CORRIERE DELLA SERA

9 marzo 2016 (modifica il 9 marzo 2016 | 20:52)

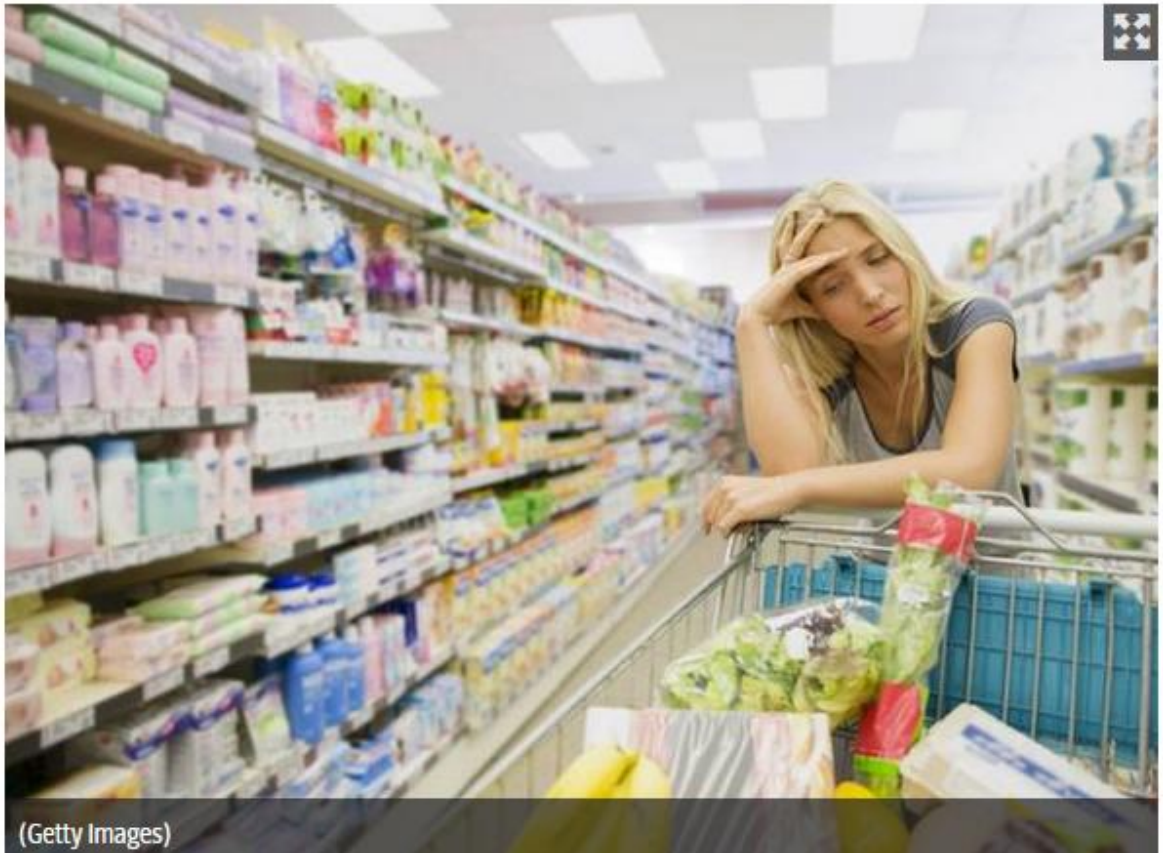
RICERCA USA

Lo stress a lungo termine erode la memoria a breve termine

In risposta alla situazione di disagio si verifica uno stato d'infiammazione cerebrale innescato dal sistema immunitario: lo studio (sui topi da laboratorio) potrebbe aprire una possibile strada all'identificazione di farmaci per controllare il disturbo cronico

di Emanuela Di Pasqua

Chi vive una condizione di stress continuativo nel tempo, tra i tanti effetti collaterali, potrebbe trovarsi a perdere pezzi di memoria, in particolare quella relativa ai ricordi recenti



(Getty Images)

L'ipotesi è che lo stress causi un'infiammazione a livello dell'ippocampo, zona del cervello deputata alla memoria, la quale induce una proliferazione di macrofagi, che a loro volta bloccano in modo rilevante lo sviluppo di nuovi neuroni».

frontiers in NEUROSCIENCE

REVIEW ARTICLE
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Monocyte trafficking to the brain with stress and inflammation: a novel axis of immune-to-brain communication that influences mood and behavior

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HIGHLIGHTS

- Psychological stress activates neuroendocrine pathways that alter immune responses.
- Stress-induced alterations in microglia phenotype and monocyte priming leads to aberrant peripheral and central inflammation.
- Elevated pro-inflammatory cytokine levels caused by microglia activation and recruitment of monocytes to the brain contribute to development and persistent anxiety-like behavior.
- Mechanisms that mediate interactions between microglia, endothelial cells, and macrophages and how these contribute to changes in behavior are discussed.
- Sensitization of microglia and re-distribution of primed monocytes are implicated in re-establishment of anxiety-like behavior.

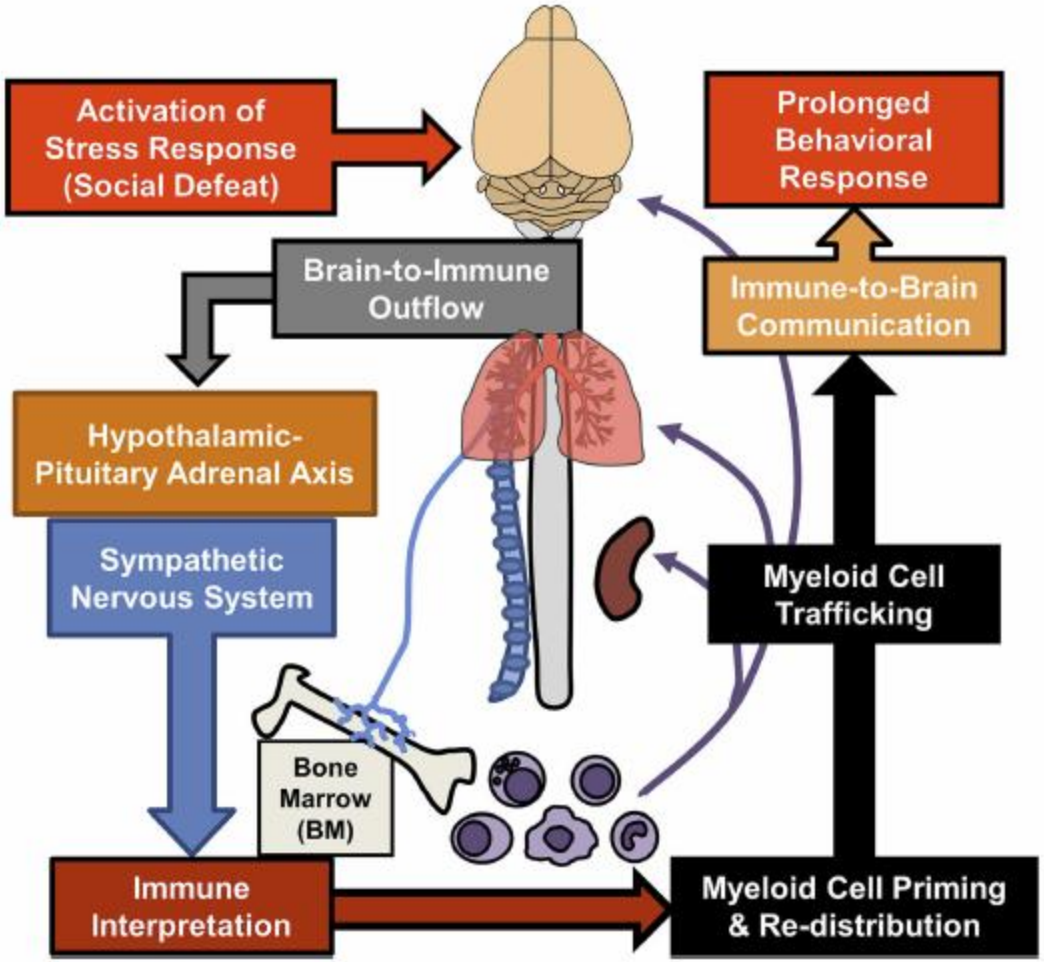

FIGURE 1 | Overview of bi-directional neuroimmune communication in response to stress. Stress responses initiate brain-to-immune outflow that influences myeloid cell function. Re-distribution of primed myeloid cells augments immune-to-brain communication contributing to prolonged anxiety- and depressive-like behaviors.

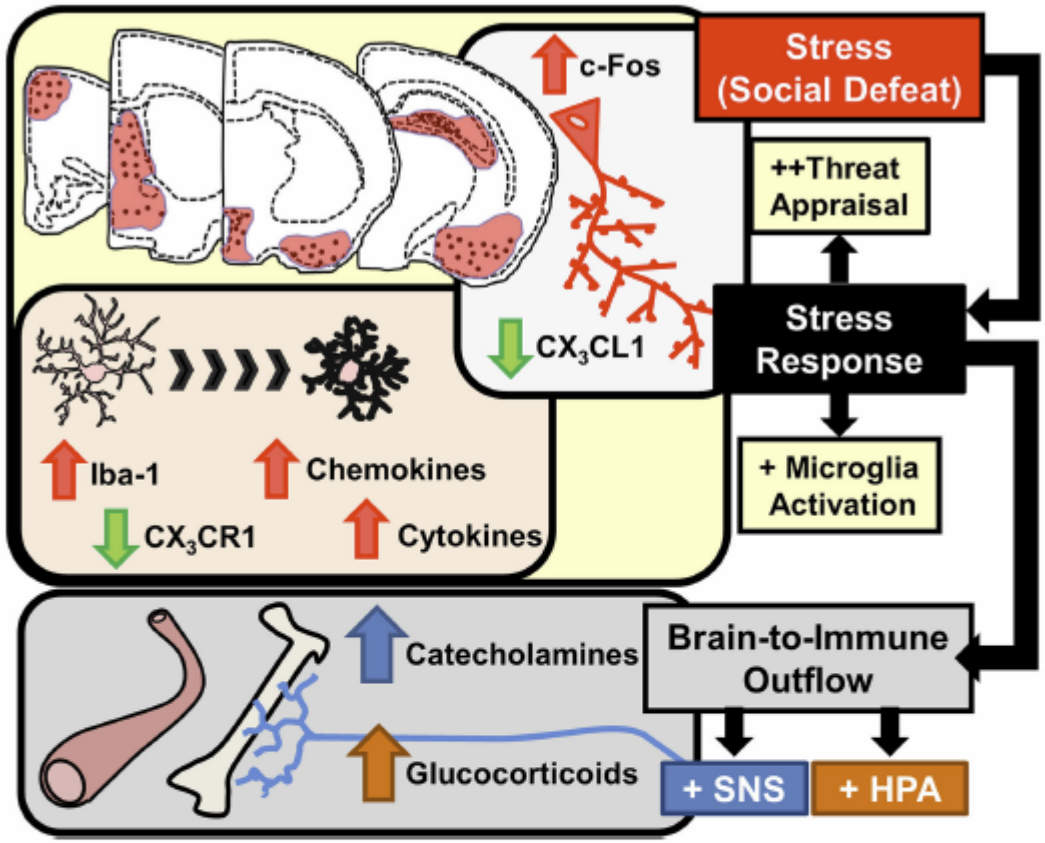

FIGURE 2 | Stress response in the brain leads to microglia activation and brain-to-immune outflow alters immune activity in peripheral immune organs. Interpretation of stressful stimuli activates threat appraisal neurocircuitry including specific cortico-limbic brain regions that show increased c-Fos immunolabeling. In corresponding brain regions microglia demonstrate morphological changes, increased pro-inflammatory cytokine and chemokine expression and deficits in immunoregulatory markers such as CX_3CR1 are evident. Stress responses also cause brain-to-immune outflow with HPA and SNS activation that profoundly alter peripheral immune activity.

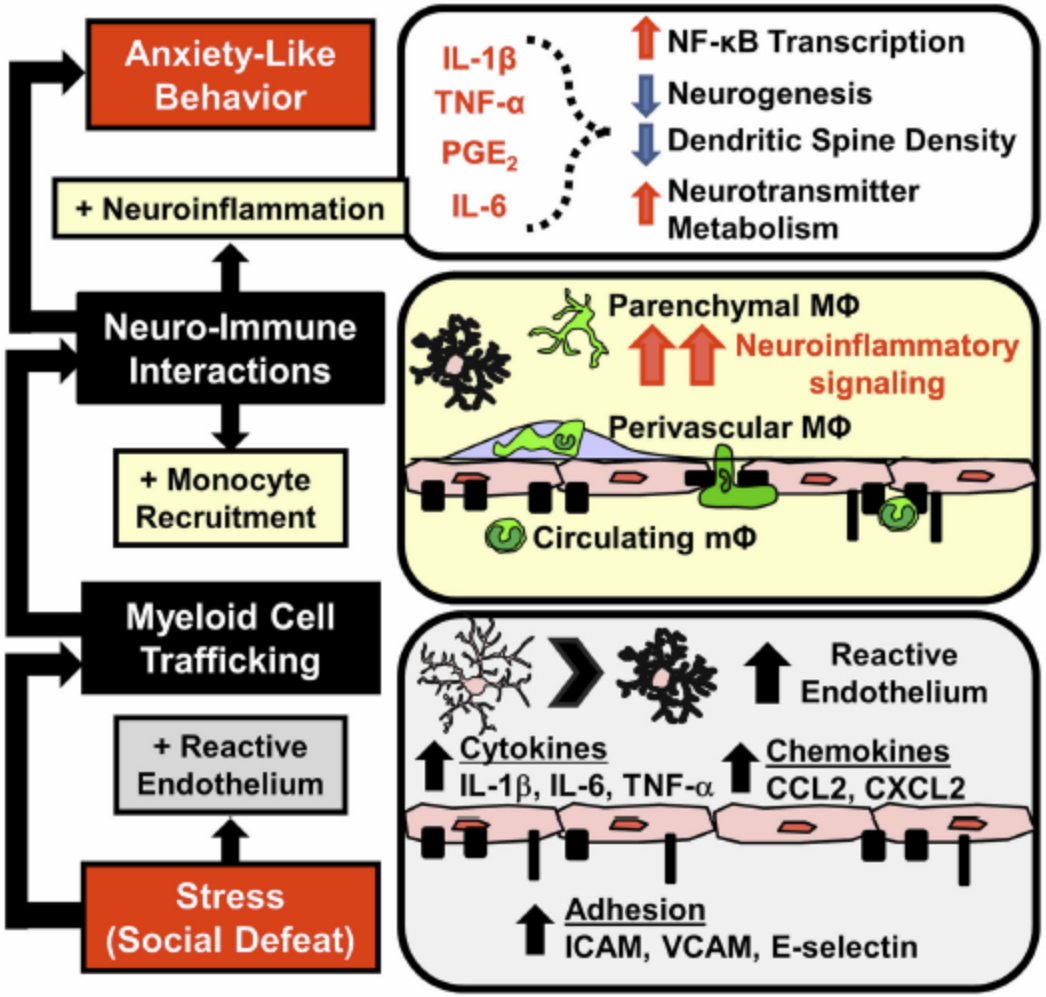

FIGURE 4 | Stress-induced microglia activation and macrophage recruitment to the brain contributes to development of prolonged anxiety-like behavior. Repeated social defeat leads to microglia activation with increased pro-inflammatory cytokine and chemokine production that contributes to the development of reactive endothelium. Vascular endothelial cells increase cell adhesion molecule (CAM) expression that facilitates the adherence and extravasation of peripehrally derived monocytes (mΦ) that differentiate into perivascular and parenchymal

Hemodynamic and Autonomic Adjustments to Real Life Stress Conditions in Humans

Daniela Lucini, Guido Norbiato, Mario Clerici, Massimo Pagani

s represents a risk factor for hypertension, but mechanisms are not known in detail. In this hypothesis that real-life stress conditions produce changes in autonomic cardiac and vascular in magnitude. University students, a well-established model of mild real-life stress, were university examination, and a second time 3 months afterward, during holiday. Autonomic vas assessed by a noninvasive approach, based on autoregressive analysis of RR interval ic arterial pressure (SAP) V. The overall level of stress in the two sessions was gauged from ol (5.6±0.5 versus 2.4±0.2 ng/mL, $P<0.05$) and altered cytokine profile ($P<0.05$). During val was reduced and arterial pressure increased significantly; simultaneously, the normalized of RRV (a marker of sympathetic modulation of the sinoatrial node) was increased and the reflex gain) reduced. Concomitantly, the autonomic response to the sympathetic excitation altered: cardiac response was impaired and vascular responsiveness increased. Markers of sinoatrial node correlated significantly with cortisol levels, both at rest and also considering suggesting a gradual range of effects. The data support the concept that mild real-life stress nd impairs cardiovascular homeostasis. These changes, assessable with spectral analysis of might contribute, in susceptible individuals, to the link between psychological stress and k of hypertension. (*Hypertension*. 2002;39:184-188.)