Heterogeneity and Accumulation Processes

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Late-Life heterogeneity

Tremendous heterogeneity exists within the older adult population

- Trajectories of functional impairment [Maddox and Clark, 1992]
- Reaction time [Hultsch et al., 2002]
- Perceptual-motor performance [Salthouse, 2013]
- etc.

Variability that is of importance, not just means!

Sources of heterogeneity

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   - Epigenetic modifications (DNA methylation that affects gene expression)
   - *Gene-Environment Interplay* (GxE)
     - Diathesis-stress: Dormant genes until turbulent environment
     - Social control: Environments *suppress* genetic effects
     - Social compensation: Environments *maximise* genetic effects

Sources of heterogeneity

1. **Multifacedness of ageing**
2. **Environmental influences and adaptation**
   - Agency in shaping/responding to environments
   - Selection into environments (deviation from the mean) (e.g., migration, military, social class)

Sources of heterogeneity

1. **Multifacedness of ageing**
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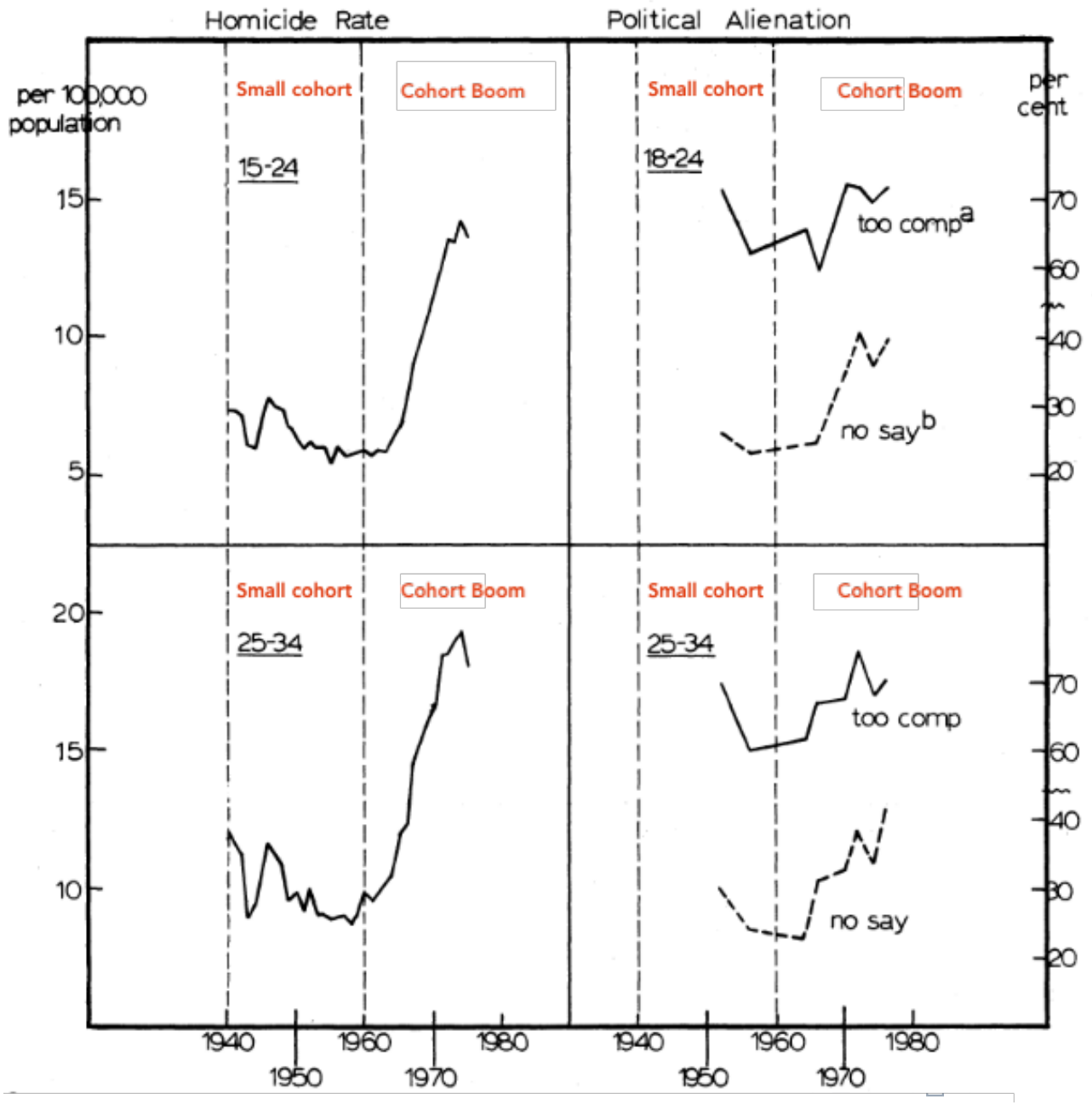
3. **Cohort effects**
   - Population shocks (armed conflicts, recessions, pandemics)
   - Easterlin hypothesis [Easterlin, 1978]
     - Inverse relationships between cohort sizes and health/mortality/socioeconomic outcomes
     - Large cohort size → Reduction in educational resources → Diminished educational attainments
     - Large cohort size → Increased worker supply → Diminished wage/job mobility & employment

Bigger cohorts → Increased adversity → Poorer outcomes


Homicide Rate
Political Alienation
per 100,000 population
per cent
Small cohort
Cohort Boom
15-24
18-24
25-34
too comp[a]
no say[b]
too comp
no say
15
10
5
20
70
60
40
30
1940
1950
1960
1970
1980

Sources of heterogeneity

1. **Multifacedness of ageing**
2. **Environmental influences and adaptation**
3. **Cohort effects**
4. **Stochasticity and within-person change**
   - Randomness in life events
   - Unit of analysis or error term (ϵ)?

Constraints of heterogeneity

1. **Survivorship bias**
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1. **Survivorship bias**
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2. **Selection in/out of studies**
   - *Informed consent* required in studies implies some degree of self-selectivity
   - Non-random attrition in longitudinal studies
   - Genetically influenced too! See Benonisdottir and Kong [2023]

How do researchers examine accumulation?

1. **Accumulation as outcome**
   - Risk factors of comorbidity (multiple diseases)
   - e.g., Educational attainment and **allostatic load** in later life [Ding et al., 2019]
2. **Accumulation as predictor**
   - Cumulative exposures on later life outcomes
3. **Accumulation as moderator**
   - Differentiated outcomes according to extent of accumulation
   - Cumulative adverse childhood experiences $\times$ Age predicted hair cortisol in later life [Iob et al., 2020]
4. **Reciprocal effects**
   - Cumulative risk $\rightarrow$ Health outcomes $\rightarrow$ Cumulative disadvantage

Material vs nonmaterial accumulation

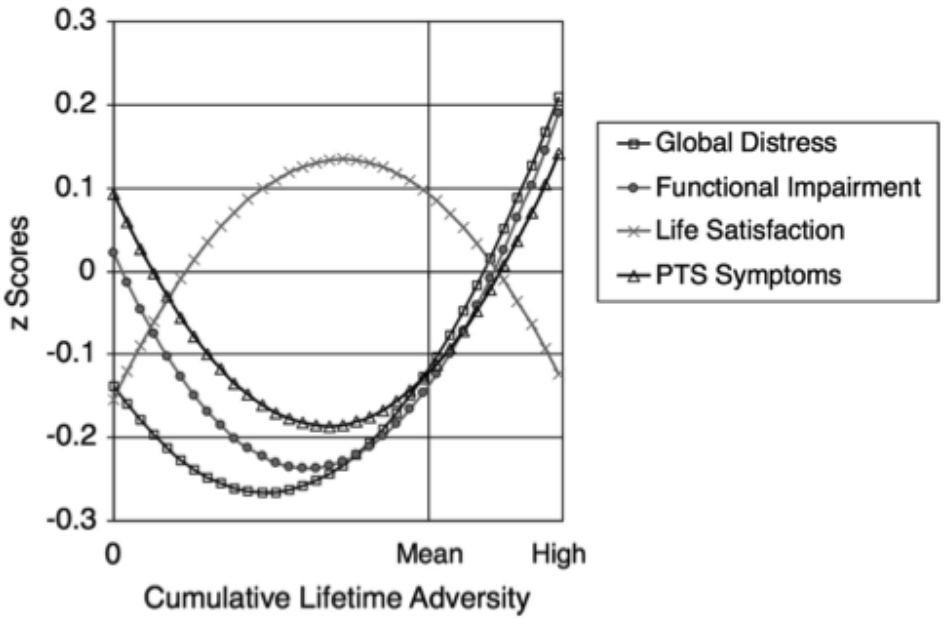
- **Material accumulation**
  - Observable and measurable
  - e.g., Lead, air pollution
- **Nonmaterial accumulation**
  - Cannot be observed directly
  - e.g., Discrimination, stress, adversity
    - Subjectivity in interpretation
    - Difficulty in measurement

Are exposures to varied types of stressors 'additive'?

Do they 'sum up' linearly to affect health outcomes?

Desirable vs Undesirable exposures

- Subjective nature of desirability
  - *"The strongest steel emerges from the fiercest of flames"*
  - *"Dough rises when you let it rest"*
- Non-linear effects of stress exposure on mental health [Seery et al., 2010]



Onset I

- **Critical/sensitive periods**
  - Developmental phase where exposures have the most impact
  - The Long Arm of Childhood – early life adversity linked to outcomes as late as mortality [Hayward and Gorman, 2004]

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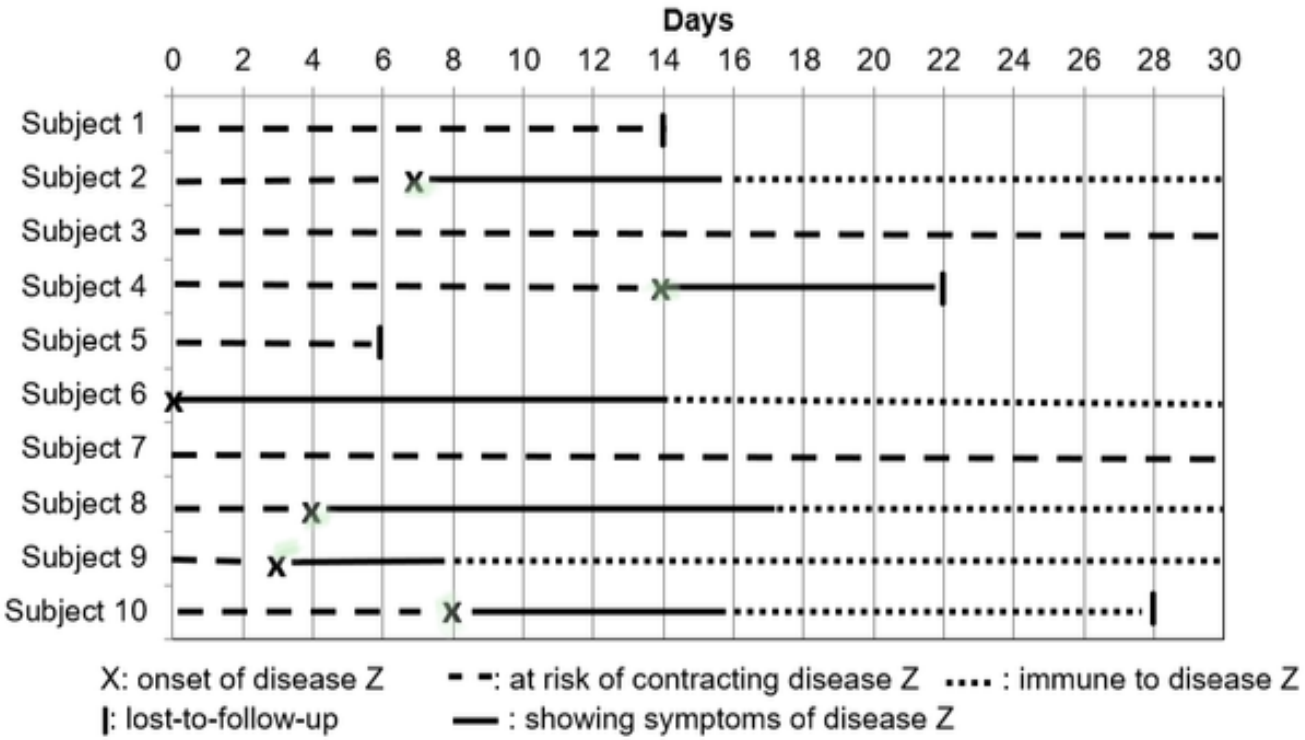
- **Duration**
  - Prolonged 'spells' of risk of exposure
- **Quantity of exposures**
  - Number of exposures over time
  - Thresholds – at what point does accumulation become detrimental?

Onset II

Rate of exposure



- Number of *cases* per unit time
- Weighted metric to account for unequal or inconsistent exposures across the sampled population

Onset III

Pace/tempo of exposure

- Intermittent vs continuous exposures
- Accelerating vs decelerating accumulations
- Halting or reversing accumulation

Overall useful to identify the *temporal patterns* of accumulation rather than just the *quantity* of accumulation

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Discussion Questions

1. How might survivorship bias affect our result interpretations empirically? Any examples of how it might affect your research, and/or how did you 'adjust' for it?
2. We discussed how heterogeneity might arise simply due to stochastic processes. Do you think we should treat such stochasticity as a unit of analysis? Might there be an underlying structure behind such stochasticity?
3. In measuring cumulative risks, what are your thoughts about summing up different kinds of of non-material exposures (e.g., depression, poverty) *linearly* to create a single 'cumulative risk score'?
4. Apart from critical periods, duration, and the pace of exposure, can you think of how the temporal dimension of exposures might affect later life outcomes? And/or: what do you think about the **recency/proximity** of exposures? Is it worth investigating?
5. Feel very free to raise your personal thoughts, comments, or questions about these chapters!

Thank you!

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