Currently reading Case 2014.

Do I want to try to impute any missing values wrt. Sleep?

What is the validity and reliability of self-reported measures of sleep?

Do I want to exclude anyone who would have CRP values related to things I’m not interested in? For instance, history of inflammatory disorders (although this may be part of what I’m interested in measuring, but do I want to mix it with future risk?), high levels due to trauma or infection? These are markers of immune reaction, so what exactly am I trying to mark?

This makes me realize I need to learn more about CRP. What is its range of biological roles, and how has it been used in public health?

Case 2014 has a good description of the methods for sampling and storing CRP.

Will any transformation be done on CRP?

Case 2014 adjusted for diabetes, antihypertensive medication use, lipid-lowering medication use, oral contraceptive use, HRT use. Do these have an effect on CRP?

Classification of education level in relation to age – would the automatic lack of education bias towards the null? i.e. effect of SES would be clouded by potentially high SES young people. With that in mind I may want to exclude people under the age of say, 22.

Potential mediators explored by Case 2014: BMI, current smoking, alcohol use. They provide literature for this.

Case 2014 evaluated internal consistency (Cronbach alpha) and inter-item correlation of the depressive symptom index.

Reading Covassin 2016

U-shaped association between sleep duration and hypertension risk in cross-sectional studies. Possibly using an absolute value of the difference from mean?

However a meta analysis of prospective studies mainly support short sleep as the independent risk factor for hypertension (9 in covassin). Women have been found more susceptible, and a couple of studies support a race interaction whereby the risk is heightened for non-Hispanic Blacks.

Didn’t finish.

Reading Jackowski 2013

Experimental evidence that sleep impacts health usually have short follow up, so observational studies compliment.

Probably a larger association in the elderly but little evidence. Sleep is a particularly modifiable risk factor. Inconsistent associations between CRP and sleep parameters.

Uses a cutoff of 3 mg/L for CRP and provides literature. Controls for age, wealth, BMI, current smoking, physical activity, limiting long standing illness, and depressive symptoms, and provides literature. Tested a curvilinear association btw sleep duration and inflammation using the 7-8hr category as the reference group.

Probably important to look at fibrinogen too, because lack of association with fibrinogen makes it less likely a real association with inflammatory processes.

Mechanisms for long sleep duration are less well understood than for short duration.

It seems (from the discussion) that various studies will find the association in one group and not in another, and that these differences are not consistent. For example some found it in men but not women, and vice versa, some found it only in African Americans, some found it stronger in whites.

Reading Pollit 2007

Strongest association of CRP with adult SES, less so with childhood and less so with neighborhood. Suggestion of mediation by BMI, smoking, and HDL.

Some good references in the beginning for SES->CVD association as well as SES→inflammation, although these might be out of date. Early life SES interest stimulated by the fetal origins of adult disease hypothesis. Also some good references about the role of sustained elevated inflammatory response and atherosclerosis and metabolic syndrome risk (although isn’t this disproved by the MR study?)

Low ICC in hierarchical models for whites indicating low impact of neighborhood SES compared to individual. No evidence for need for hierarchical model in blacks. Stronger association in whites than blacks, but interaction term??

Effects of SES on inflammation levels were modest -- will this be enough to find a significant mediation effect of sleep?

Reading Hall 2015

Sleep duration predicts all-cause mortality (with 8 citations). Both short and long sleep as predictors of mortality supported by meta analyses, experimental for short, but no experimental evidence for long.

Primary study objective was to determine if the link between sleep duration and all-cause mortality is mediated by inflammatory markers.

They adjusted for a wide range of chronic diseases and medications related to either sleep or inflammation. I may want to exclude people using sleep or inflammation medications.

In crude mortality rates, lowest among 7hrs, highest among >8hrs.

They also set 7hrs to be the reference group.

In the adjusted model, the HR for short sleep on mortality was more attenuated with the addition of inflammation, whereas the long sleep HR was primarily attenuated by health covariates.

Limitations: lack of ability to generalize to younger adults.

Reading Prather 2013

Prospective. Poor PSQI subjective sleep quality predicts 5-year increases in fibrinogen, IL-6, and CRP independent of confounders in women, but not men.

Adjusted for age, gender, ethnicity, education, BMI, snoring, smoking, physical activity, alcohol, depressive symptoms, hypertension, MI, CHF, T2D, stroke, beta-blockers, statins, ACE inhibitors, HRT/oral contraceptive, antidepressant, benzodiazepine history, cardiac function (SBP, resting LVEF)

Interaction for gender.

Reading Grunewald 2009

Intro has references for SES--> inflammation, inflammation-->CHD/diabetes/mortality, SES-->physical activity/smoking-->infammation.