al. JAMA 1996)**
- **Childhood IQ was associated with late-onset dementia (Whalley et al. Neurology 2000), but with VaD rather than AD (McGurn et al. Neurology 2008)**

A LIFECOURSE PERSPECTIVE ON POLICY INTERVENTIONS

Functional capacity across the lifecycle



Lifecourse strategy for disease prevention



Adapted from Godfrey et al DOI: <http://dx.doi.org/10.1016/j.tem.2009.12.008>