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Anger is associated with subclinical atherosclerosis in low SES but not in higher SES men and women. The Cardiovascular Risk in Young Finns Study

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Abstract We investigated the associations of anger and cynicism with carotid artery intima-media thickness (IMT) and whether these associations were moderated by childhood or adulthood socioeconomic status (SES). The participants were 647 men and 893 women derived from the population-based Cardiovascular Risk in Young Finns Study. Childhood SES was measured in 1980 when the participants were aged 3–18. In 2001, adulthood SES, anger, cynicism, and IMT were measured. There were no associations between anger or cynicism and IMT in the entire population, but anger was associated with thicker IMT in participants who had experienced low SES in childhood. This association persisted after adjustment for a

host of cardiovascular risk factors. It is concluded that the ill health-effects of psychological factors such as anger may be more pronounced in individuals who have been exposed to adverse socioeconomic circumstances early in life.

Keywords Atherosclerosis · Intima-media thickness (IMT) · Cynicism · Anger · Hostility · Socioeconomic status (SES)

Anger and hostility are considered to be independent risk factors for coronary heart disease (CHD), although there are mixed findings (Rozanski et al. 1999; Smith et al. 2004). Hostility is a multidimensional construct consisting of components that are cognitive (cynical and mistrustful attitudes toward others), affective (anger), and behavioral (anger expressed outwardly, suppressed, or repressed), although the term hostility refers primarily to cynical attitudes (Greenglass and Julkunen 1989). Some components of hostility might be more harmful than others to the risk of atherosclerosis. Recently studies have shown an association between incident CHD events and the experience and expression of anger (Chang et al. 2002; Kawachi et al. 1996; Williams et al. 2000), which can be considered as representing the affective and behavioral components of hostility.

Carotid intima-media thickness (IMT) measured by ultrasound is a sensitive marker of subclinical atherosclerosis which is a preclinical state of CHD. IMT is related to vascular risk factors, the extent of coronary atherosclerosis, and the occurrence of coronary events (Bots et al. 1997; Burke et al. 1995; Heiss et al. 1991). The advantage of the ultrasound measurement of IMT is that it can be safely applied to asymptomatic people, enabling studies of

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atherosclerosis among healthy young people, who do not yet have clinically manifest disease. Exposure to risk factors in youth is associated with increased IMT in adulthood (Raitakari et al. 2003).

Anger and hostile attitudes have been shown to be associated with IMT cross-sectionally (Bleil et al. 2004; Everson-Rose et al. 2006; Knox et al. 2000; Matsumoto et al. 1993). In prospective studies both anger (Matthews et al. 1998; Räikkönen et al. 2004) and cynical hostility (Julkunen et al. 1994; Pollitt et al. 2005) have been shown to predict higher IMT or progression of IMT. However, in one study hostile attitudes and suppression of anger did not predict progression of IMT (Räikkönen et al. 2004). In summary, the research on the relationship between anger or cynical hostility and IMT has produced mixed findings and has mainly concentrated on either clinical samples or middle-aged populations.

In addition to anger and cynicism, low socioeconomic status (SES), both in childhood and in adulthood, might be considered a risk factor for atherosclerosis (Ranjit et al. 2006; Rosvall et al. 2002). Low SES has also been shown to be associated with high hostility (Gallo and Matthews 2003). It has been suggested that SES might moderate the effects of psychosocial factors as predictors of atherosclerosis. For instance, a study by Lynch et al. (1998) showed that the effect of cardiovascular reactivity on atherosclerotic progression depended on SES, i.e. a stronger association was observed in individuals with low SES. Although SES might moderate the effect of anger and cynicism on atherosclerosis, only a few studies have explicitly examined the effect of participants' SES on that relationship. According to Mendes de Leon (1992) impatience/irritability and anger-out are associated with CHD in middle-aged low SES male patients. On the other hand, Eng et al. (2003) found that anger-out may be a protective factor for stroke and nonfatal myocardial infarction (MI) among male health professionals and they concluded that anger expression may have differential effects depending on the SES of study populations. In line with these studies, Mittleman et al. (1997) found that the risk of episodes of anger triggering the onset of nonfatal MI declined as the SES of patients increased, meaning that SES moderated the role of anger as a trigger of MI and that anger produced the greatest risk for low-SES individuals. Therefore, it is possible that anger and cynicism may produce differential atherosclerosis risk within subgroups of SES, although Everson-Rose et al. (2006) did not observe an interaction between cynical hostility and education level in predicting IMT among middle-aged women. According to our knowledge, there is no previous study on the interaction between anger or cynicism and SES on IMT in a young, population-based sample where SES is measured at

different developmental stages. SES over the life-course may have important effects and it is not known whether SES at different developmental transitions affects the relationship between cynicism or anger and subclinical atherosclerosis.

The aim of the current study was to examine the relationships of cynicism and anger with carotid IMT and to determine whether these relationships were moderated by childhood and adulthood socioeconomic backgrounds. The sample used was nationally representative of Finnish young adults. We also tested whether these associations were independent of physiological risk factors, health-related behaviors, and lack of social support, all of which have been hypothesized to mediate the association between hostility and atherosclerosis (Smith et al. 2004).

Methods

Participants

Participants were 1,540 healthy men and women aged 24–39 in 2001 participating in the ongoing population-based research project called Cardiovascular Risk in Young Finns. In 1980, which was the baseline of the study, 4,320 participants in age cohorts of 3, 6, 9, 12, 15, and 18 were invited to take part and 3,596 participated. To ensure a nationally representative sample, Finland was divided into five areas according to the location of the university cities with a medical school. In each area, urban and rural boys and girls were randomly selected from the Social Insurance Institution's population register, which covers the whole population of Finland (Åkerblom et al. 1991). The study was approved by local ethics committees and all participants gave written informed consent.

Two thousand two hundred and sixty four participants had valid IMT measures at the 21-year follow-up in 2001, of which 1,729 had valid psychological variables, 2,216 had SES data, 2,047 had data on health-related behaviors, 2,208 had complete data on physiological risk factors, and 1,732 had data on perceived social support. When these data were combined, the final sample consisted of those for which there was complete information on all study variables, a total of 1,540 participants (647 men and 893 women), which constitutes 68% of the total participants in 2001. It has been previously shown that those who dropped out during the 21-year follow-up period were slightly biased towards male gender, lower socio-economic status, and a more sedentary lifestyle than those who remained in the study, but no differences in physiological risk factor levels were found (Pulkki-Råback et al. 2005; Raitakari et al. 2003).

Measures

Anger and cynicism

Anger (the suggested affective component of hostility) and cynicism (the suggested cognitive component of hostility) were measured in 2001 and were self-reported by the participants.

Anger was measured with a 7-item Irritability Scale of the Buss-Durkee Hostility Inventory (Buss and Durkee 1957). The Irritability Scale has been shown to correlate with the Spielberger State-Trait Anger Scale (Moreno et al. 1993). In addition, this scale has been suggested to be an adequate measure of anger by temperament research (Buss 1991). The items of the anger scale are “I lose my temper easily but get over it quickly”, “I am irritated a great deal more than people are aware of”, “It makes my blood boil to have somebody make fun of me”, “Sometimes people bother me just by being around”, “I often feel like a powder keg ready to explode”, “I sometimes carry a chip on my shoulder”, and “Lately, I have been kind of grouchy”.

Cynicism was measured with a 7-item Cynicism scale derived from the Minnesota Multiphasic Personality Inventory (items 89, 93, 117, 124, 265, 316, and 319) (Comrey 1957, 1958). This scale shares five items with the 9-item cynical distrust factor derived by Greenglass and Julkunen (1989) and six items with the 13-item cynicism scale derived by Barefoot et al. (1989) from the Cook-Medley Hostility scale. The items of the cynicism scale are “It takes a lot of argument to convince most people of the truth”, “I think most people would lie to get ahead”, “Most people are honest chiefly through fear of being caught”, “It is safer to trust nobody”, “Most people will use somewhat unfair means to gain profit or an advantage rather than to lose it”, “I think nearly anyone would tell a lie to keep out of trouble”, and “Most people inwardly dislike putting themselves out to help other people”. The cynicism scale has previously been associated with low social support (Keltikangas-Järvinen and Ravaja 2002) and cardiovascular risk factors, such as smoking, alcohol use and triglycerides (Keltikangas-Järvinen and Ravaja 2002; Pulkki et al. 2003). Each item was rated on a five-point scale, ranging from 1 (totally disagree) to 5 (totally agree), and the mean of each scale was calculated for only those who had responded to at least 50% of the items on the scale. Cronbach's α s for anger and cynicism scales were .76 and .79, respectively.

These scales, even though not standardized measures of hostility, approximate the affective and cognitive components of hostility. In addition, these very scales (Keltikangas-Järvinen and Ravaja 2002; Pulkki et al. 2003) as well as similar constructs (Koskenvuo et al. 1988) have

been used previously in a Finnish population-based studies of cardiovascular risk.

Socioeconomic status

Participant's childhood SES was measured by parents' years of education in 1980, using data of the more educated parent. Childhood SES was classified as low-SES (<9 years of education) and medium/high SES (≥ 9 years of education). Likewise, adulthood SES was measured in 2001 as a dichotomous variable. However, because the general educational level has risen during the past two decades in Finland (Statistics Finland 2005), the cut-off point for low-SES was less than 13 years of education. In Finland, 9 years of education corresponds to having completed comprehensive school, and 13 years to having completed secondary education. Education was used as the marker of SES because the participants were fairly young at the measurement of their own adulthood SES (24–39 years), and therefore the youngest participants had not yet established their own occupational position in the labor market. In Finland, educational level correlates strongly with occupational position, and in a sub-analysis of the oldest participants of the present study ($n = 1173$, mean age = 35.8) education had a correlation of .61 ($P < .001$) with occupational status.

Carotid intima-media thickness

To assess carotid IMT, ultrasound studies were performed using Sequoia 512 ultrasound mainframes (Acuson, CA) with 13.0-MHz linear array transducers. The studies were performed between September 2001 and January 2002. The left common carotid artery was scanned by ultrasound technicians following a standardized protocol (Raitakari et al. 2003). A magnified image was recorded from an angle showing the greatest distance between lumen-intima interface and the media-adventitia interface. A moving scan with a duration of 5 s, which included the beginning of the carotid bifurcation and the common carotid artery, was recorded and stored in digital format on optical discs for subsequent off-line analysis. The digitally stored scans were manually analyzed by a single reader blinded to participants' details. The analyses were performed using ultrasonic calipers. From the 5-s clip image, the best quality end-diastolic frame was selected (incident with the R-wave on a continuously recorded electrocardiogram). From this image, at least four measurements of common carotid far wall were taken approximately 10 mm proximal to the bifurcation to derive a mean carotid IMT. We have reported a 6.4% between-visit coefficient and a 5.2% between-observer coefficient of variation in the IMT measurements (Raitakari et al. 2003). Common carotid

artery and carotid bulb areas were scanned for the presence of plaques. Plaques were defined as a distinct area of the vessel wall protruding into the lumen >50% of the adjacent intima-media layer. Wall regularity or echogenicity were not measured. None of subjects had plaques in the common carotid.

Atherosclerosis risk factors

The atherosclerosis risk factors below were measured in 2001. Health-related behaviors included smoking status (daily smoker versus non-smoker), heavy alcohol consumption, and physical activity. Heavy alcohol consumption was measured as how often alcohol was used with at least six portions at a time (one portion equals to 12 g) ranging from 1 (once a year or never) to 6 (at least twice a week). Physical activity was measured with an index of intensity, frequency, and duration of physical activity (Telama et al. 2005). High scores on the Physical Activity Index (Cronbach's $\alpha = .76$) indicate high physical activity.

Physiological risk factors included body-mass index (BMI), cholesterol levels, and blood pressure. BMI was calculated as weight (kg)/height² (m²). Measurements of lipid levels were taken in duplicate in the same laboratory. Standardized enzymatic methods were used for measuring the level of serum high-density lipoprotein (HDL) cholesterol, and low-density lipoprotein (LDL) cholesterol concentration was calculated according to the Friedewald formula (Friedewald et al. 1972). Blood pressure was measured with random zero sphygmomanometer in sitting position after 5 min rest. Readings to the nearest even number of millimeters of mercury were performed at least three times on each subject, and the average of these measurements was used in statistical analysis (Juonala et al. 2004).

Social support was assessed with the Perceived Social Support Scale-Revised consisting of 12 items (e.g., "My family always supports me when I need help") (Blumenthal et al. 1987). The participants rated the items on a 5-point scale ranging from totally disagree (1) to totally agree (5). The items were inversely coded, and the mean of 12 items was calculated to present social support received from family and friends, with high scores indicating low support. The Cronbach's α for the social support scale was .94.

Statistical analyses

First we tested whether genders should be analyzed together or separately. This was done with triplicate interactions between cynicism or anger, gender and SES predicting IMT. The bivariate associations between all the

study variables were examined with partial correlation analysis. The interaction effects between SES and cynicism or anger on IMT were tested with linear regression analysis with centered variables. When a significant interaction effect was found the population was split in two according to the moderator variable (SES) as recommended by Kraemer et al. (2001). Thus, subsequent analyses of the associations between cynicism or anger and IMT were performed separately within low and medium/high SES groups with multiple linear regression analysis adjusted for different blocks of atherosclerosis risk factors as follows: Model 1 included age and gender, Model 2 included age, gender, and the other SES variable as a covariate, Model 3 included age, gender, and health-related behaviors, Model 4 included age, gender, and physiological risk factors, Model 5 included age, gender, and social support, and Model 6 included all the covariates. Age and gender were used as covariates in all the analyses. Because the distribution was positively skewed, social support was log transformed before analyses. Statistical analyses were performed using SPSS 15.0.

Results

Gender differences and descriptives of the sample

The triplicate interactions concerning gender differences were found to be non-significant (all P 's > .10). Thus, genders were analyzed together in subsequent analyses. Table 1 shows the descriptives of the sample.

Bivariate associations between anger, cynicism, risk factors, and IMT

Age- and gender-adjusted correlation coefficients are shown in Table 2. High cynicism and anger were associated with low adulthood SES, adverse health-related behaviors, high BMI and low social support (r s ranged between $-.071$ and $-.271$, P s < .05). In addition high cynicism correlated with low childhood SES ($r = .106$, $P < .05$). Low childhood SES was associated with smoking, low physical activity and low social support (r s ranged between $-.051$ and $-.061$, P s < .05) and low adulthood SES with adverse health-related behaviors, higher BMI and less social support (r s ranged between $-.061$ and $-.211$, P s < .05). High IMT correlated with high LDL-cholesterol, BMI, systolic and diastolic blood pressure (r s ranged between .05 and .17, P s < .05). Cynicism, anger, and SES were not associated with IMT. The mediating role of anger in the association between childhood SES and IMT was also tested. The childhood SES–IMT association without the mediator was $\beta = 0.03$,

Table 1 Characteristics of the study group $N = 1,540$

Variable	$N = 1,540$		Mean (<i>SD</i>)
	<i>n</i>	%	
Age in 2001			31.56 (5.05)
Gender			
Men	647	42.0	
Women	893	58.0	
Cynicism ^a			2.70 (0.69)
Anger ^a			2.51 (0.71)
Childhood SES in 1980 ^b			
Low	507	32.9	
Medium and high	1033	67.1	
Adulthood SES in 2001 ^c			
Low	412	26.8	
Medium and high	1128	73.2	
Smoking			
Non-smoker	1225	79.5	
Smoker	315	20.5	
Heavy alcohol consumption ^d			2.53 (1.37)
Physical activity index ^e			9.66 (2.29)
HDL-cholesterol (mmol/l)			1.31 (0.32)
LDL-cholesterol (mmol/l)			3.27 (0.85)
Body mass index (kg/m ²)			24.79 (4.29)
Systolic blood pressure (mmHg)			116.33 (13.00)
Diastolic blood pressure (mmHg)			70.50 (10.59)
Social support ^a			1.79 (0.78)
IMT (mm)			0.58 (0.09)

^a Cynicism, anger, and social support have possible values ranging from 1 to 5

^b Low versus medium and high childhood SES = under 9 years versus 9 years or more of education

^c Low versus medium and high adulthood SES = under 13 years versus 13 years or more of education

^d Heavy alcohol consumption has values ranging from 1 (= once a year or never) to 6 (=at least twice a week)

^e Physical activity index has values ranging from 5 to 16

$P = .21$, and with anger as the mediator $\beta = 0.03$, $P = .18$, thereby providing no evidence for mediation.

Interactions between cynicism or anger and SES in predicting IMT

Table 3 shows cynicism \times SES and anger \times SES interactions in predicting IMT. Adulthood SES did not have any interactive effects with hostility in predicting IMT. The interactions were additionally re-conducted using a three-grade adulthood SES measure (1 = comprehensive school, 2 = secondary education, 3 = academic education), which did not show any interactive effects in predicting IMT (data

not shown). Also, childhood SES did not moderate the relationship between cynicism and IMT. However, the childhood SES \times anger interaction was significant ($t = -3.29$, $P = .001$).

As shown in Fig. 1 and Table 4, anger was positively associated with IMT in those with low childhood SES, whereas anger was not associated with IMT in those having medium/high childhood SES. Results of separate analyses for the low and medium/high childhood SES groups are shown in Table 4. Among participants with low childhood SES, higher anger was associated with higher IMT ($B = 0.02$, $t = 3.73$, $P < .001$). This means that a one-point increase in anger corresponds to a 0.02 mm increase in IMT among low-childhood-SES individuals. This association remained significant after separate adjustment for the participants' adulthood SES, health-related behaviors, physiological risk factors, and social support. Anger also remained significantly associated with IMT after simultaneous adjustment for these atherosclerosis risk factors among low-childhood-SES participants ($B = 0.02$, $t = 3.39$, $P = .001$). If participants had higher childhood SES, anger was not associated with IMT ($B = -0.00$, $t = -0.98$, $P = .33$). In an additional analysis where only anger was used as an explanatory factor of IMT, anger explained 1.6% of the variance in IMT among low-childhood-SES participants.

Discussion

The present findings emphasize the modifying role of childhood SES in the association between anger and pre-clinical atherosclerosis. In the present sample of young men and women, anger, cynicism or SES had no independent associations with atherosclerosis in all participants, but within the subgroup of low-childhood-SES individuals, anger was associated with higher IMT. Among those who had grown up in a low-SES environment, carotid artery IMT was the highest in individuals who reported higher experience of anger. Experiences of anger were not related to carotid artery IMT in the higher-childhood-SES group. In low-SES participants, the anger—IMT association was independent of, or only marginally attenuated by, participant's adulthood SES, adverse health-related behaviors, physiological atherosclerosis risk factors, and lack of social support. The present study stresses that the behavioral risk factors for atherosclerosis might differ according to the participants' SES during developmental years in childhood.

The explanation for why anger, but not cynicism, was associated with atherosclerosis among low-childhood-SES individuals is unknown and requires further investigation. However, previous research provides more consistent

Table 2 Age- and gender-adjusted partial correlations between the study variables

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Cynicism													
2. Anger	.39***												
3. Childhood SES	-.06*	-.05											
4. Adulthood SES	-.15***	-.07*	.20***										
5. Smoking	.09***	.07**	-.05*	-.21***									
6. Alcohol consumption	.12***	.08**	.04	-.08**	.27***								
7. PAI	-.07**	-.11***	.05*	.12***	-.17***	.07**							
8. HDL-cholesterol	-.04	-.01	.00	.04	-.08**	.14***	.10***						
9. LDL-cholesterol	-.02	.01	-.02	-.04	.02	-.03	-.08**	.00					
10. BMI	.11***	.11***	-.02	-.06*	-.01	.05*	-.05*	-.24***	.22***				
11. SBP	.03	.01	-.04	.01	-.07**	.05	.05*	-.01	.07**	.32***			
12. DBP	.05	.01	.02	.01	-.09**	.08**	.04	-.05*	.11***	.30***	.67***		
13. Social support	.25***	.27***	-.06*	-.09***	-.02	-.01	-.11***	-.03	.01	.05*	.03	.05	
14. IMT	-.01	.04	.03	-.01	.01	.04	.03	-.02	.05*	.17***	.14***	.14***	-.00

PAI = Physical Activity Index, HDL = high-density lipoprotein, LDL = low-density lipoprotein, BMI = body mass index, SBP = systolic blood pressure, DBP = diastolic blood pressure, IMT = intima-media thickness

* $P < .05$, ** $P < .01$, *** $P < .001$

evidence for the importance of anger in CHD than for hostility (Strike and Steptoe 2004). For example, anger may act as a trigger of MI (Mittleman et al. 1997). Anger might also be more strongly related to physiological reactivity than cynicism. In laboratory situations anger-provoking tasks are likely to elicit a positive association between hostility and cardiovascular reactivity (Suarez and Williams 1990), which is associated with IMT (Treiber et al. 2003). Thus, physiological reactivity may be a pathway whereby anger is associated with IMT.

Interestingly, childhood SES, but not adulthood SES, modified the association between anger and subclinical atherosclerosis. This was true, even though childhood and adulthood SES measures correlated with each other. This supports the idea that early life environment may have long-lasting effects, although these effects may be mediated by later life factors (Power et al. 1998). In the present sample, it has been found that a stressful home environment in childhood had more severe effects on cardiovascular risk factors (insulin resistance syndrome) in individuals coming from low-SES rather than high-SES families (Pulkki et al. 2003). The present findings thus extend our previous results and suggest that individuals with low childhood-SES may be more vulnerable to psychosocial stressors than their higher-SES counterparts. Low-SES individuals have less access to financial, human, and social capital, a fact which affects children's development and later health (Bradley and Corwyn 2002; Repetti et al. 2002). Gallo and Matthews (2003) have suggested in their reserve capacity model that low-SES

individuals have fewer resources for managing stress and, as such, are more vulnerable to negative emotion and cognition. Thus, according to this model, anger has more detrimental effects on cardiovascular health in low-SES individuals because they are less equipped to deal with their negative emotionality.

It is also possible that childhood SES, instead of adult SES, was an important moderator in these relationships due to the young age of the sample and therefore relative recency of the childhood environment. For these young adults, their childhood SES probably accounts for most of their lifetime SES exposure. It is possible that the same pattern of findings would not be found in an older sample, in which adult SES factors may be more important determinants of health. The Cardiovascular Risk in Young Finns study will enable examination of this issue in future follow-up examinations, as participants get older.

The present results suggest that other atherosclerosis risk factors, such as health-related behaviors and lack of social support, contributed only minimally to the association between anger and IMT among low-childhood-SES individuals. Therefore, the mechanism linking anger to atherosclerosis remains unclear. Exaggerated cardiovascular and neuroendocrine reactivity and slower recovery have been suggested as pathways through which emotional factors may associate with cardiovascular disease (Björntorp 1991; McEwen 2000; Smith et al. 2004). These same physiological disturbances have been associated with low SES (Gump et al. 1999; McEwen 2000). Further studies should examine, in population-based samples, whether

Table 3 Results of linear regression analyses regressing carotid IMT on anger or cynicism, SES, and their interaction effects ($N = 1,540$)

	β^a	B^b	SE	t	P	R^2
<i>Childhood SES</i>						
Age	0.34	0.01	0.00	13.69	<.001	
Gender	0.08	0.01	0.00	3.34	.001	
Childhood SES	0.03	0.01	0.00	1.28	.20	
Cynicism	0.04	0.00	0.01	0.95	.34	
Childhood SES \times cynicism	-0.06	-0.01	0.01	-1.47	.14	.12
<i>Adulthood SES</i>						
Age	0.34	0.01	0.00	13.73	<.001	
Gender	0.09	0.02	0.00	3.60	<.001	
Childhood SES	0.04	0.01	0.00	1.44	.15	
Anger	0.14	0.02	0.01	3.58	<.001	
Childhood SES \times anger	-0.13	-0.02	0.01	-3.29	.001	.13
<i>Adulthood SES</i>						
Age	0.33	0.01	0.00	13.80	<.001	
Gender	0.08	0.01	0.00	3.29	.001	
Adulthood SES	-0.02	-0.00	0.01	-0.73	.47	
Cynicism	-0.06	-0.01	0.01	-1.32	.19	
Adulthood SES \times cynicism	0.06	0.01	0.01	1.21	.23	.12
<i>Adulthood SES</i>						
Age	0.33	0.01	0.00	13.78	<.001	
Gender	0.09	0.02	0.00	3.50	<.001	
Adulthood SES	-0.01	-0.00	0.00	-0.40	.69	
Anger	-0.01	-0.00	0.01	-0.17	.86	
Adulthood SES \times Anger	0.05	0.01	0.01	1.21	.23	.12

^a β = standardized regression coefficient

^b B = unstandardized regression coefficient

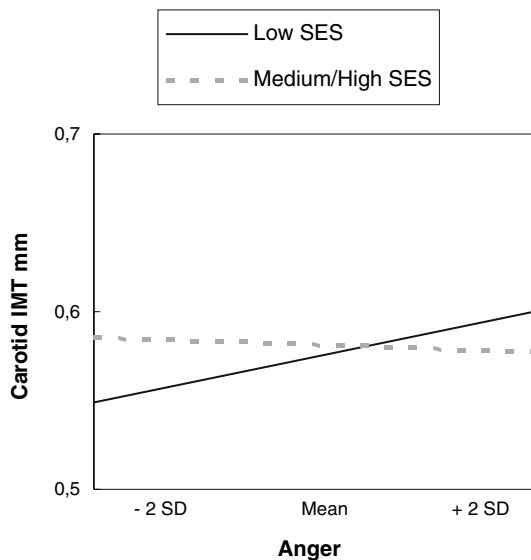


Fig. 1 Age- and gender-adjusted regression lines of anger (range from $-2 SD$ to $+2 SD$) predicting carotid artery intima-media thickness (IMT) in participants with low and medium/high childhood socioeconomic status (SES)

low-SES individuals with high levels of anger have different cardiovascular and neuroendocrine stress-responses compared to their higher-SES counterparts.

Among low-SES participants, anger explained only 1.6% of the variance in IMT. A one-point increase in mean anger, as measured on a five-point scale, corresponded to a 0.02 mm increase in IMT among low-childhood-SES individuals. This may seem rather small. However, a 0.16 mm difference in IMT has been shown to increase the risk of acute myocardial infarction by approximately 50% in subjects aged 55 years or more in the Rotterdam Study (Bots et al. 1997). Also, in the Atherosclerosis Risk in Communities Study, a 0.19 mm difference in mean IMT was shown to increase the risk of cardiovascular events by 69% and 36% among 45–64 years old women and men, respectively (Chambless et al. 1997). Individual differences in IMT are rather small among young and healthy people, and, therefore, in this healthy young sample even a small increase of IMT may be considered significant in terms of future risk.

Table 4 Standardized (β) and unstandardized (B) regression coefficients of anger predicting IMT separately adjusted for different blocks of atherosclerosis risk factors for participants with low and medium/high childhood SES

	$N = 1,540$											
	Low childhood SES $N = 507$						Medium/high childhood SES $N = 1,033$					
	$R^{2\dagger}$	β	B	SE	t	P -value	$R^{2\dagger}$	β	B	SE	t	P -value
Model 1 ^a	.13	0.16	0.02	0.01	3.73	<.001	.13	-0.03	-0.00	0.00	-0.98	.33
Model 2 ^b	.13	0.16	0.02	0.01	3.77	<.001	.13	-0.03	-0.00	0.00	-1.08	.28
Model 3 ^c	.13	0.16	0.02	0.01	3.76	<.001	.13	-0.03	-0.00	0.00	-0.98	.33
Model 4 ^d	.17	0.14	0.02	0.01	3.37	.001	.16	-0.04	-0.01	0.00	-1.49	.14
Model 5 ^e	.13	0.16	0.02	0.01	3.68	<.001	.13	-0.03	-0.00	0.00	-0.81	.42
Model 6 ^f	.18	0.15	0.02	0.01	3.39	.001	.16	-0.04	-0.01	0.00	-1.34	.18

[†] R^2 is for the whole model

^a Model 1: Anger adjusted for age and gender

^b Model 2: Anger adjusted for age, gender, and adulthood SES

^c Model 3: Anger adjusted for age, gender, and health related behaviors (smoking, alcohol consumption, and physical activity index)

^d Model 4: Anger adjusted for age, gender, and physiological risk factors (LDL-cholesterol, HDL-cholesterol, systolic blood pressure, diastolic blood pressure, and BMI)

^e Model 5: Anger adjusted for age, gender, and social support

^f Model 6: Anger adjusted for all covariates (age, gender, adulthood SES, health related behaviors, physiological risk factors, and social support)

The strengths of the study were that the participants were young and the sample was nationally representative of young Finnish adults and large enough to compare different gender and SES groups. We also were able to control for several factors that are plausible mechanisms relating anger to atherosclerosis. The current study demonstrated that in the presence of both low SES and high anger, effects on subclinical atherosclerosis become visible at a relatively young age, long before clinical manifestations of cardiovascular disease occur.

The cross-sectional design limits the conclusions that can be drawn regarding directionality. Specifically, although the participants in the present sample were apparently healthy, we cannot conclude whether it is anger that causes thickening of intima-media or vice versa. Another limitation was that only education was used as an indicator of SES. Education was used because it is usually fixed after young adulthood and is unlikely to be influenced by poor health later in life. Education also makes it possible to classify non-working people (e.g., home mothers) into socioeconomic groups.

Conclusions

The finding that it is childhood SES, but not adulthood SES, which modifies the association between anger and preclinical atherosclerosis, lends support to the idea that the health effects of anger are dependent on context and have their origins early in life. Our findings suggest that

higher anger is related to higher IMT only among individuals with low-SES backgrounds, but the mechanisms underlying this association require further investigation.

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