

Effects of Dominance Status on Neural Activity in Stress-Sensitive Neuronal Pathways

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Abstract

The central amygdala, CeA, is an important brain region that regulates behavioral changes in response to stressful and fear-inducing stimuli. Social subordination is a stressful experience that can activate neurons in the CeA. Social subordination is associated with increased stress sensitivity and elevated fear responses while social dominance is associated with stress resilience. The bed nucleus of the stria terminalis (BNST) is a stress-sensitive brain region that receives robust input from CeA neurons. The objective of this study was to determine if dominant and subordinate hamsters differ in stress/fear-induced activation of CeA cells projecting to the BNST. I hypothesize that subordinate hamsters will activate a greater proportion of BNST-projecting CeA neurons during social defeat stress compared to their dominant counterparts. This experiment involved creating dominant-subordinate relationships in pairs of Syrian hamsters, injecting animals with the neuronal tracer cholera toxin B (CTb), exposing hamsters to social defeat stress with a resident aggressor, collecting brains to be sliced and processed through immunohistochemistry, and quantifying dual cFos-CTb expression. Our findings indicate that males express more c-Fos in CeA neurons following social defeat stress than females. The latency to submit during social defeat stress is also positively correlated with c-Fos expression in the CeA. However, no significant effects of dominance status were found in the stress-induced activation of CeA-BNST neurons. Thus, it is likely that the neural ensembles driving the effects of dominance status on stress reactivity likely exist within different neural circuits.

Introduction

Animals establish social dominance relationships with other members of their species for various reasons: establishment of territories, access to mates, and defense of food resources (Bernstein, 1981; Blanchard et al., 1988; Drews, 1993). A social dominance relationship is created when an animal consistently wins competitive interactions against an opponent and the subordinate animal submits preemptively. Thus, social dominance over other animals can be achieved through various behavioral traits such as aggression, intimidation, and coercion (Rodriguez-Santiago et al., 2020).

Establishing social dominance results in modulation of neuroendocrine, neuropeptide, and neuronal circuits that regulate social behavior and responses to environmental challenges. Social dominance is associated with stress resilience while social subordination is associated with increased stress sensitivity (Karamihalev et al., 2020; Kohn et al., 2016; Overli et al., 2004). For example, mice that achieve social dominance after repeated aggressive encounters exhibit reduced anxiety responses in an open field and less visceral fat than subordinate mice (Bartolomucci et al., 2001; Bartolomucci et al., 2005). Similarly, dominance status in male and female mice confers resilience to the effects of chronic social defeat stress, while subordination confers susceptibility (LeClair et al., 2021). In humans, low social status individuals suffer from higher levels of anxiety, depression, and mortality compared to high social status individuals (Sapolsky, 2005; Zell et al., 2018). Also, human participants that achieve high social status in a laboratory setting, show lower cortisol reactivity in a Trier Social Stress Test (Knight & Mehta, 2017).

To delineate the neuronal mechanisms that govern status-dependent differences in stress resilience, our lab uses a Syrian hamster model (*Mesocricetus auratus*) because both male and female hamsters establish dominance relationships rapidly and maintain their dominance status

throughout a series of social encounters. Socially dominant Syrian hamsters display fewer signs of anxiety following acute social defeat when compared to subordinate or control counterparts (Dulka et al., 2020). We have shown that dominant hamsters exhibit increased neural activity in the posteroventral medial amygdala (MePV) and increased androgen receptor expression in the posterodorsal medial amygdala (MePD) (Cooper et al., 2021; Morrison et al 2014). These findings suggest neural activity within the MePV and MePD contributes a stress resilience in dominance hamsters. In addition, the medial amygdala (MeA) sends robust projections to the bed nucleus of the stria terminalis (BNST), which plays an essential role in regulating stress and fear responses to unpredictable threats (Been and Petrulis, 2011, Goode et al 2019). These findings support the possibility that maintaining social dominance activates specific neuronal ensembles in the MeA that projects to the BNST and may be important for stress resistance.

However, there are other neural pathways that may contribute to the behavioral differences between dominant and subordinate individuals, which include central amygdala (CeA) projections to the BNST. Previous literature has shown that CeA and BNST neurons send projections to the same hypothalamic and brainstem targets to produce phasic and sustained fear, respectively (Davis et al., 2010). These pathways are crucial to the development of anxiety-like behavior following stressful experiences. Other literature has found that neural activity in the CeA-BNST pathway is necessary for the conditioned defeat response. For example, corticotrophin releasing factor acts within a neural circuit that includes CeA neurons projecting to the BNST, which modulates the display of anxiety-like behavior following social defeat stress (Jasnow et al, 2004). Further, literature indicates that infusion of muscimol, an orthosteric agonist for GABAA receptors, into the CeA and basolateral amygdala (BLA) can block the acquisition and expression of conditioned defeat, which indicates that GABAergic neurotransmission within the amygdala is involved in the

acquisition and expression of stress-induced behavioral changes (Jasnow et al, 2001). Based on these findings, I hypothesize that dominant hamsters will activate a CeA-BNST pathway less than their subordinate counterparts during social defeat stress.

Materials and Methods

Subjects

Male and female Syrian hamsters (*Mesocricetus auratus*) were obtained from our breeding colony, which was derived from animals purchased from Charles River Laboratories (Wilmington, MA). Subjects were young adults and 9 weeks old (130-150 g) at the start of the study. One week prior to an experiment, animals were housed individually in polycarbonate cages (12 cm x 27 cm x 16 cm) with corncob bedding, cotton nesting materials, and wire mesh tops. Because Syrian hamsters are highly territorial, low-ranking animals can be wounded when housed in a social group, and adults thrive in the laboratory when housed individually. In our breeding colony, juvenile animals are raised in social groups with environmental enrichment, such as plastic shelters and paper cups. Food and water were available *ad libitum*. Cages were not changed for one week prior to an experiment to allow individuals to scent mark their territory and animals were handled daily to habituate them to the stress of human contact. Animals were housed in a temperature controlled colony room (21 ± 2 °C) and kept on a 14:10 hour light:dark cycle to facilitate gonad development and aggressive behavior. All behavioral protocols were performed during the first three hours of the dark phase of their light:dark cycle. All procedures were approved by the University of Tennessee Institutional Animal Care and Use Committee and are in accordance with ARRIVE guidelines and the National Institutes of Health Guide for the Care and Use of Laboratory Animals.

Formation and Maintenance of Dominance Relationships

We used a resident-intruder format to elicit territorial aggression, weight-matched animals, and assigned them to same-sex resident-intruder dyads. Animals received 12 daily dominance encounters with their partner in 10 min aggressive encounters until a clear winner/loser was established (Figure 1). After a dominance relationship was established, aggressive encounters were reduced to 5 min duration to minimize wounding. Because female hamsters have a remarkably stable 4-day estrous cycle and because aggression levels are inconsistent when hamsters are in the estrus phase of their cycle, we cycle-matched females and skipped encounters when both animals were in estrus. This format resulted in 3 encounters followed by one “skip” day, and 12 encounters occurred over a 16-day period. To use identical protocols for male and female hamsters, male dyads similarly received 12 encounters over 16 days. For each dyadic encounter we recorded live the latency to attack, the number of attacks, the latency to show submissive posture, and the number of flees.

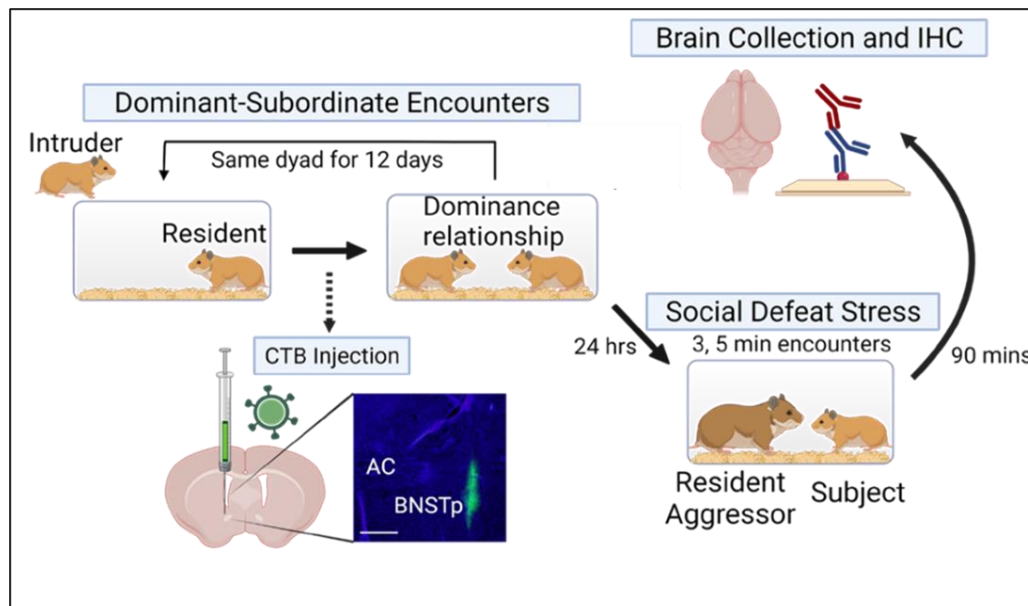


Figure 1: Diagram of the Experiment Design

Stereotaxic Surgery

After the 6th dominance encounter, animals were anesthetized with isoflurane, mounted in a stereotaxic instrument, and injected unilaterally with a fluorescently labeled retrograde tracer cholera toxin B (CTb; Alexa Flour CTb 488). Injections targeted the middle and posterior regions of the BNSTp using stereotaxic coordinates of 1.5 mm anterior to the bregma and 1.35 mm lateral to bregma and 6.2 mm below dura. Injections deposited 100 nL of CTb solution over a 10-minute period, and the needle was left in place an additional 10 minutes following the injection to maximize diffusion. Animals were given 72 hours of surgical recovery before resuming their daily dominance encounters. We have previously shown that interrupting daily dominance encounters for surgery does not reduce the stability of dominance relationships (Dulka et al, 2018, Grizzell et al, 2020).

Social Defeat Stress

After the 12-day dyadic encounters were complete, all of the hamsters received a social defeat stress. Social defeat consisted of 3, 5-minute aggressive interactions at 5-minute inter-trial intervals in which the subject lost to a larger, same-sex aggressor. Novel aggressors were used for each encounter and interactions occurred in the aggressor's home cage. Although aggressors were pre-screened for territorial aggression, variation existed in the latency with which they attacked subjects. Moreover, there was variability in the extent to which subjects defended themselves and the latency with which they submitted. To account for variability in social defeat, we quantified the latency for subjects to submit, the number of attacks subjects received, and the duration of aggression subjects received.

Histology and Immunofluorescent Microscopy Preparation

Ninety minutes after social defeat stress, Animals were anesthetized with isoflurane and perfused transcardially with 100 mL of 0.1M phosphate buffer (PB) followed by 100 mL of 4% paraformaldehyde. Brains were removed and post-fixed in 4% paraformaldehyde for 24 hours, followed by 0.1M PB/30% sucrose solution for 48 hours, and then stored in 0.1M PB/sodium Azide at 4°C. To prepare the brain for immunofluorescent microscopy analysis, the brains were sliced on a freezing microtome at 40 µm and stored as free-floating, coronal sections in 0.05M PB Na Azide. To visualize CTb injection sites, BNST sections were washed with distilled water, mounted on glass slides, and cover-slipped using mounting medium with DAPI. Amygdala tissue sections were processed for c-Fos immunohistochemistry. Sections were rinsed in five PB washes, incubated in a blocking solution of 1% goat serum for 25 minutes, and then incubated in a cFos primary antibody solution for 24 hours at room temperature (1:5000 concentration in PB/Triton, rabbit anti-cFos, Santa Cruz, sc-52). Then, sections were rinsed in PB/Triton and incubated in a secondary antibody solution for 2 hours (1:500 concentration of Alexa Flour 594 in PB/Triton, with 1% goat serum). Sections were rinsed in PB, mounted onto slides, and cover-slipped using mounting medium with DAPI.

Cell Quantification

Quantification of c-Fos-positive, CTb-positive, and c-Fos/CTb-positive cells was performed using an Olympus BX51 microscope. CTb injection sites were plotted on templates from the Syrian hamster stereotaxic atlas (Figure 2; Wood et al, 2001). Animals were excluded from statistical analysis if the center of their CTb injection was outside the BNST. Images of CTb fluorescence and c-Fos immunoreactivity were collected in the MePD, MePV, and CeA at 10x magnification and using a 500 x 500 µm imaging window. This project is focused on the

activity of CeA neurons and is an extension of a project on BNST-projecting neurons in the MeA. We collected 3-6 images per animal and centered the imaging window on a population of CTb cells within the CeA boundary. This procedure resulted in green CTb-positive cells and red c-Fos-positive cells, and merged images were evaluated for dual c-Fos/CTb-positive cells (Figure 3). We used MCID image analysis software to quantify single-labeled c-Fos⁺ cells and CTb⁺ cells above background, and we quantified dual c-Fos/CTb⁺ cells manually. I quantified images for 22 subjects using this process.

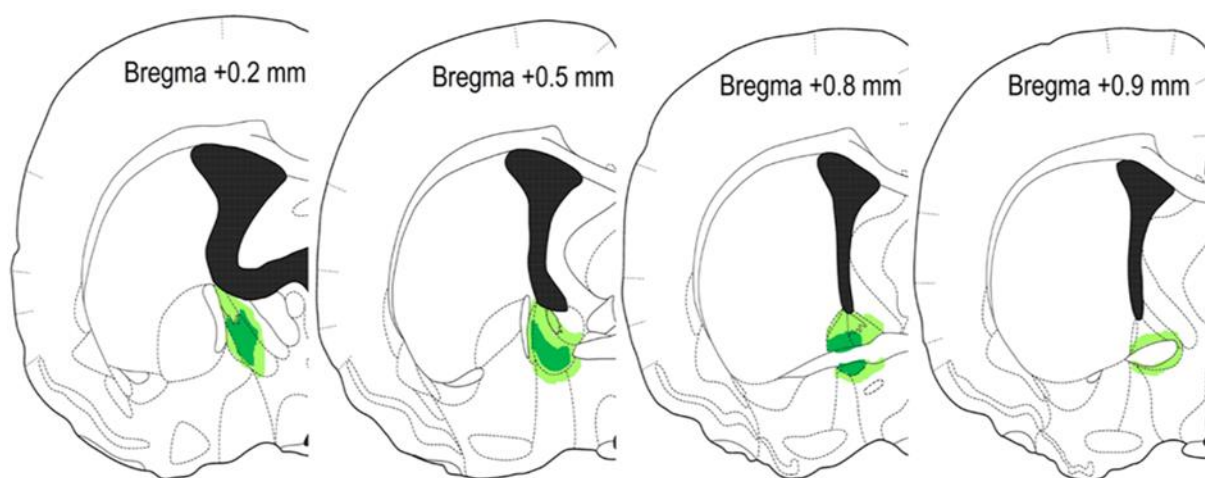


Figure 2: Stereotaxic atlas images depicting the maximum (light green) and minimum (dark green) spread of CTB injections in the BNST.

Due to high variability in the intensity and irregular geometry of CTB fluorescence, the MCID software had difficulty properly quantifying CTB⁺ cells. As a result, the remaining 15 subjects were quantified by an operationalized hand count process to avoid Type II errors. This involved using similar criteria as MCID for c-Fos and CTB identification and establishing reliability with the software. After quantifying c-Fos and CTB images manually, we overlaid the two images while adjusting the opacity value for the overlaid image to reveal which c-Fos⁺ and CTB⁺ were colocalized. Interrater reliability for cell counting was established at 90% agreement

between researchers and between researchers and the software.

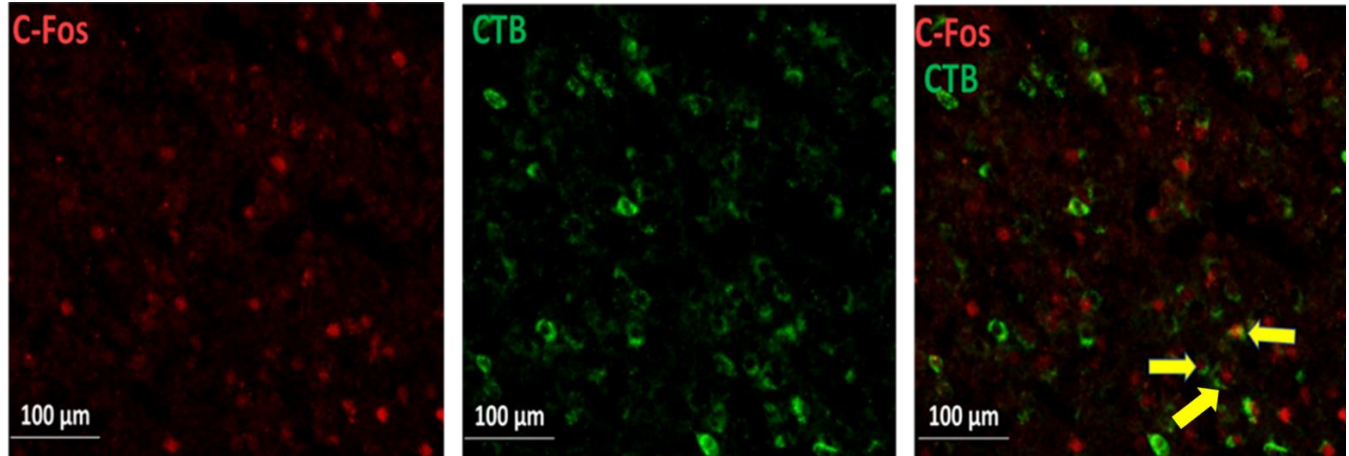


Figure 3: Example images from CTB 202 Section 5 of a c-Fos image, CTB image, and dual image. Yellow arrows indicate examples of dual-labelled cells.

Data Analysis

All data regarding cFos and CTB quantification were analyzed using Generalized Linear Mixed Models fit to a Negative Binomial Distribution in R. ANOVA was not used due to data not being normally distributed. Sex, dominance status, and interactions of sex and dominance status were included in the data analysis. Linear regressions were used to test correlations between latency to submit and cell counts. The level of significance was set at $p \leq 0.05$.

Results

c-Fos Expression

To examine the number of cFos+ cells after social defeat, the number of c-Fos+ cells was compared between males and females and between dominant and subordinate animals. There was a main effect of sex on c-Fos expression in the CeA indicating that male hamsters expressed more c-Fos+ neurons than female hamsters (Sex χ^2 (1, N =37) = 0.2802, p = 0.001406.). There were no significant main effects found for status (Status χ^2 (1, N =37) = 0.2802, p = 0.596600) or status by sex (Status:Sex χ^2 (1, N =37) = 1.0049, p = 0.316116). Furthermore, a linear regression analysis of the latency to submit and the number of c-Fos+ neurons showed that c-Fos expression was positively correlated with longer latencies to submit (#c-Fos Sum2 (1, N =37) = 20896, F =8.5503 p = 0.00664.). Specifically, the longer the latency to submit, the greater number of c-Fos+ cells animals showed in the CeA.

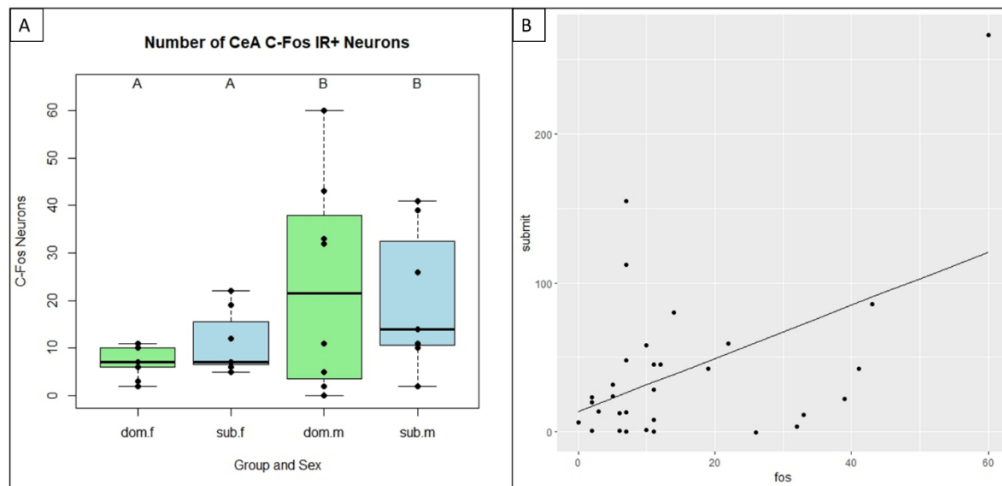


Figure 4: Quantification of c-Fos expression in CeA neurons. A) Graph showing the number of c-Fos+ cells in dominant females, subordinate females, dominant males, and subordinate males after acute social defeat stress. Bars with different letters (A or B) are significantly different from one another, which indicates a main effect of sex on c-Fos expression.

B) Graph visualizing a correlation between latency to submit during social defeat stress and the number of c-Fos+ cells in the CeA.

CTb Expression

To examine CTb+ expression, the number of fluorescent CTb+ cells in the CeA was analyzed for effects of sex and dominance status. Main effects of sex and dominance status were not found such that subjects did not significantly differ in the proportion of CTb+ cells in CeA neurons that projected to the BNST (Status χ^2 (1, N =37) = 0.6777, p = 0.4104. Sex χ^2 (1, N =37) = 1.0356, p = 0.3088. Status:Sex χ^2 (1, N =37) = 2.5430, p = 0.1108.). Nevertheless, there was a non-significant trend for dominant males to have fewer CTb+ cells compared to other treatment groups.

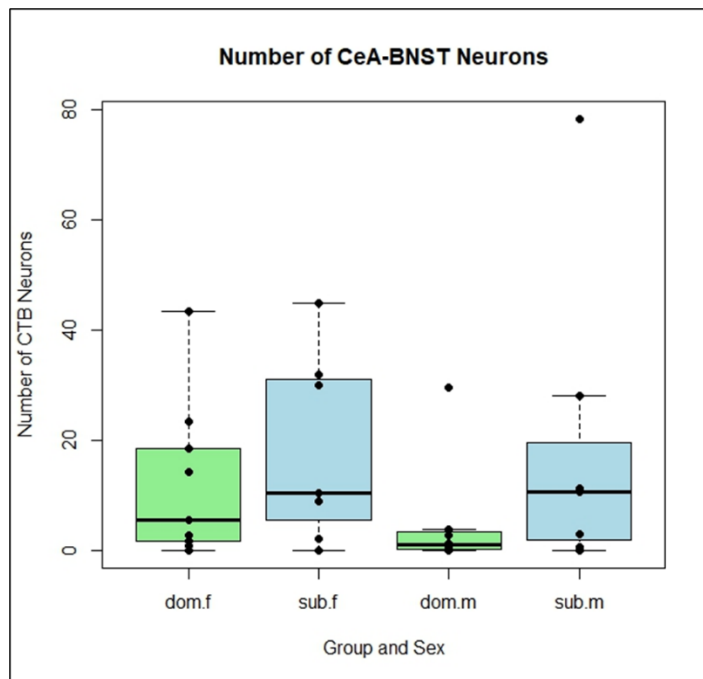


Figure 5: Graph showing the number of CTb+ neurons in dominant females, subordinate females, dominant males, and subordinate males.

Co-labelled Neurons

To quantify co-labelled neurons, the number of c-Fos+ and CTb+ neurons were used to calculate the number co-labelled neurons for each subject across all slide sections belonging to the subject. The number of c-Fos+, CTb+, and co-labelled cells were averaged into a single value

based on the number of tissue sample available, and were grouped together by sex and status. For the number of c-Fos+ and CTb+ neurons, there were no significant effects found for dominance status or sex (Status χ^2 (1, N=37) = 1.2624, p = 0.2611, Sex χ^2 (1, N=37) = 0.4706, p = 0.4927, and Status by Sex (χ^2 (1, N=37) = -0.1624, p = 0.6869). However, there was a non-significant trend for dominant males to have the least amount of co-labelled neurons. Moreover, there was a non-significant trend for subordinate males and females to have more co-labelled neurons compared to their dominant counterparts. Similarly, for the proportion of activated CeA-BNST neurons, no significant effects were found (Status χ^2 (1, N=37) = 0.6777, p = 0.4104, Sex χ^2 (1, N=37) = 1.0356, p = 0.3088, and Status by Sex (χ^2 (1, N=37) = 2.5430, p = 0.1108).

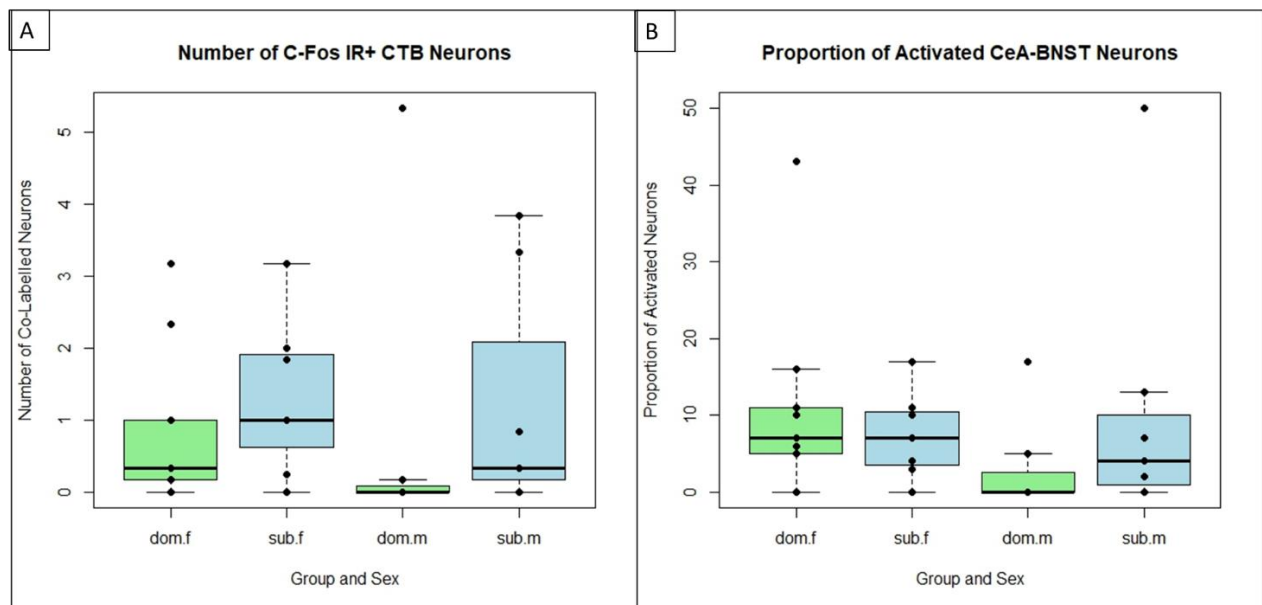


Figure 6: A: Graph showing the number of co-labelled neurons in the CeA-BNST pathway. B: Graph showing the proportion of activated neurons in the CeA-BNST pathway. Proportions were calculated as c-Fos+CTB / CTB

Discussion

CeA-BNST Pathway

CTb is a fluorescently labeled retrograde tracer that we used to label CeA neurons projecting to the BNST. We found that all subjects had a similar number of cells in a CeA-BNST pathway, although dominant males tended to have the fewest number of labeled cells. However, male subjects expressed more c-Fos-positive cells in the CeA after social defeat stress than female subjects. This sex difference is consistent with previous literature showing that males have a greater conditioned defeat response than females (Huhman et al., 2003). Moreover, the latency to submit was positively correlated with c-Fos expression in the CeA. This positive correlation could be due to the increased emotional intensity of a longer period of mutual aggression during the social defeat stress encounter. An acute social defeat encounter with a larger, more aggressive hamster will produce intense emotional and physiological responses from the subject. As such, a longer period of mutual aggression and delayed submission may be more stressful and generate more neural activity in the CeA. However, we did not find significant effects of dominance status on c-Fos expression in CeA-BNST neurons. Thus, dominance status may not modulate the emotional intensity of social defeat stress, at least insofar as it is represented in the CeA. Overall, it appears likely that the CeA-BNST neural circuit does not drive the status-dependent changes in the conditioned defeat response.

One possibility for our findings is that other neural ensembles in the CeA mediate the effects of dominance status on stress vulnerability. For example, the CeA receives projections from the paraventricular nucleus of the thalamus (PVT) and the BLA, which have key roles in stress and fear responses (Davis et al., 2010). CeA cells that receive these inputs may also not exclusively project to the BNST. The CeA also projects to many areas of the hypothalamus and

the brainstem nuclei, and these projections contribute to the expression of phasic and sustained fear responses (Davis et al., 2010). Moreover, there was a non-significant trend for dominant males to show fewer CTb/c-Fos co-labelled cells than subordinate males. This trend could be influenced by dominant males possibly having fewer neurons projecting to BNST or as an effect of CTb injection variability in the BNST. Thus, for possible future directions, the current data could be expanded by testing whether c-Fos expression in the CeA is associated with c-Fos expression in either brain regions projecting to the CeA or downstream brain regions.

This study was conducted in conjunction with a project focused on activity within a MeA-BNST pathway. Interestingly, the CeA-BNST data are opposite to the pattern found in the MeA-BNST pathway. Our preliminary data indicate that dominant males have more c-Fos+/CTb+ dual labeled cells in the MePD and MePV during the maintenance of dominance status and during social defeat stress compared to subordinate males; although there was no effect of dominance status in females (Hooker et al., 2022). However, the CeA-BNST data are consistent with literature demonstrating a role for CeA neurons controlling stress reactivity and sustained anxiety. Moreover, other neural circuits may mediate effects of social status include the MeA-BNST. Interestingly, activity in the MeA and CeA appear inversely correlated such that those that achieve a dominant social status show more activity in the MeA while those that acquire a subordinate social status show more activity in the CeA.

Limitations

This research study originally had 42 subjects to work with, but the subject number went down to 37 due to some tissue slides for individual subjects being damaged or overexposed and no replacements were available. Fluorescence can be affected by age of the tissue or light exposure, which can interfere with the ability to identify stained cells. As a result, reliable cell

quantification for these dropped subjects could not be achieved. Moreover, this research study also relied on a small subject population; as such, trends in the data could be further strengthened if the number of subjects were increased. Variability in BNST injection sites could also have affected c-Fos/CTB co-labelling. Injections targeted posterior regions of the BNST, while CeA cells send robust projections to anterior regions of the BNST. If injection sites had been localized to more anterior portions of the BNST, more CTB cells may have been found in the CeA and different findings may have emerged. Additional data that are needed to extend these findings includes quantification of behavior during acute social defeat scores. This is important as the number of attacks and duration of aggression subjects receive from the resident aggressor may impact the activity of the CeA-BNST pathway.

Overall, this work provides insight into experience-dependent neural plasticity contributing to sex and status differences in stress resilience. This line of research should contribute to our understanding of the mechanisms underlying differences between men and women in the prevalence and etiology of stress-related disorders. These insights could then be used to develop novel behavioral and pharmacological treatments for stress-related mental illness.

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References

- Bartolomucci, A., Palanza, P., Gaspani, L., Limiroli, E., Panerai, A.E., Ceresini, G., Poli, M.D., Parmigiani, S. (2001). Social status in mice: behavioral, endocrine and immune changes are context dependent. *Physiology and Behavior*. 73, 401–410.
- Bartolomucci, A., Palanza, P., Sacerdote, P., Panerai, A.E., Sgoifo, A., Dantzer, R., Parmigiani, S. (2005). Social factors and individual vulnerability to chronic stress exposure. *Neuroscience Biobehavior. Rev.* 29, 67–81.
- Been, L. E., & Petrulis, A. (2011). Chemosensory and hormone information are relayed directly between the medial amygdala, posterior bed nucleus of the stria terminalis, and medial preoptic area in male Syrian hamsters. *Hormones and behavior*, 59(4), 536–548.
<https://doi.org/10.1016/j.yhbeh.2011.02.005>
- Bernstein, I. S. (1981). Dominance: The baby and the bathwater. *Behavioral and Brain Sciences*, 4(3), 419–429. <https://doi.org/10.1017/S0140525X00009614>
- Blanchard, R. J., Flannelly, K. J., & Blanchard, D. C. (1988). Life-span studies of dominance and aggression in established colonies of laboratory rats. *Physiology & Behavior*, 43(1), 1–7. [https://doi.org/10.1016/0031-9384\(88\)90089-3](https://doi.org/10.1016/0031-9384(88)90089-3)
- Cooper, M.A., Clinard, C.T., Dulka, B.N., Grizzell, J.A., Loewen, A.L., Campbell, A.V., Adler, S.G. (2021). Gonadal steroid hormone receptors in the medial amygdala contribute to experience-dependent changes in stress vulnerability. *Psychoneuroendocrinology*, 129, 105249.
- Davis, M., Walker, D., Miles, L., Grillon, C. (2010). Phasic vs Sustained Fear in Rats and Humans: Role of the Extended Amygdala in Fear vs Anxiety. *Neuropsychopharmacology* 35, 105–135

<https://doi.org/10.1038/npp.2009.109>

Drews, C. (1993). The Concept and Definition of Dominance in Animal Behaviour. *Behaviour*, 125(3/4), 283–313.

Dulka, B. N., Bress, K. S., Grizzell, J. A., & Cooper, M. A. (2018). Social Dominance Modulates Stress-induced Neural Activity in Medial Prefrontal Cortex Projections to the Basolateral Amygdala. *Neuroscience*, 388, 274–283.

<https://doi.org/10.1016/j.neuroscience.2018.07.042>

Dulka, E. A., DeFazio, R. A., & Moenter, S. M. (2020). Chemogenetic Suppression of GnRH Neurons during Pubertal Development Can Alter Adult GnRH Neuron Firing Rate and Reproductive Parameters in Female Mice. *eNeuro*, 7(3), ENEURO.0223-20.2020.

<https://doi.org/10.1523/ENEURO.0223-20.2020>

Goode, D. T., Ressler, L. R., Acca, M.R., Miles W.O. Maren, S. (2019) Bed nucleus of the stria terminalis regulates fear to unpredictable threat signals. *eLife* 8:e46525.

<https://doi.org/10.7554/eLife.46525>

Grizzell, J.A., Clarity, T.T., Graham, N.B., Dulka, B.N., and Cooper, M.A. (2020) Activity of a vmPFC-DRN pathway corresponds with resistance to acute social defeat stress. *Front. Neural Circuits* 14:50. doi: 10.3389/fncir.2020.00050

Huhman, K. L., Solomon, M. B., Janicki, M., Harmon, A. C., Lin, S. M., Israel, J. E., & Jasnow, A. M. (2003). Conditioned defeat in male and female Syrian hamsters. *Hormones and behavior*, 44(3), 293-299.

Hooker, M.K., Jenkins, M.S., Whitten, C.J., and Cooper, M.A. (2022). Effects of dominance status on stress-induced activity of medial amygdala neurons projecting to the bed nucleus of the stria terminalis. Poster presentation at the Stress Neurobiology Workshop,

Columbia, SC.

Jasnow, A. M., Davis, M., & Huhman, K. L. (2001). Activation of GABAA receptors in the amygdala blocks the acquisition and expression of conditioned defeat in Syrian hamsters, *Brain Research, Volume 920, Issues 1–2*, P. 142-150, ISSN 0006-8993, [https://doi.org/10.1016/S0006-8993\(01\)03054-2](https://doi.org/10.1016/S0006-8993(01)03054-2).

Jasnow, A. M., Davis, M., & Huhman, K. L. (2004). Involvement of central amygdala and bed nucleus of the stria terminalis corticotropin-releasing factor in behavioral responses to social defeat. *Behavioral neuroscience*, 118(5), 1052–1061. <https://doi.org/10.1037/0735-7044.118.5.1052>

Karamihalev, S., Brivio, E., Flachskamm, C., Stoffel, R., Schmidt, M. V., & Chen, A. (2020). Social dominance mediates behavioral adaptation to chronic stress in a sex-specific manner. *elife*, 9, e58723.

Knight, E. L., & Mehta, P. H. (2017). Hierarchy stability moderates the effect of status on stress and performance in humans. *Proceedings of the National Academy of Sciences*, 114(1), 78–83. <https://doi.org/10.1073/pnas.1609811114>

Kohn, J. N., Snyder-Mackler, N., Barreiro, L. B., Johnson, Z. P., Tung, J., Wilson, M. E. (2016). Dominance rank causally affects personality and glucocorticoid regulation in female rhesus macaques. *Psychoneuroendocrinology*. 74:179-88.

LeClair, K. B., Chan, K. L., Kaster, M. P., Parise, L. F., Burnett, C. J., & Russo, S. J. (2021). Individual history of winning and hierarchy landscape influence stress susceptibility in mice. *eLife*, 10, e71401. <https://doi.org/10.7554/eLife.71401>

Morrison, K. E., Bader, L. R., Clinard, C. T., Gerhard, D. M., Gross, S. E., Cooper, M. A. (2014).

- Maintenance of dominance status is necessary for resistance to social defeat stress in Syrian hamsters. *Behavioral Brain Research*. 270:277-86.
- Rodriguez-Santiago, M., Nührenberg, P., Derry, J. Deussen, O., Francisco, O.A., Garrison L.K., Garza S. F., Hofmann, H.A., Jordan, A. (2022). Behavioral traits that define social dominance are the same that reduce social influence in a consensus task. *Proceedings of the National Academy of Sciences*, <https://doi.org/10.1073/pnas.2000158117>
- Sapolsky, R. M. (2005). The Influence of Social Hierarchy on Primate Health. *Science*, 308(5722), 648–652. <https://doi.org/10.1126/science.1106477>
- Øverli, Ø., Korzan, W. J., Höglund, E., Winberg, S., Bollig, H., Watt, M., Forster, G. L., Barton, B. A., ØVerli, E., Renner, K. J., & Summers, C. H. (2004). Stress coping style predicts aggression and social dominance in rainbow trout. *Hormones and behavior*, 45(4), 235–241. <https://doi.org/10.1016/j.yhbeh.2003.12.002>
- Wood, I. R., Morin, P. L. (2001). A Stereotaxic Atlas of the Golden Hamster Brain
- Zell, E., S., J. E., & Krizan, Z. (2018). Subjective social status and health: A meta-analysis of community and society ladders. *Health Psychology*, 37(10), 979–987. <https://doi.org/10.1037/hea0000667>
- Zhang, W. H., Zhang, J. Y., Holmes, A., & Pan, B. X. (2021). Amygdala circuit substrates for stress adaptation and adversity. *Biological psychiatry*, 89(9), 847-856.