### Considerations for Intro

What exactly are all the conditions associated with CRP / inflammation?

Fibrinogen is a measure of coagulation and therefore primarily implicated in CVD and perhaps not other inflammatory disorders. Do I want to stick to CVD risk? Probably.

Is there sufficient evidence that inflammation is causal? CRP is probably not, but inflammation?

Is there sufficient evidence that sleep causes inflammation and not vice versa?

What studies show an association between sleep duration and CRP, fibrinogen, versus did not?

What studies show an association between sleep quality and CRP, fibrinogen, versus did not?

What are the reasons SES would be associated with sleep?

Mechanisms for short vs. long sleep -- more clearly defined in short, e.g. increased blood pressure and heart rate, altered metabolic, endocrine, and catecholamine signalling, all relating to increased sympathetic activation and pro-inflammatory response (discussion of Hall 2014).

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### Considerations for Methods

How were blood samples collected and stored?

How were fibrinogen and CRP measured?

Will fibrinogen and CRP be transformed, dichotomized?

How was sleep measured and what are its validity and reliability?

How is SES operationalized? Is there a summation score, or separate measures?

What are the confounders to adjust for? I.e. what else affects inflammation besides sleep? BMI, smoking, alcohol, diet, diabetes, HDL/LDL, physical activity, conditions such as arthritis and sleep apnea which could interfere with sleep and cause inflammation through a separate pathway?

* Different sets of confounders for the SES-> sleep relationship vs. the sleep-->inflammation relationship.
* The latter involves other mediators of SES-->inflammation such as stress, access to health care and food, exposure to infection, and health behaviors. It also involves direct confounders like medications and chronic diseases that would interrupt or change sleep patterns and also be caused by inflammation -- reverse causality?
* The former there are fewer. Things that cause SES could be a major illness, but this is less likely.

Probably need to start a DAG.

How exactly will I like at direct, indirect, and total effect, and how will I test for significance given the survey methodology?

* Kershaw 2010 used probit regression -- thought to be more accurate for mediation

Whether to test for interactions with gender, race. A number of studies found associations in women but not men (introduction of Prather 2013).

Whether to impute missing values

How to classify sleep duration? Hall 2014 does <6, 6, 7, 8, >8.

Does NHANES measure WBC or TNF-a? Should I include these?

What medications affect sleep, inflammation?

## Considerations for Discussion

How do the associations I found for SES -> sleep, SES -> CRP, and sleep → CRP compare to the literature? If I didn’t find as big of an effect, would a bigger effect in other studies indicate higher likelihood of mediation?

In studies finding mediation by BMI and physical activity and smoking, was the association fully explained or was there an unexplained portion? If so, hypothesize other mediators that might be present?

Effect of participation rate, selection bias? Was there social desirability bias in sleep questions? What about income and education questions?

Very little missing data for main variables, only for confounders. However, confounders may be poorly controlled as a result.