Supporting Information

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SI Text

Role of Positive Interactions. To further ensure that negative and competitive social interactions are related to inflammatory activity over and above the effects of positive social interactions, we included positive interactions as a covariate in the main regression analyses. When controlling for number of positive social interactions, the association between negative social interactions and baseline type II soluble receptor for TNF-α(sTNFαRII) remained significant ($\beta = 0.242$, $P = 0.013$). Similarly, competitive social interactions continued to predict baseline levels of IL-6 ($\beta = 0.182$, $P = 0.051$) and sTNFαRII ($\beta = 0.177$, $P = 0.057$). These findings indicate that the effect of negative and competitive social interactions on inflammatory activity is over and above that of positive social interactions.

Including positive social interactions as a covariate weakened the association between negative social interactions and inflammatory reactivity. For IL-6 25-min poststressor, the relationship became marginally significant ($\beta = 0.120$, $P = 0.076$), and for sTNFαRII 25-min poststressor, the relationship was no longer significant ($\beta = 0.083$, $P = 0.216$). The relationship between positive social interactions and sTNFαRII 25-min poststressor also became nonsignificant when controlling for negative social interactions ($\beta = 0.092$, $P = 0.163$). The predictive values of negative social interactions and of positive social interactions on inflammatory reactivity may be driven, in part, by shared variance between the two variables.

When controlling for positive interactions in our area-under-the-curve analyses, we found that negative social interactions continued to significantly predict total output of sTNFαRII ($\beta = 0.236$, $P = 0.016$). Competitive social interactions also continued to significantly predict total output of IL-6 ($\beta = 0.186$, $P = 0.045$). However, the relationship between competitive social interactions and total output of sTNFαRII became marginally significant ($\beta = 0.162$, $P = 0.080$).

We also tested for interactions between the different types of social interactions. Although competitive social interactions were not related to stress-induced reactivity, there were marginally significant interaction effects of positive and competitive social interactions on IL-6 25-min poststressor ($\beta = 0.114$, $P = 0.075$) and on sTNFαRII 25-min poststressor ($\beta = 0.120$, $P = 0.051$). There were no other significant interactions.

1. O'Connor MF, et al. (2009) To assess, to control, to exclude: Effects of biobehavioral factors on circulating inflammatory markers. *Brain Behav Immun* 23:887–897.

Ethnicity Analyses. Because of ethnic variation in the sample, we conducted a series of two-step hierarchical regression analyses. Cytokine levels were regressed on ethnicity, social interaction, and relevant covariates in the first step to examine whether the relationships between social interaction and proinflammatory levels were confounded by ethnicity. When ethnicity was entered, competitive social interaction significantly predicted baseline IL-6 ($\beta = 0.181$, $P = 0.049$; from $\beta = 0.175$, $P = 0.054$ without ethnicity in the model). Baseline IL-6 was higher for those who experienced more competitive social interactions independent of ethnicity.

For sTNFαRII, adding ethnicity reduced the significance of competitive social interactions' relationship with baseline sTNFαRII ($\beta = 0.175$, $P = 0.054$, from $\beta = 0.178$, $P = 0.050$ without ethnicity in the model). This reduction may stem from loss of degrees of freedom or may indicate that the variance in sTNFαRII accounted for by competitive social interactions may partially overlap with that accounted for by ethnicity; however, there was no significant effect of ethnicity ($\beta = -0.065$, $P = 0.478$). All other associations remained significant when controlling for ethnicity.

In the second step, an interaction term was entered into the model. For IL-6, the interaction of ethnicity and negative social interactions was marginally significant ($\beta = -0.201$, $P = 0.058$). None of the other interactions were significant. Similarly, none of the interactions were significant for sTNFαRII. Ethnicity did not moderate the relationship between social interactions and inflammatory activity.

Sex Analyses. Evidence suggests that there may be sex differences in levels of C-reactive protein, with women having higher levels (1). Although evidence on sex differences in IL-6 and sTNFαRII is inconclusive (1), and although sex was not correlated with most cytokine assessments, we nevertheless examined whether sex confounded the effect of social interactions on proinflammatory levels and whether sex moderated the relationship. We conducted a series of two-step hierarchical regression analyses in which sex was added to initial models in the first step, and an interaction term was added to the previous step in the second step. Controlling for sex in initial models that did not already include sex as a covariate did not alter results. There was a significant effect of sex on baseline sTNFαRII, such that females tended to exhibit lower baseline levels of sTNFαRII ($\beta = -0.199$, $P = 0.030$). There were no significant interactions between sex and social interactions.

Table S1. Regression analyses predicting IL-6 levels and reactivity from daily social interactions

	Baseline IL-6			25-min poststressor IL-6			80-min poststressor IL-6		
Interaction	β	B	SE	β	B	SE	β	B	SE
Positive social interactions									
Intercept		0.240	0.160		0.078	0.108		0.063	0.097
Baseline				0.744**	0.748	0.062	0.823**	0.871	0.055
Positive interactions	−0.001	0.000	0.000	0.077	0.003	0.003	0.076	0.003	0.002
Negative social interactions									
Intercept		0.147	0.159		−0.026	0.110		0.113	0.097
Baseline				0.736**	0.769	0.063	0.820**	0.868	0.055
Negative interactions	0.066	0.012	0.97	0.132*	0.024	0.011	0.042	0.008	0.010
Competitive social interactions									
Intercept		−0.021	0.164		0.100	0.116		0.071	0.101
Baseline				0.738**	0.771	0.065	0.811**	0.858	0.056
Competitive interactions	0.175***	0.073	0.038	0.039	0.017	0.027	0.067	0.030	0.023

*$P \leq 0.05$; **$P \leq 0.01$; ***marginally significant ($P = 0.054$).

Table S2. Regression analyses predicting sTNFαRII levels and reactivity from daily social interactions

	Baseline sTNFαRII[†]			25-min poststressor sTNFαRII			80-min poststressor sTNFαRII[‡]		
Interaction	β	B	SE	β	B	SE	β	B	SE
Positive social interactions									
Intercept		2.716**	0.142		0.552**	0.179		1.149**	0.183
Baseline				0.761**	0.770	0.060	0.760**	0.684	0.053
Positive interactions	0.039	0.09	0.003	0.128*	0.004	0.002	0.036	0.001	0.002
Negative social interactions									
Intercept		2.527**	0.141		0.740**	0.061		1.142**	0.172
Baseline				0.731**	0.740	0.061	0.747**	0.672	0.053
Negative interactions	0.219*	0.031	0.012	0.124*	0.018	0.009	0.072	0.010	0.008
Competitive social interactions									
Intercept		2.529**	0.157		0.676**	0.170		1.133**	0.175
Baseline				0.742**	0.750	0.062	0.749**	0.674	0.053
Competitive interactions	0.178*	0.057	0.029	0.058	0.020	0.021	0.067	0.021	0.018

*$P \leq 0.05$; **$P \leq 0.01$.
[†]Sex was entered as a covariate.
[‡]Sex and ethnicity were entered as covariates.