Response to: 'Inflammatory and non-inflammatory triggers of acute coronary syndromes' by Eyuboglu

We thank Dr Eyuboglu for his comments¹ on our article 'Siblings of rheumatoid arthritis patients are at increased risk for acute coronary syndrome'.²

Familial risks tell us if risk factors for a disease are shared within families. By studying familial risks, we can also investigate shared susceptibility between diseases. In our present study, we have, by using the full siblings of patients with rheumatoid arthritis (RA), created a study population that are enriched for both the genetic and (familial) environmental risk factors involved in RA. If any disease has an increased frequency in our cohort of siblings of patients with RA, a cohort with an overrepresentation of RA risk factors compared with the general population, it provides evidence for a shared susceptibility between that disease and RA.

What a potential observed risk increase does not tell us is whether it is due to genetic or environmental factors. And, as Dr Eyuboglu points out, it also does not tell us what type of underlying mechanisms are involved. But it does tell us that there is a relationship between the two diseases, regardless of mechanisms of actions.

We share Dr Eyuboglu's interest in what the underlying mechanisms are and completely agree that knowing more about the pathophysiology underlying the identified acute myocardial infarctions (AMI) among the acute coronary syndromes (ACS) in our study population would be of great interest. Unfortunately, that information was not within our data and it was not possible for us to shed light on this in our present study. We do know, however, that close to 90% of the AMI registered during 2011 in Sweden were type 1 AMI,³ meaning that the majority of the events among the siblings of the patients with RA, are likely to be type 1 AMI with an underlying atherothrombotic coronary event.

So in conclusion, the increase in ACS risk among the siblings tells us that there is a family-shared link between RA and ACS, but it does not tell us how and why. We thus agree with Dr Eyuboglu that looking further into the underlying mechanisms of the ACS events are of great interest and importance to learn more about the disease mechanisms, and we hope to study this further in coming studies.

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Competing interests JA has or has had research agreements with Abbvie, Astra-Zeneca, BMS, Eli Lilly, MSD, Pfizer, Roche, Samsung Bioepis and UCB, mainly in the context of safety monitoring of biologics via ARTIS/Swedish Biologics Register. Karolinska Institutet has received remuneration for JA participating in advisory boards arranged by Pfizer and Lilly.

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