PER-OLA SUNDIN (1971) received his medical degree from Uppsala University in 1996. He completed his period as a registrar in Internal Medicine between 1998-2003, and in Nephrology between 2003-2005 at the Örebro University Hospital. He is currently working as a Consultant in the Department of Geriatric Medicine, Örebro University Hospital. He registered as a PhD student at Örebro University in 2012 and has conducted his research at Clinical Epidemiology and Biostatistics under the supervision of Scott Montgomery, Professor in Clinical Epidemiology, Örebro University. His research interests include studying pathways to adult Chronic Kidney Disease (CKD) and assessment of individual future risks of adverse events following identification of CKD.

This thesis utilizes routinely collected health data from Swedish National Registers and The Swedish Military Conscription Register to examine risk factors for CKD from a life-course perspective. This includes investigating potential markers in adolescence of increased risk for End-Stage Renal Disease in middle-age. Another project assesses potential long-term increased risk of CKD following severe infections. The risk of subsequent CKD after hospital admission with pneumonia, including the temporal pattern of risk, indicates a high magnitude raised risk of CKD in the years immediately following infection, then declining but remaining raised for more than 15 years after the pneumonia episode.

A second theme of this thesis is identification of the increased all-cause mortality risk associated with CKD. Endogenous filtration markers used clinically to estimate the Glomerular Filtration Rate (GFR), which is the most important measure of kidney function used to identity CKD, are associated with all-cause mortality risk. Potential associations with all-cause mortality risk for the filtration markers creatinine and cystatin C independent of GFR, as determined by the clearance of an exogenous filtration marker, are investigated and the utility of combining these measures to predict mortality risk optimally is evaluated. Another project investigates potential effect modification by grip strength for the association between estimations of GFR from creatinine and all-cause mortality risk, or in other words, whether individuals with different levels of muscle strength as determined by grip strength, have different levels of vulnerability to the adverse effects of reduced kidney function in terms of increased mortality risk.

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