The contagion of social defeat stress: Insights from rodent studies

Luca Carnevali\textsuperscript{a}, Nicola Montano\textsuperscript{b,c}, Eleonora Tobaldini\textsuperscript{b,c}, Julian F Thayer\textsuperscript{d}, Andrea Sgoifo\textsuperscript{a}

\textsuperscript{a}Stress Physiology Lab, Department of Chemistry, Life Sciences and Environmental Sustainability, University of Parma, Parma, Italy
\textsuperscript{b}Department of Internal Medicine, Fondazione IRCCS Ca’ Granda, Ospedale Maggiore Policlinico, Milan, Italy
\textsuperscript{c}Department of Clinical Sciences and Community Health, University of Milan, Milan, Italy
\textsuperscript{d}Department of Psychological Science, The University of California, Irvine, Irvine, CA, USA.

**Corresponding author:** Luca Carnevali, Stress Physiology Lab, Department of Chemistry, Life Sciences and Environmental Sustainability, University of Parma, Parco Area delle Scienze 11/A, 43124, Parma, Italy. Email address: luca.carnevali@unipr.it
Abstract
Stressful experiences can be transmitted among individuals through social interactions. Like humans, rodents are social creatures whose behavior and physiology can be influenced by the emotional state of fellow rodents. This paper will review rodent studies which have explored two conditions of potential social stress contagion using the social defeat paradigm. In the vicarious social defeat model, mice and rats that witness a conspecific being socially defeated exhibit physiological stress responses and develop a host of depressive- and anxiety-like behavioral deficits. Likewise, social interaction with a stressed partner in the aftermath of social defeat stress results in physiological stress responses and social avoidance behavior. After summarizing the existing literature on this newly emerging area of social defeat stress contagion in rodents, we will discuss the potential utility of these rodent models for investigating the neurobiological processes and sensory channels of information that allow for the spread of psychophysiological effects of stress across individuals.
1. Introduction

Emotional contagion, a term coined by psychology professor Elaine Hatfield (Hatfield et al., 1993), has been construed as a simple or automatic process in which one simply “catches” aspects of another person’s emotional state, producing similar affective and physiological responses that result directly from the observation (Hatfield et al., 1993; Hoffman, 2000). Findings of brain regions with mirror properties that are active when individuals perform an action as well as when they observe others perform the same or similar actions have fueled speculations about neural mechanisms underlying the social sharing of emotions (Ferrari and Rizzolatti, 2014; Iacoboni et al., 1999). Specifically, within the social domain, mirroring would occur when the same neurons are activated by emotions experienced directly and by observing/interacting with others who are experiencing emotions (Carr et al., 2003; Rizzolatti et al., 2001; Wicker et al., 2001). Emotional contagion, also known as the “resonance” of emotions among individuals, may form the basis - together with more complex processes - for a full capacity for empathy (Preston and de Waal, 2002). Such capacity has been long considered uniquely human. However, studies in nonhuman primates (e.g., Palagi et al., 2014), pigs (e.g., Reimert et al., 2013), dogs (e.g., Huber et al., 2017) and rodents (e.g., Atsak et al., 2011) have shown that emotional contagion exists across species, does not require advanced cognitive capabilities, and is crucial to successfully navigate the social environment (Decety and Lamm, 2009; Panksepp and Panksepp, 2013). Recent years have witnessed growing interest in the study of “empathic stress” or “stress contagion” or “stress resonance”, as it has been variably called in human studies (Engert et al., 2019; White and Buchanan, 2016). Indeed, stress often occurs in social settings and can be transmitted among individuals as a consequence of social interactions in dyads and groups. Such “contagious stress” may induce emotional and physiological responses also in those who are not directly exposed to the stressor and may ultimately represent an additional pathway to the deleterious mental and physical consequences associated with stress exposure, beyond the daily stressors experienced firsthand. Therefore, in this paper, the term “contagious
stress” or “stress contagion” refers to the presence of behavioral (e.g., anxiety-like symptoms) and/or physiological (e.g., hypothalamic-pituitary-adrenal (HPA) axis activation) sequelae of stress exposure also in those individuals who are not directly exposed to the stressor. Specifically, we focus on two conditions of potential contagion that are both based on experiences of traumatic and stressful events but are conceptually distinct and empirically separable. One condition is the vicarious experience of traumatic life events. For example, several lines of evidence demonstrate that post-traumatic stress disorders (PTSD) can be triggered not only in people who directly experience traumatic events, but also in those who witness them (Blanchard et al., 2004; Perlman et al., 2011; van Wingen et al., 2011). Moreover, recent human studies have demonstrated similar physiological stress responses between an observer and a target undergoing a stressful challenge (e.g., Engert et al., 2014; Dimitroff et al., 2017). Another condition that can occur as part of the broader process of stress contagion is the response of an individual to the aftermath of stress of a social partner, a phenomenon often referred to as “stress crossover” (Wethington, 2000). For example, in a human study mothers were exposed to a social stressor in a separate room from their babies. Upon their reunion with their stressed mothers, babies showed increased heart rate and social avoidance compared to babies in a control condition (Waters et al., 2014), suggesting that mothers’ stressful experiences were contagious to their infants in the aftermath of actual exposure. Moreover, studies have shown that stress-related depression in family or friends may increase the likelihood that a person will exhibit depressive behaviors later in life (Bastiampillai et al., 2013; Joiner, 1994). Like humans, rodents are highly social animals whose behaviors and physiology can be influenced by the emotional state of fellow rodents. Such responses are thought to be adaptive for group survival; the observation of one individual under stress may indicate a threat, so other rodents may benefit from noticing and responding appropriately (Meyza et al., 2017; Meyza and Knapska, 2018; Panksepp and Lahvis, 2011). However, prolonged or repeated emotional and physiological attunement to a stressed social partner may become maladaptive. The purpose of this paper is to review rodent studies which have explored the consequences of
vicarious social stress and social stress crossover using the social defeat paradigm, one of the most robust model of PTSD, depression, and other stress-related illnesses (Carnevali et al., 2017b; Hollis and Kabbaj, 2014; Padurariu et al., 2017; Schoner et al., 2017; Sgoifo et al., 2014). Importantly, we do not aim at providing a comprehensive theoretical framework to understand the existence of simple forms of empathic behaviors in rodents, which has already been elegantly done by others (Meyza et al., 2017; Panksepp and Lahvis, 2011).

Instead, by describing the behavioral and physiological consequences of vicarious social defeat stress and social defeat stress crossover in mice and rats, we aim at highlighting the potential utility of these rodent models for investigating the neurobiological processes and sensory channels of information that allow for the contagion of social defeat stress across individuals.

2. Traditional rodent models of emotional contagion

Before addressing this newly emerging area of social defeat stress contagion in rodent research, it is worth recalling that most studies aimed at rodent empathic-like behaviors have traditionally focused on negative emotional states such as pain or fear (for a thorough review of these studies the reader is referred to Meyza et al., 2017). Briefly, the ability of rodents to sense what their fellow rodents are experiencing has been studied using experimental paradigms such as (i) exposure to a conspecific in pain, (ii) vicarious fear (i.e., witnessing a partner subjected to fear conditioning), (iii) fear learning by proxy (i.e., interacting with a conspecific that was previously conditioned during a fear memory retrieval), and (iv) socially transferred fear (i.e., interacting with a recently conditioned partner in a familiar environment). With the use of these models, it has been shown that rodents can experience contagion of pain and fear both during direct observation of an adverse event (i.e., injection of acetic acid or mild footshocks) and during social interaction with a previously exposed partner in the safe environment of the home cage. Notably, the magnitude of these behavioral responses was modulated by familiarity in models of pain contagion and, to a
lesser extent, in models of fear contagion (Gonzalez-Liencre et al., 2014; Langford et al., 2006). These studies convincingly demonstrated that rodents can acquire a state of distress vicariously through social observation of others suffering from adverse events. However, depression- or anxiety-like behaviors, which are common occurrences of witnessing traumatic and stressful life events, were not evaluated. Moreover, these studies have implemented physical stress of footshock and pain that bears little resemblance to the nature of stress in humans, in which social stressors predominate (Bjorkqvist, 2001; Rohde, 2001).

More recently, more refined mouse and rat models of stress contagion that are based on the social defeat paradigm, an ethologically relevant model of social stress, have started to investigate the consequences of two conceptually distinct and empirically separable conditions of potential social subordination stress contagion, namely vicarious social defeat stress and social defeat stress crossover, on behavioral, physiological, and neurobiological readouts that are relevant in the context of human psychopathology.

3. Vicarious social defeat

Social defeat (also referred to as the resident-intruder test (Miczek, 1979)) is a relatively severe stressor in mice and rats (and also other animals) based on social hierarchy and dominance. Although there are a number of small variations of the social defeat model (for an overview of different protocols see Hollis and Kabbaj, 2014), the basic principle remains the same: a male animal is introduced into the home cage of an older and aggressive male (i.e., resident), who will then threaten and physically assault the intruder until there are clear signs of submission (i.e., social defeat). Upon social defeat, intruders are usually removed from direct physical contact with the resident by a wire partition or cage for the remainder of the test, allowing for psychogenic exposure to aggressive threats without physical harm. This model has been extensively applied to investigate the behavioral, physiological, and neurobiological consequences of single or repeated episodes of social defeat that are relevant in the context of human PTSD, depression, anxiety, and other stress-related
illnesses (Carnevali et al., 2017b; Hollis and Kabbaj, 2014; Padurariu et al., 2017; Schoner et al., 2017; Sgoifo et al., 2014). Notably, because socially defeated animals are exposed to both physical and emotional stress, more recent studies have added a witness component to this model in an attempt to tease apart the various aspects of social defeat stress. The result is a novel “social defeat witness model” or “vicarious social defeat stress paradigm” or “trauma witness model”, as it has been variably called (Patki et al., 2014; Sial et al., 2016; Warren et al., 2013), in which a mouse or rat is forced to witness a male conspecific undergoing social defeat from behind a wire partition or cage within the resident home cage.

We will now summarize the results of studies in mice and rats that demonstrate the viability of adding a witness component to the social defeat model for delineating the consequences of vicarious social defeat stress (Table 1).

3.1. Studies in mice

In the very first study which addressed this topic, adult male mice witnessed the defeat of a conspecific by a CD-1 aggressor mouse for 10 consecutive days (Warren et al., 2013). Twenty-four hours after the last defeat, witness mice showed behavioral signs of social avoidance when confronted with a novel CD-1 mouse compared to the control condition. Remarkably, reduced interaction with a social target was even more evident one month after cessation of vicarious social stress exposure and similar to that exhibited by intruder mice. This behavioral change is particularly relevant because avoidance of trauma-related cues is a hallmark of PTSD and subsets of depression (Foa et al., 2006; Nemeroff et al., 2006) and strongly suggests that witnessing social defeat can vicariously provoke a lasting sensitivity to trauma-related stimuli. Of note, the expression of social avoidance behavior after witnessing social defeat was prevented by chronic fluoxetine treatment. Importantly, the authors of this study demonstrated that sensory exposure to an aggressive resident in the absence of social defeat had no effect on social interaction. Other consequences associated with the vicarious experience of social defeat in this study included (i) deficits in body weight gain, (ii) passive coping in the forced swim test, decreased time spent in the open arms of the
elevated plus maze, and increased plasma corticosterone levels both 24 hours and 1 month after the last defeat, and (iii) depressive-like anhedonia (i.e., reduced preference for the consumption of a sucrose solution) only one month after the last defeat (Table 1). These abnormalities nearly matched those of intruder mice, suggesting that witnessing social defeat is a potent stressor in mice with long-lasting consequences at the behavioral, physiological, and neuroendocrine level. Moreover, witnesses and intruders showed considerable overlap in gene expression dysregulation in the ventral tegmental area (Warren et al., 2013) and nucleus accumbens (Warren et al., 2014). These brain areas form part of a highly complex circuitry that plays an important role in discerning and reacting to rewarding and aversive stimuli in the environment, as well as influencing future responses based on past experience (Russo and Nestler, 2013). Importantly, alterations in this circuitry have been associated with mood disorders (Russo and Nestler, 2013). Interestingly, while the emergence of aberrant behavioral reactivity to social stimuli has been described both in adult and adolescent male witness mice (Li et al., 2018; Warren et al., 2014), neurobiological changes in the nucleus accumbens seemed to depend on the developmental stage of the witness mice (Warren et al., 2014). The emergence of contextual social avoidance behavior was also reported in adult female mice that vicariously experienced the defeat of a male counterpart (Iniguez et al., 2018). This behavioral abnormality was corrected by acute treatment with ketamine or chlordiazepoxide, pharmacological agents used to treat mood-related disorders in the clinical population (Frussa-Filho et al., 1999; Parise et al., 2013).

Alongside social functioning deficits, female witness mice showed depressive-like anhedonia, passive coping in the tail suspension test, a strong trend for anxiety-like behavior on the elevated plus maze test, increased plasma corticosterone levels, and lower body weight gain (Table 1), thus extending to the female sex previously obtained results in male mice (Warren et al., 2013). The expression of aberrant behavior was recently described also in pregnant mice witnessing the defeat of their mates (Miao et al., 2018), including depressive-like behavior during the late period of gestation and anxiety-like behaviors after lactation (Table 1). These behaviors were associated with decreased brain derived...
neurotrophic factor (BDNF) expression in the hippocampus and medial prefrontal cortex, and
increased BDNF expression in the amygdala of pregnant witness mice. Taken together, the
results of these studies strongly suggest that the stress of witnessing social defeat induces
PTSD-like symptomatology and other depressive and anxiety-like phenotypes in mice and
support the utility of the vicarious social defeat model in mice for further investigating the
underlying neurobiological mechanisms in both sexes and different age groups.

3.2. Studies in rats

The emergence of depressive- and anxiety-like behavioral symptoms following the vicarious
experience of social defeat of a cage-mate was demonstrated also in a study conducted in
adult male rats (Patki et al., 2014). These behavioral abnormalities were accompanied by
increased plasma corticosterone levels, deficits in body weight, and impaired long-term, but
not short-term, memory function (Table 1), and resembled those of intruder rats. Notably, a
subsequent study from the same group showed that anxiety-like behaviors and cognitive
deficits in witness rats persisted for up to 6 weeks after the last defeat episode but seemed
to be reversible beyond this time period (i.e., after 8 weeks) (Patki et al., 2015). On the
contrary, the presence of a depression-like behavioral phenotype was still evident 8 weeks
after the last defeat. The authors of this study argued that the different time course of
normalization of behavioral and cognitive responses in witness rats may be due to the fact
that depression affects more complex circuits and mechanisms as compared to anxiety and
memory function, and hence could take more time to normalize (Patki et al., 2015). A more
recent study investigated the cardiovascular consequences of vicarious social defeat in male
rats (Finnell et al., 2017). Remarkably, witnesses exhibited increases in mean arterial
pressure and heart rate that were nearly identical to those of intruders, both during acute
and repeated social stress exposure. This finding is quite surprising given that witness rats
were merely observing the defeat bout of a same-sex conspecific without actually being
engaged in any physical effort. Moreover, re-exposure to the stress environment 6 days after
the last defeat in the absence of the resident produced robust tachycardic and pressor
responses in witness rats that were comparable to those of intruders, which is another important indication that witnessing social defeat can vicariously provoke a lasting sensitivity to trauma-related stimuli, a hallmark of PTSD. Other consequences associated with the vicarious experience of social defeat in this study included a reduction in sucrose solution consumption preference, increases in resting systolic blood pressure, and signs of HPA axis hyperactivity (i.e., elevated plasma corticosterone levels and increased adrenal weight) (Table 1). These findings prompted the same research group to study the effects of vicarious social defeat stress in female rats (Finnell et al., 2018). Similar to the male-based results of their previous investigation, female witnesses showed robust tachycardic and pressor responses to the social defeat of a male intruder (Table 1). These responses did not habituate over time. Importantly, vicarious stress-induced tachycardia was associated with a higher, although modest, incidence of ventricular arrhythmias compared with the control condition. Moreover, daily exposure to vicarious social defeat provoked an increase in resting systolic blood pressure and heart rate and reductions in heart rate variability (Table 1). From a behavioral point of view, female witness rats showed anxiety-like burying during social defeat episodes, depressive-like anhedonia, and passive coping during the forced swim test after 5 days of vicarious social defeat stress (Table 1). Notably, cardiovascular and behavioral alterations were not evident in ovariectomized female rats exposed to the same procedure of vicarious social defeat. Moreover, upon re-exposure to the stress environment in the absence of the resident, intact, but not ovariectomized, female witness rats exhibited increases in peripheral cytokine concentrations and corticotropin-releasing factor and interleukin-1β levels in the central amygdala. According to the authors of this study, these results provide preliminary insights into a putative neuronal mechanism by which ovarian hormones sensitize behavioral and cardiovascular responses to witness stress, as both inflammation and corticotropin-releasing factor are known to activate several brain regions, including the central amygdala (Nadjar et al., 2005; Reul et al., 1998). Taken together, the results of these studies further support the utility of the vicarious social defeat model in rats for elucidating the neurobiological processes that mediate, potentially in a sex-dependent
manner, the negative behavioral and cardiovascular consequences associated with vicarious social stress exposure. An important factor to consider in future studies on sex differences in the vicarious social defeat model is that the behavior of resident animals (for example, the intensity of the attacks) could also be different depending on whether the resident is observed by a male or a female observer. This, in turn, could affect male and female observers in a sex-specific manner.

3.3. Sensory channels of vicarious social defeat stress perception

As mentioned above, in the social defeat witness model a rodent is forced to witness a conspecific undergoing social defeat from behind a wire partition or cage within the resident home cage. Therefore, the term “witness” in this model generally refers to all sensory stimuli associated with the vicarious experience of social defeat and not visual stimuli alone. An obvious question would then be to determine the specific sensory channel(s) through which vicarious social stress can be perceived. In their original study, Warren and colleagues (Warren et al., 2013) used opaque non-perforated dividers to confine separate groups of witness male mice within the resident cage during social defeat. They found that this manipulation completely prevented the acquisition of social avoidance behavior in witness mice. Similar results were obtained in female witness mice (Iniguez et al., 2018), suggesting that visual cues play a central role in the perception of vicarious social stress. However, although visual stimuli were completely blocked in these studies by the use of opaque dividers without holes, the transmission of auditory and chemosensory stimuli might have been blunted as well. To further examine the contribution of olfactory and auditory stress vs visual reinforcement, Patki and colleagues (Patki et al., 2015) exposed male rats only to odor and urine of the aggressive rat or to ultrasound vocalizations emitted by a cage-mate undergoing social defeat (witness rats were kept outside the resident’s cage with visual stimuli blocked by opaque black paper). They demonstrated that smelling the odor and urine of the aggressive rat without social defeat (olfactory stress) or only hearing the social defeat (auditory stress) had no effect on depressive- and anxiety-like behaviors or memory function.
These findings indicate the importance of visually witnessing the traumatic effects of social defeat for the development of behavioral and cognitive alterations in rats.

3.4. The role of social support in buffering the effects of vicarious social defeat

A related matter to the adverse effects of vicarious social stress exposure on subsequent social interaction is the fact that social interaction can in turn play a role in buffering or moderating the effects of that stressor. In rodents, most studies of social buffering have focused on the presence or absence of a conspecific such as the cage-mate after a stressor (DeVries et al., 2003; Kikusui et al., 2006). Specifically, it has been shown that the effects of social defeat on a variety of behavioral, physiological, and neurobiological outcomes were substantially reduced in animals that were group-housed after being directly exposed to the defeat episode (e.g., Lehmann and Herkenham, 2011; McQuaid et al., 2013; Nakayasu and Ishii, 2008; Ruis et al., 2001). Can social buffering also protect against the negative consequences of witnessing social defeat? In the study by Patki and colleagues (Patki et al., 2014), a group of witness and intruder rats was paired housed after each defeat episode. The authors reported that the witness rat was aloof and restless upon initial reunion with the socially defeat partner, but then tried to huddle with its mate and spent time licking and surrounding it for the next hour (Patki et al., 2014). They concluded that these qualitative assessments are representative of comforting and supporting behavior. Interestingly, they documented that depressive- and anxiety-like behaviors were significantly lower in both social defeat experiencing and witnessing rats in the pair-housing condition as compared to when rats were isolated in a single cage after firsthand or vicarious social defeat. These findings were only partially replicated in a subsequent study in adolescent mice (Li et al., 2018), in which social support following social defeat exerted beneficial effects on social behavior only in witness mice but not in mice that had directly experienced the defeat as compared to the single housing condition. In rodents, many different variables are thought to affect the efficacy of social buffering, including the familiarity of the conspecific, the relative hierarchy, sex of the individual and partner, sensory modalities of exposure to that individual,
timing of the availability of social support, presence or absence during stress exposure, and
whether the cage-mate was also stressed (Beery and Kaufer, 2015). These last two aspects
are obviously particularly important in the context of vicarious social stress, and future
studies exploring these variables in all combinations will likely reveal how social support can
buffer against the negative consequences of social defeat stress both in social stress
experiencing and witnessing individuals.

4. Social defeat stress crossover
In the vicarious social defeat model, witness rodents are exposed to a partner that is in
immediate danger of being physically assaulted by the resident. Therefore, this model
seems particularly suitable for addressing specific experimental questions related to the
vicarious experience of traumatic life events. However, another condition that falls under the
umbrella term of stress contagion is the response of an individual to the aftermath of stress
of a social partner. To address this issue, the social defeat stress crossover model implies
that the partner rat or mouse is still stressed due to recent social defeat but the danger is
remote. For example, in a recent study by our group (Carnevali et al., 2017a), a male
‘demonstrator’ rat was paired up with a same-sex ‘observer’ rat for several days to achieve
familiarity before the beginning of the social defeat stress procedure. The demonstrator rat
was then removed from the cage and underwent social defeat stress in another, soundproof
room. Upon the return of the demonstrator rats to the original cage, the cage-mate observers
showed a stress response characterized by a transient increase in heart rate and a reduction
in heart rate variability compared to the control condition. Remarkably, this response
occurred despite the fact that the observer rats had not seen or heard the social defeat
experience of the demonstrator rats. Moreover, we ruled out the potential for olfactory
signals from the aggressive rat to influence response of observers by showing that exposure
to the bedding from the cage of the aggressive rat did not elicit cardiovascular responses in
the observers in the absence of the demonstrator. Most importantly, following repeated
exposure to socially defeated demonstrators, observer rats showed clear behavioral signs of social avoidance when tested in a new social context that nearly matched those of their respective stressed demonstrators. Moreover, observer rats showed elevated plasma corticosterone levels compared to the control condition. This work is novel in showing that social subordination stress occurring out of sight and immediate hearing and smell range can be contagious between rats. Clearly, the social transmission of stress between social partners could exploit different sensory channels. We hypothesized that observer rats may have acquired the stress state of their social partners also through observation of distinctive patterns of overt behavior (e.g., freezing) expressed by demonstrator rats upon their return to the home cage following social defeat. However, future work should address which specific olfactory, visual, and/or auditory signals from the demonstrator rats induced the observer rats to respond to the aftermath of stress of their cage-mates in the safe environment of the home cage. A number of other questions arise when the results of this study are critically evaluated. What are the neurobiological mechanisms underlying emotional-state matching between observer and demonstrator rats? Do the degree of relatedness, sex and/or age of the observer and partner play a role in these contagious stress responses? Would the observer rats have shown similar behavioral and physiological responses if the demonstrator rats had been exposed to a different (nonsocial) stressor? Nevertheless, this study provides preliminary clues about how the stress of those around us may affect our behavior and physiology and prompts a systematic investigation of these research questions.

5. Conclusion

Many studies in humans and nonhuman primates have suggested that stressful experiences may be transmitted among individuals through social interactions within a shared social setting (de Waal and Preston, 2017; Engert et al., 2019; White and Buchanan, 2016). Such contagious stress transcends subjective feeling states to affect the individual’s behavior and
even physiology beyond the daily stressors experienced firsthand (de Waal and Preston, 2017; Engert et al., 2019; White and Buchanan, 2016). The study of stress contagion in rodent research is very much in its early days. However, the findings reviewed here demonstrate that the behavior and physiology of mice and rats can be influenced by the stress state of their conspecifics in two distinct conditions of social defeat stress contagion. In the vicarious social defeat model, witness mice and rats exhibit physiological stress responses and develop a host of behavioral deficits that include contextual social avoidance and other depressive- and anxiety-like phenotypes. Likewise, social interaction with a recently socially defeated partner in the safe environment of the home cage (social defeat stress crossover model) results in increased heart rate and corticosterone as well as increased social avoidance behavior in rats. Importantly, the behavioral and physiological consequences of vicarious social defeat stress seem relatively stable across mouse and rat strains and both sexes, whereas a systematic investigation of strain- and sex-specific responses to the social defeat crossover model is currently lacking. Thus, these rodent models seem to be well-suited for a more in-depth evaluation of the sensory channels of information that allow the contagion of behavioral and physiological effects of social defeat stress among individuals. One of the main questions to be addressed by future studies is whether the contagion is specific to the social aspect of the stressor or is just a consequence of general stress produced by social defeat. Furthermore, while some of the brain areas affected by vicarious social defeat stress exposure and the underlying neural mechanisms have been unveiled by these rodent studies, much remains to be known. The neural basis of stress (and other forms of emotional) contagion revolves around the idea of shared neural networks or neural resonance between individuals. Specifically, one of the most intriguing and intensely debated hypotheses proposed so far is that mirror neurons play an important role in the neural resonance of emotional states (Ferrari and Rizzolatti, 2014; Hickok, 2009). The availability of new techniques of imaging and manipulation of neuronal circuits with single-cell resolution in rodents encourages the use of these models of social defeat stress contagion for investigating the brain structures and neurochemistry involved in the social
sharing of stressful experiences and also for testing the hypothesis about the role of mirroring mechanisms. Given the frequent social situations where stress is likely to occur in our daily life, beyond the daily stressors experienced firsthand, and the deleterious mental and physical consequences associated with stress exposure, a more detailed understanding of the neurobiological processes underlying the contagion of psychophysiological effects of stress across individuals is likely to have important implications for health.
<table>
<thead>
<tr>
<th>Strain/species</th>
<th>Procedure</th>
<th>Observer response</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult male c57BL/6J mice</td>
<td>One 10-min episode of vicarious social defeat daily for 10 consecutive days</td>
<td>Contextual social avoidance (SIT) Depression- (FST and SPT) and anxiety- (EPM test) like behaviors Increased plasma corticosterone levels Deficits in body weight gain Dysregulated gene expression in the VTA and NAc</td>
<td>Warren et al., 2013; Warren et al., 2014</td>
</tr>
<tr>
<td>Adolescent male c57BL/6J mice</td>
<td>One 10-min episode of vicarious social defeat daily for 10 consecutive days</td>
<td>Contextual social avoidance (SIT) Dysregulated gene expression and altered spine density in the NAc</td>
<td>Warren et al., 2014</td>
</tr>
<tr>
<td>Adolescent male c57BL/6J mice</td>
<td>Ten 15-min episodes of vicarious social defeat over a 7-day period</td>
<td>Contextual social avoidance (SIT) Deficits in body weight gain</td>
<td>Li et al., 2018</td>
</tr>
<tr>
<td>Adult female c57BL/6J mice</td>
<td>One 10-min episode of vicarious social defeat daily for 10 consecutive days</td>
<td>Contextual social avoidance (SIT) Depression-like behaviors (TST and SPT) and a strong trend for anxiety-like behavior (EPM test) Increased plasma corticosterone levels Deficits in body weight gain</td>
<td>Iniguez et al., 2018</td>
</tr>
<tr>
<td>Pregnant female c57BL/6J mice</td>
<td>One 5-min episode of vicarious social defeat daily for 17 consecutive days</td>
<td>Depressive-like behavior (SPT) during the late period of gestation Anxiety-like behaviors (EPM and LD tests) after lactation Deficits in body weight gain Changes in BDNF expression in the hippocampus, amygdala and medial prefrontal cortex</td>
<td>Miao et al., 2018</td>
</tr>
<tr>
<td>Adult male Sprague-Dawley rats</td>
<td>One episode of vicarious social defeat daily for 7 consecutive days. Each defeat episode lasted 30 min, including phases of sensory, but not physical, contact between resident and intruder rats</td>
<td>Depressive- (FST and SPT) and anxiety- (EPM, LD, and OPF tests) like behaviors Increased plasma corticosterone levels Impaired long-term memory function (RAVW test) Deficits in body weight gain</td>
<td>Patki et al., 2014; Patki et al., 2015</td>
</tr>
<tr>
<td>Adult male Sprague-Dawley rats</td>
<td>One 15-min episode of vicarious social</td>
<td>Robust pressor and tachycardic responses during acute and</td>
<td>Finnell et al., 2017</td>
</tr>
<tr>
<td>Animal Type</td>
<td>Details</td>
<td>Behavioral and Physiological Effects</td>
<td>References</td>
</tr>
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<td>---------------------</td>
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<td>---------------------------------------------------------------------------</td>
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<tr>
<td>Dawley rats</td>
<td>Defeat daily for 5 consecutive days</td>
<td>Repeated vicarious stress exposure, and during context re-exposure. Increases in resting systolic blood pressure. Depression-like anhedonia (SPT) Elevated plasma corticosterone levels Increased adrenal weight Robust pressor and tachycardic responses during acute and repeated vicarious stress exposure Larger vulnerability to arrhythmias during acute vicarious stress exposure Increases in resting systolic blood pressure and heart rate and reductions in heart rate variability Depression-like (FST and SPT) and anxiety (burying)-like behaviors Elevated peripheral cytokine levels and increased corticotropin-releasing factor and interleukin-1β levels in the central amygdala after context re-exposure</td>
<td>(Finnell et al., 2018)</td>
</tr>
<tr>
<td>Adult female</td>
<td>One 15-min episode of vicarious social defeat daily for 5 consecutive days</td>
<td>Increases in heart rate and increases in heart rate variability upon return of the socially beaten partner in the home cage Social avoidance behavior in a new social context (SAAP test) Elevated plasma corticosterone levels</td>
<td>(Carnevali et al., 2017a)</td>
</tr>
<tr>
<td>Adult male Wistar</td>
<td>Cohabitation with a socially defeated male partner without witnessing the social defeat experience of the partner. Each defeat episode lasted 15 min and was repeated for 4 consecutive days</td>
<td></td>
<td></td>
</tr>
</tbody>
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Abbreviations: BDNF: brain derived neurotrophic factor; LD: light-dark; EPM: elevated plus maze; FST: forced swim test; NAc: nucleus accumbens; OPF: open field; RAVW: radial arm water maze; SAAP: social approach/avoidance test; SIT: social interaction test; SPT: sucrose preference test; TST: tail suspension test; VTA: ventral tegmental area. Detailed experimental procedures are described in the original papers.
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