Inequity in Mind

On the Social and Genetic Risk Factors of Dementia and Their Interactions

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- I. Hasselgren C., Ekbrand H., Fässberg M.M., Zettergren A., Zetterberg H., Blennow K., Skoog I., & Halleröd B. (2018). APOE ε4 and the long arm of social inequity: Estimated effects of socio-economic status and sex on the timing of dementia onset. Ageing & Society, 39(9): 1951-1975.
- II. Hasselgren C., Dellve L., Ekbrand H., Zettergren A., Zetterberg H., Blennow K., Skoog I., & Halleröd B. (2018). Socioeconomic status, gender and dementia: The influence of work environment exposures and their interactions with APOE ε4. Social Science & Medicine (SSM) Population Health, 5: 171-179.
- III. Wu J., Hasselgren C., Zettergren A., Zetterberg H., Blennow K., Skoog I., & Halleröd B. (2018). The impact of social networks and APOE & on dementia among older adults: Tests of possible interactions. Aging & Mental Health. Advance online publication, https://doi.org/10.1080/13607863.2018.1531368.
- IV. Hasselgren C., Ekbrand H., Halleröd B., Fässberg M.M., Zettergren A., Johansson L., Skoog I. & Dellve L. (submitted manuscript). Sex differences in dementia: On the potentially mediating effects of educational attainment and experiences of psychological distress.



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Abstract

The present thesis seeks to further explain the occurrence of Alzheimer's disease and other dementias by studying the long-term impact of class- and gender-based inequities as well as the extent to which they potentially moderate genetic risk. Central to this endeavour is the recognition of social inequity as multifaceted, and of potential intersections between different drivers of structural (dis)advantage in relation to individual health prospects. The main point of departure is that even though the causes of dementia are heterogeneous and cannot be reduced to either genetic or environmental factors, dementia is, just like many of its potential risk/protective factors, unevenly distributed in the population. Nevertheless, our knowledge of whether and how systems of structural inequity intersect and interact with individual genetic endowments in the development of disease is still scarce.

The thesis encompasses four empirical studies, all of which should be considered examples of interdisciplinary efforts to incorporate theory and expertise from different fields in order to create a more holistic understanding of dementia aetiology. The analyses are based on data derived from the longitudinal Gothenburg H70 Birth Cohort Study (H70) and the Prospective Populations Study on Women (PPSW) from Gothenburg, Sweden. The baseline sample (N =1019) was first examined in 2000 and followed up in 2005 and 2009.

Study I lays the foundation upon which the other studies rest. It does so by asking whether socioeconomic status (SES) could in fact moderate the increased risk of dementia that carrying one or more copies of the APOE (apolipoprotein E) &4 allele implies. Having identified that high SES seems to buffer the effect of APOE £4 among men but not among women, Study II and III set out to explore two mechanisms that could possibly shed further light on the link between socio-economic (dis)advantage and dementia risk as well as on the previously identified sex difference: work environment exposures and access to social networks. The findings of Study II suggest that work control is the most influential aspect of the work environment, with respect to moderation of genetic endowments, but that it is only protective among men. While no significant gene-social network interactions were revealed in Study III, the results indicate that there might be important differences between men and women in the impact of social networks on dementia risk. Finally, Study IV tests the assumption that the higher lifetime risk of dementia among women could, at least in part, be the result of differences in educational attainment and/or in experiences of general psychological distress. The results confirm that education ought to be considered a 'gendered' dementia risk factor and propose that psychological distress constitutes a potential, and hitherto rarely acknowledged, pathway between dementia and female sex, on the one hand, and dementia and low educational attainment, on the other.

In light of the findings presented in this thesis, it is evident that dementia is an emergent phenomenon that must not be reduced to the sum of its parts, especially considering the results suggesting that genetic endowments can actually be moderated by externally imposed factors. Additionally, all four studies underline that the risk/protective factors that are more proximate to the individual, such as work environment exposures, social networks or distress, must not be studied as if they were distinct from the social structures that 'put people at risk of risks'. Consequently, I argue, class and sex/gender must be attended to as fundamental, and intersecting, causes of dementia if we are to better understand why some individuals develop the disease, while others do not.

Key words: Dementia, APOE ε4, health inequity, longitudinal population studies, class, sex/gender, fundamental cause theory