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| RK | conceptualization of analyses, conduction of analyses, interpretation of the data, drafting and revision of the manuscript |
| SS | data collection, revision of the manuscript, interpretation of the data |
| TS | data collection, revision of the manuscript, interpretation of the data |
| FS | data collection |
| DY | data collection |
| DG | data collection, provided data infrastructure |
| UD | financially enabled the study |
| TH | financially enabled the study, interpretation of the data, revision of the manuscript |
| AD | financially enabled the study |
| JS | provided data infrastructure |
| OS | provided data infrastructure |
| IN | financially enabled the study, interpretation of the data |
| TK | interpretation of the data, financially enabled the study, revision of the manuscript |
| AJ | conceptualization of analyses, conduction of analyses, interpretation of the data, provided data infrastructure, drafting and revision of the manuscript, financially enabled the study |

RK, AJ conceptualization of analyses & conduction of analyses, drafting manuscript; RK, AJ, SS, TS, TH, IN, TK interpretation of data; SS, TS, FS, DY, DG data collection; RK, AJ, SS, TS, TH, RK revision of the manuscript; RK, AJ, SS, TS, TH, IN, TK interpretation of the data; DG, JS, OS, AJ provided data infrastructure; UD, TH, AD, IN, TK, AJ financially enabled the study

Contribution to the field:

The present study introduces a network model explaining amygdala hyperactivity in healthy subjects at risk for depression, i.e. the limbic-cortical model. This model has previously been evaluated to explain disrupted networking in Major Depression. We transferred it to healthy subjects with increased risk for depression. We operationalized a genetic risk (i.e. genetic liability) and an environmental risk (i.e. childhood maltreatment) in a large cohort of healthy subjects. Subjects with either kind of risk are well known to exhibit hyper activity in the amygdala as response to emotional expressions. The medial prefrontal cortex is supposed to regulate amygdala response, and we hypothesized a disruption of this function. Therefore, we use brain imaging with effective connectivity analyses. We show that childhood maltreatment rather than genetic liability was associated with malfunction of amygdala inhibition by prefrontal cortex, and highlight this possible biomarker for early therapeutic intervention.