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Foreword

As scientists, it is our duty to communicate our findings with the general public. As junior scientists, however, it is our plight to conduct work worthy of communicating to the public. We learned many things during our times at Concordia: That sample statistics estimate population parameters, that we are apt to overestimate the extent of our own knowledge, and that it is strictly forbidden to eat in any lab spaces. But one of the lessons that we had to learn for ourselves was that scientific inquiry, regardless of its results or impact, is a worthwhile endeavour. It is with that lesson in mind that we are proud to present the first edition of Concordia's Journal of Psychology and Neuroscience (CJPN). The articles within this issue reflect the range of research being conducted in Psychology and Neuroscience at Concordia.

CJPN is dedicated to improving Concordia's Psychology department by providing publishing and editing experience to graduate and undergraduate students alike. Our mission is to provide students with the unique ability to peer review and publish their high-quality work. With an increased demand for publications at the undergraduate level, we believe CJPN will be a key resource for psychology and neuroscience students to establish themselves in their respective fields. It is our hope that the psychology department will be strengthened by CJPN's commitment to excellence and our desire to help students flourish.

We are thankful for the support of the Department of Psychology and the many professors who believed in our mission to highlight undergraduate researchers and communicate psychological science. We also want to sincerely thank our team for all the hard work they have put into the review process over the last 6 months. Lastly, we would like to congratulate all the authors on their publications! We hope that the experiences you have gained throughout the editorial process will provide you with a solid foundation upon which you can continue to grow as researchers.

“Our errors are surely not such awfully solemn things. In a world where we are so certain to incur them in spite of all our caution, a certain lightness of heart seems healthier than this excessive nervousness on their behalf.” — William James

Ryan Aberback & Jillian Caplan
Editors in Chief

In Memoriam: Dr. Nadia Chaudhri

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Social Anxiety and Cannabis Problems: Examining the Moderating Role of Perceptions of Parental Use and Approval *

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Cannabis problems (e.g., addiction, misuse) are prevalent among young adults. Some literature points to social anxiety (SA) as a predictor; however, the theoretical and empirical evidence suggests that the pathway is unclear. Perceptions of parental cannabis use (descriptive norms) and approval of cannabis use (injunctive norms), as well as gender differences, might influence the SA-cannabis problems association. The purpose of the current study was to examine the moderating role of parental injunctive and descriptive norms and gender on the SA-cannabis problems association. Undergraduate participants ($N = 172$) self-reported on age, gender, SA, parental injunctive and descriptive norms, and cannabis problems. The results suggested that SA was a positive predictor of cannabis problems, but only among men, and this effect was particularly strong for men high in parental injunctive norms. This research has the potential to clarify the risk trajectory and inform prevention and intervention efforts targeting problematic cannabis use among those high in SA.

Social Anxiety and Cannabis Problems

Cannabis is the most commonly used drug in Canada, particularly among young adults (18 to 24 years old) [1, 2]. There is a myriad of unfavourable consequences associated with cannabis use, such as motor vehicle accidents, and increased likelihood of using other illegal drugs [3]. Because of the problems associated with cannabis use, there is a need to better characterize the risk pathway to cannabis misuse, which could contribute to the development of preventative interventions aimed at mitigating cannabis problems, particularly among young adults.

Anxiety disorders and in particular social anxiety (SA) have been implicated in predicting cannabis problems [4, 5, 6]. SA is defined as a phobic disorder characterized by fear of being judged and scrutinized by others in social situations that are either endured with extreme discomfort or avoided altogether [7, 8]. Research shows that young adults with SA are five times more likely to develop cannabis problems than those without SA [6].

Three theoretical models lend themselves to elucidating the association between SA and cannabis problems. The tension-reduction model suggests that cannabis is used to reduce symptoms of anxiety and emotional distress in social situations (i.e., to cope) [5]. Supporting this, using questionnaires in a cross-sectional study, Buckner and colleagues (2007) found that undergraduates high in SA used cannabis as a way to reduce tension and cope with SA symptoms [5]. The substance refusal efficacy theory postulates that those high in SA may have difficulty refusing substances offered by their peers [9]. This fits with the definition of SA, such that those high in SA fear neg-

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ative evaluation from their peers, which might happen if they refuse substances [5]. Supporting this theory, with the use of multiple self-report questionnaires, cross-sectional studies show that those high in SA use cannabis to conform with their cannabis-using peers [5, 10]. Finally, the buffer perspective suggests a protective association between SA and cannabis problems, such that those high in SA will avoid social situations, thus have reduced opportunities and pressure to use cannabis. Indeed, a study conducted by Nelemans and colleagues (2016) found that the link between SA and cannabis use was moderated by peer involvement, such that elevated SA was associated with a reduced likelihood of cannabis use, among those who had less involvement with their peers [11].

Taken together, theoretical models and empirical evidence support a complex link between SA and cannabis problems. On the one hand, those high in SA may use cannabis to reduce tension and conform with their peers, while on the other hand, those high in SA may avoid cannabis because it is typically used in a social context that is not sought out. This complexity points to the need to consider how other factors may be impacting this association.

Moderators of the Risk Pathway

Social Norms

According to Azjen's (1991) theory of planned behaviour, behavioural intentions are determined by one's perception of whether others perform and approve of the behaviour (i.e., social norms) [12]. There are two types of social norms that have been investigated as contributing to the SA-cannabis use association: (1) descriptive norms, which are an individual's perceptions of others' cannabis use, and (2) injunctive norms, which are an individual's perceptions of others' approval of cannabis use and related behaviours [13, 14, 15].

Research demonstrates that peer use and approval are positively correlated with self-reported cannabis problems [17]. Buckner and colleagues (2006) found that peer cannabis use moderated the SA-cannabis association, such that SA was a positive predictor of cannabis problems when peers were perceived as heavy cannabis users [18]. In addition, Buckner (2013) found that college students who experienced negative affect in social situations were more likely to use cannabis to cope with their anxious symptoms and conform with their peers if they perceived their peers to approve of cannabis use [5, 13].

The role of perceived parental approval in the SA-cannabis problems association has also been investigated. Ecker and Buckner (2014) found that elevated SA was associated with increased cannabis problems when individuals perceived that their parents approved of cannabis use [17]. Likewise, Foster and colleagues (2016) found that elevated SA predicted increased cannabis problems among those with high perceived parental approval [16]. Interestingly, this work suggests that norms may play a role even if those high in SA avoid social contexts typical of cannabis use, as they may look to parents for approval. Notably, researchers have not yet investigated perceptions of actual parental cannabis use (i.e., descriptive norms).

Gender

Evidence is mixed regarding the potential role of gender in the SA-cannabis problems relationship. Some studies show no gender differences in the association between SA and cannabis problems [5, 14], while others support gender effects. For example, some evidence indicates that

men, and more specifically, men with SA, have more cannabis problems than women [17,19]. However, Buckner and colleagues (2006) found that the association between SA and cannabis use – and the interaction between SA and peer influence on cannabis problems – was only supported for women, such that an increase in SA was linked to cannabis problems when perceiving that friends approve of cannabis use, only among women [18]. This work suggests that gender plays a role in the SA-cannabis problems association; however, the direction of the association is unclear.

The Current Study

The goal of the current online study was to test the effect of SA on cannabis problems among young adults, as moderated by parental descriptive norms, parental injunctive norms, and gender. A cross-sectional online questionnaire study was used to assess the following hypotheses: (1) parental descriptive and injunctive norms, and gender would moderate the association between SA and cannabis problems, (2) elevated SA would positively predict an increase in cannabis problems among those who perceive their parents as using cannabis (i.e., high descriptive norms), and this effect would be particularly strong for men, and (3) elevated SA would positively predict cannabis problems among those who perceive their parents as approving of cannabis use (i.e., high injunctive norms), and this effect would be particularly strong for men.

Method

Participants

The sample included 172 young adults, of which 154 (83.8% women, 16.2% men) were retained for analyses for two main reasons. First, these individuals had complete data on the questionnaires of interest. Second, given that the sample size of self-reported non-binary individuals was small ($n = 4$), we chose to only include individuals who self-reported as man or woman. These young adults ranged from 18 to 34 years old ($M = 21.67$, $SD = 2.471$). Based on a priori power analysis (conducted in G*Power), a sample size of 103 has sufficient power ($>.80$) to detect an effect size of .15 (with $\alpha=.05$) for hypothesis testing. Thus, the total sample size of 154 was sufficiently powered. The participants were undergraduate students from Concordia University and young adults from the greater Montreal area. Participants reported their racial/ethnic background (Caucasian = 66.2%; Aboriginal = .6%; Arab = 5.2%; African American/Black = 3.2%; Chinese = 3.2%; Filipino = .6%; Latin American = 3.2%; South Asian = 6.5%; West Asian = 3.9%; Multiracial = 7.1%). Inclusion criteria were an age of at least 18 years and fluency in English, as the questionnaires used were all in English. There were no exclusion criteria for the current study.

Measures

Demographics questionnaire. Participants completed a demographic questionnaire wherein they self-reported their age and gender.

Liebowitz Social Anxiety Scale (LSAS) [20]. The LSAS is a 24-item self-report questionnaire assessing fear/anxiety and avoidance in various social situations (e.g., ‘using a telephone in public’). Participants indicated their level of fear/anxiety using a 4-point scale from 0 (none) to 3 (severe), and their level of avoidance using a 4-point scale from 0 (never) to 3 (usually). One global sum score was calculated by combining the total score from the fear/anxiety subscale and the total score from the avoidance sub-scale (i.e., sum of 24 items). Previous studies have suggested that the LSAS demonstrates significant test-retest reliability, and convergent, divergent, and discriminant validity [5, 21]. In the current study, the LSAS demonstrated high internal consistency

(Cronbach's $\alpha = 0.93$).

Drinking Norms Rating Form [22] adapted for Cannabis Use. This adapted measure is a 16-item self-report questionnaire assessing perceptions of parents' actual cannabis use (i.e., parental descriptive norms; e.g., 'How often do you think your parents/legal guardian(s) use cannabis?'). Participants reported their perceptions with the use of open-ended response options and behavioural anchors. The items were summed to reflect the amount of cannabis consumed (in grams) per week. Higher scores reflected perceiving higher parental cannabis use. Baer's (1991) Drinking Norms Rating Form has demonstrated adequate face validity, predictive utility, and moderate test-retest reliability (0.69) [23].

Perceived Approval of Risky Drinking Inventory (PARDI) [24] adapted for Cannabis Use. The PARDI adapted for Cannabis Use is a 29-item self-report questionnaire assessing participants' perceptions of parental approval of cannabis use and associated behaviours (i.e., parental injunctive norms; e.g., 'You use cannabis in order to flirt, have sex, or increase the likelihood of hooking up with someone'). Participants indicated to what degree they believe their parents would approve of a list of behaviours on a 7-point scale from 1 (strongly disapprove) to 7 (strongly approve). The PARDI adapted for Cannabis Use was scored by summing each participant's responses, with higher sums denoting higher perceived parental approval of cannabis behaviours. The PARDI is a new measure that has not yet been used in other cannabis use studies; however, it is currently being analyzed for its psychometric properties. In the present study, the internal consistency was high (Cronbach's $\alpha = 0.97$).

Cannabis Use Problems Identification Test (CUPIT) [25]. The CUPIT is a 16-item self-report questionnaire assessing participants' current and potential cannabis problems using various scales and behavioural anchors (e.g., 'How difficult do you think you would find it to stop using or go without cannabis altogether'). This measure was scored by summing each participant's responses. Higher scores reflect a higher number of cannabis problems. The CUPIT had high internal consistency in the current study (Cronbach's $\alpha = 0.88$) and has shown excellent test-retest reliability (0.89-0.99), and significant construct and discriminative validity in previous testing [25].

Procedure

The online study was advertised on Concordia's participant pool website and through flyers posted around the greater Montreal area. Interested undergraduate students contacted the laboratory manager, who gave them a unique code and the link to access the survey. Participants were then asked to provide informed consent, and those who did, gained access to the study. The questionnaires took approximately an hour and a half to complete. Participants from the Concordia participant pool were compensated with one course credit and participants from the community were given the option to enter a draw for one of two \$50 cash prizes. This online study was approved by the Concordia University Human Research Ethics Committee.

Results

Data Integrity

Listwise deletion, rather than pairwise deletion, was used to address missing data, as it allowed for better interpretation of the data and has been the preferred method in other SA and cannabis use studies [e.g., 32]. Fourteen of the total 172 participants had missing data on at least one variable of interest, and were therefore excluded from analyses. A t-test was conducted to as-

sess whether those with complete data versus those without (i.e., missing on at least one variable) differed in age. The results suggest that there was no statistically significant difference between the 158 participants with complete data (M age = 21.61, SD = 2.483) versus the 14 participants without (M age = 21.64, SD = 1.277) on age ($t(170)$ = -.052, p = .958). A chi-square test was conducted to ensure that participants with complete data did not differ from those without complete data based on gender. The results suggest that there was no statistically significant difference between participants with complete versus incomplete data based on gender ($\chi^2(3, N = 172)$ = 2.024, p = .567).

The data were screened for the assumptions of moderated multiple linear regression, including outliers, multicollinearity, and linearity. Outliers were defined as data falling $|3.29|$ standard deviations (SD) above and below the mean and were replaced with the maximum and minimum non-outlier values [26]. The assumption of multicollinearity was checked using the Variance Inflation Factor (VIF) and tolerance statistic. Our results suggest that the assumption of multicollinearity was met (VIF = .997, Tolerance = 1.003). Finally, the assumption of linearity was assessed by visual inspection of the data using a scatterplot of standardized residuals, in which scores should be randomly scattered [26]. The data were reasonably linear, however, perfect linearity was not expected given the nature of this population, where participants tend to score closer to 0.

Aiken and West (1991), suggest that when analyzing interaction terms, all predictor variables should be centered [27]. Therefore, SA, parental descriptive norms, and parental injunctive norms were centered by subtracting participant's mean scores from the total predictor mean. The interaction terms were computed by multiplying the centered means with one another. For example, centered SA was multiplied by centered parental descriptive norms to generate the interaction term.

Analytic Overview

A multiple regression analysis was used to test parental injunctive norms, parental descriptive norms, and gender as moderators of the association between SA and cannabis problems. Descriptive statistics and bivariate correlations of each variable of interest are presented in Table 1. Separate models were run for each of the two hypothesized moderators: parental descriptive norms and parental injunctive norms. In the first model, cannabis problems were regressed on the first-order effects of SA, parental descriptive norms and gender, all two-way interaction terms, and the three-way interaction term of interest (i.e., $SA \times gender \times descriptive\ norms$). If any of the interaction terms were statistically significant ($p < .05$), they were followed up with simple slopes analyses, where the model was conditioned on high (+1 SD) and low (-1 SD) levels of parental descriptive norms (i.e., high perceived parental cannabis use, low perceived parental cannabis use) and conditioned on gender (men, women).

Table 1*Correlations, Means, and Standard Deviations for all Study Variables.*

Variables	1	2	3	4	5
1. Social Anxiety (SA)	—				
2. Parental Descriptive Norms	-.047	—			
3. Parental Injunctive Norms	-.062	.550**	—		
4. Gender (0=Men, 1=Women)	.113	-.039	-.055	—	
5. Cannabis Problems	.061	.093	.203*	-.269**	—
	<i>M</i>	59.181	1.181	49.558	83.8% women
	<i>SD</i>	27.314	4.052	24.591	15.623
					11.518

Note. $N = 154$. $*p < .05$. $^{**}p < .01$.

The second model followed the same analytic plan as the first model, except with parental injunctive norms as the moderator of interest (instead of parental descriptive norms). Again, if any of the interaction terms were statistically significant ($p < .05$), they were followed up with simple slopes analyses, where the model was conditioned on high (+1 SD) and low (-1 SD) levels of parental injunctive norms (i.e., high perceived parental approval, low perceived parental approval) and conditioned on gender (men, women).

Hypothesis Testing

SA predicting Cannabis use Problems

A simple linear regression was conducted to test levels of SA as a predictor of cannabis problems. The results suggest that the association between SA and cannabis problems was not statistically significant ($F(1,152) = .569, p = .452, R^2 = .004$).

Model 1: SA, Parental Descriptive Norms, and Gender on Cannabis use Problems. Results of the first model suggest that the SA-cannabis problems association was statistically significant ($F(7,146) = 5.178, p < .001, R^2 = .199$). The regression coefficients indicated that the two-way interactions between SA and parental descriptive norms, and parental descriptive norms and gender were not statistically significant. Furthermore, the three-way interaction between SA, parental descriptive norms, and gender was also not statistically significant. However, the two-way interaction between SA and gender was statistically significant. The results of the first model had an observed medium to large effect size ($f^2 = .25$) [28], and are presented in Table 2. Because the two-way interaction between SA and gender was statistically significant, simple slopes analyses were conducted to determine the direction of the association for men and women. The results of the simple slopes analyses are presented in Figure 1. For men, SA positively predicted an increase in cannabis problems. However, for women, the association between SA and cannabis problems was not statistically significant.

Table 2

Results of Multiple Regression Analysis of SA, Parental Descriptive Norms and Gender Predicting Cannabis Problems

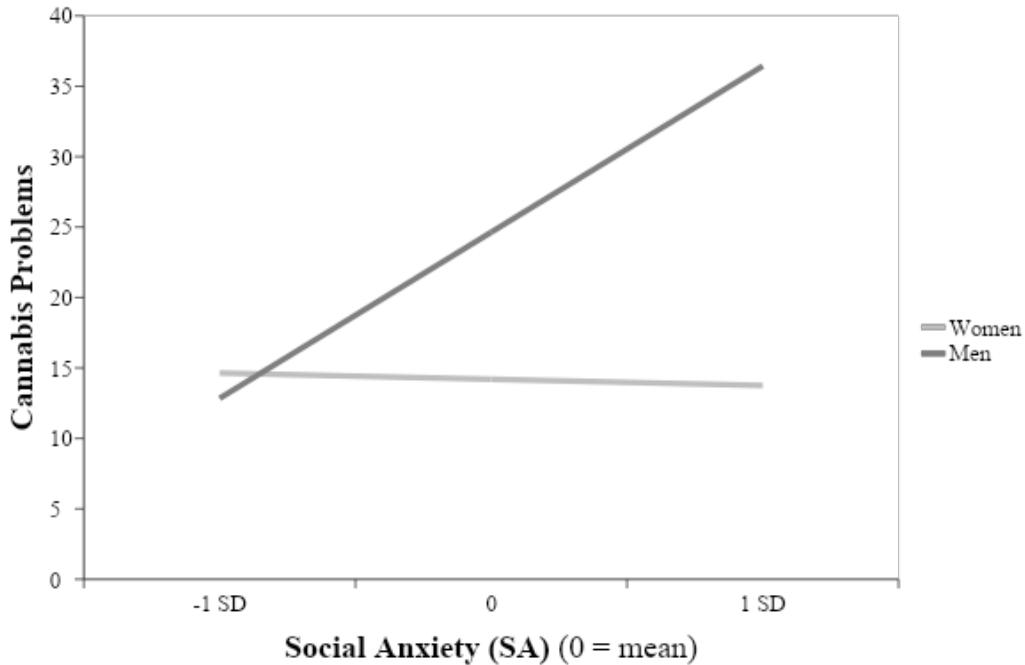
Predictor	Unstandardized coefficients				
	B	SE	B	t(151)	p
Social Anxiety (SA)	.432	.120	1.026	11.152	< .001*
Parental Descriptive Norms	-.719	.524	-.253	-1.372	.172
Gender (0=Men, 1=Women)	-10.431	2.398	-.335	-4.350	< .001*
SA x Parental Descriptive Norms	.100	.081	.802	2.238	.218
SA x Gender	-.448	.125	-.961	-3.586	< .001*
Parental Descriptive Norms x Gender	1.019	.579	.312	1.760	.080
SA x Parental Descriptive Norms x Gender	-.113	.082	-.887	-1.390	.167
Constant	24.629	2.209	—	11.152	< .001*

Note. N = 154, R = .446, R² = .199, Adjusted R² = .160.

*p < .05.

Figure 1

Simple slopes analysis for social anxiety predicting cannabis problems as moderated by gender



Model 2: SA, Parental Injunctive Norms, and Gender on Cannabis use Problems. Results of the second model suggest that the SA-cannabis problems association was statistically significant ($F(7,146) = 6.423, p < .001, R^2 = .235$). The regression coefficients suggest that the two-way interaction between gender and parental injunctive norms was not statistically significant. However, the

two-way interaction between SA and parental injunctive norms, the two-way interaction between SA and gender, and the three-way interaction between SA, parental injunctive norms, and gender were all statistically significant. The results of the second model had an observed medium to large effect size ($f^2 = .31$) [28], and are presented in Table 3. Because the three-way interaction was significant, this was the only interaction that was further analysed with simple slopes, in order to understand the direction of the association for men and women. The model was conditioned on high and low levels of parental injunctive norms (i.e., high perceived parental approval, low perceived parental approval) and conditioned on gender (men, women). Perceiving high approval versus perceiving low approval was defined as data falling at least one SD above or below the mean. The results of the simple slopes analyses are presented in Figure 2 for men and in Figure 3 for women. The simple slopes analyses suggest that high SA positively predicted cannabis problems among men high in parental injunctive norms. High SA did not predict cannabis problems for men low in parental injunctive norms, or for women high or low in parental injunctive norms.

Table 3

Results of Multiple Regression Analysis of SA, Parental Injunctive Norms and Gender Predicting Cannabis Problems

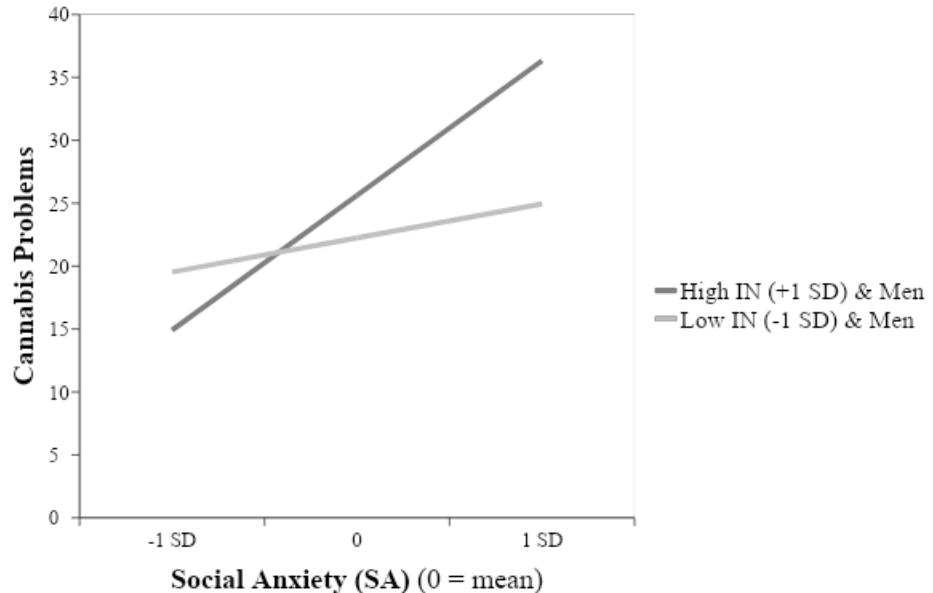
Predictor	Unstandardized coefficients				
	B	SE	B	t(151)	p
Social Anxiety (SA)	.246	.077	.583	3.181	.002*
Parental Injunctive Norms	.069	.089	.147	.770	.443
Gender (0=Men, 1=Women)	-9.615	2.340	-.309	-4.108	< .001*
SA x Parental Injunctive Norms	.006	.002	.380	2.615	.010*
SA x Gender	-.257	.084	-.552	-3.052	.003*
Parental Injunctive Norms x Gender	.002	.097	.004	.022	.982
SA x Parental Injunctive Norms x Gender	-.006	.003	-.316	-2.237	.027*
Constant	21.931	2.155	—	11.104	< .001*

Note. N = 154, R = .485, R² = .235, Adjusted R² = .199.

*p < .05.

Figure 2

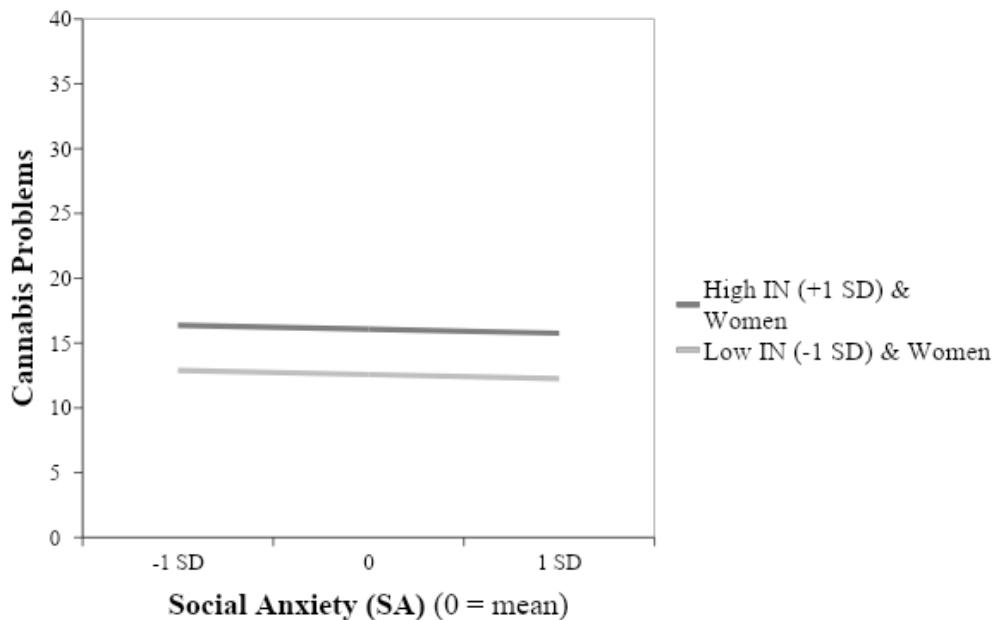
Simple slopes analysis: parental injunctive norms, selected for men



Note. Simple slopes analysis for social anxiety predicting cannabis problems, as moderated by high (+1 SD) and low (-1 SD) parental injunctive norms

Figure 3

Simple slopes analysis: parental injunctive norms, selected for women



Note. Simple slopes analysis for social anxiety predicting cannabis problems, as moderated by high (+1 SD) and low (-1 SD) parental injunctive norms

Discussion

The goal of the current study was to clarify the SA-cannabis problems risk trajectory by considering the roles of parental injunctive and descriptive norms, and gender in the association. The main findings indicate that gender and parental injunctive norms, in particular, influence the association between SA and cannabis problems. Specifically, increased SA predicts cannabis problems among men who perceive high parental approval of cannabis use.

In the current study, elevated SA did not predict an increase in cannabis problems. These findings highlight the inconsistent theoretical and empirical support for SA as a risk or protective factor for cannabis problems. Buckner and colleagues (2008) suggested that increased levels of SA predicted an increase in cannabis problems [6], whereas, Nelemans and colleagues (2016) suggested that increased levels of SA predicted a decrease in cannabis problems [11]. The inconsistent results in the literature, and the current studies' findings emphasize the importance of including moderators that impact the SA-cannabis problems association.

Contrary to our second hypothesis, gender and parental descriptive norms did not moderate the association between SA and cannabis problems. These findings are possibly due to the role of coping and conformity motives in the association. Previous research suggests that young adults with SA use cannabis to conform with their peers [17] and to cope with stressful social situations [5]. Thus, perceptions of parental cannabis use may not impact their cannabis use behaviours in stressful social situations. Another possibility is that young adults do not know about their parents' cannabis use. Cannabis was legalized in Canada in 2018, which was not very long ago [29]. Therefore, cannabis use may still be seen as taboo and restricted among parents. Accordingly, if parents use cannabis, or plan on starting to use cannabis, their children may not yet be aware. Future research should assess parental cannabis use when young adults today become parents themselves, as children in the future may be more aware of their parents' cannabis use.

Although gender and descriptive norms did not moderate the SA-cannabis problems association, gender alone did moderator the association. Specifically, elevated SA predicted a tendency to be higher in cannabis problems among men in general. These results are in line with previous research suggesting that SA is associated with cannabis problems, particularly among men [19, 30]. Indeed, Buckner and colleagues (2012) found that conformity and coping motives partially explained the association between SA and cannabis problems only among men. Their results suggested that men are more susceptible to using cannabis to avoid scrutiny from their cannabis-using peers than women are, and therefore, are more likely than women to develop cannabis problems [30]. Furthermore, a recent Statistics Canada report found that 21% of men, as compared to 12% of women have consumed cannabis [31]. Therefore, it is likely that men with elevated SA would feel more compelled than women with elevated SA to use cannabis to conform with their cannabis-using male peers.

Consistent with our third hypothesis, the SA and cannabis problems association was moderated by gender and parental injunctive norms, such that elevated SA was associated with increased risk for cannabis problems among men who perceive high parental cannabis approval. These results are in line with previous research investigating the role of parental injunctive norms in the SA-cannabis problems association. Ecker and Buckner (2014) investigated the role of parental injunctive norms in the SA-cannabis problems association and found that higher levels of SA predicted elevated cannabis problems among those who perceived higher parental approval of

cannabis behaviours [17]. The current study adds to the body of literature by considering how both parental injunctive norms and gender impact the SA-cannabis problems association. Ultimately, higher levels of SA predicted higher levels of cannabis problems, particularly among men who perceive high parental approval of cannabis. Inconsistent with the buffer perspective, the current study suggests that elevated SA predicted an increase in cannabis problems. Indeed, individuals high in SA who may be less involved with their peers, may consequently spend more time at home and be more involved with their parents. Therefore, individuals with elevated SA may still be at risk of developing cannabis problems if parents approve of cannabis use behaviours. Moreover, given that men are more fearful of scrutiny from their peers [30], approval from their parents regarding cannabis may lead to a greater desire to use cannabis during stressful social situations.

Despite the strengths of this study, such as including both gender and parental norms in the investigation of the association between SA and cannabis problems, this study has several noteworthy limitations. First, because this study was conducted during the COVID-19 pandemic, individuals with increased SA may not have been experiencing as many stressful social situations as they typically do, because of the restrictions on social events. Therefore, participants' reports of their SA may not have been entirely typical. Similarly, individuals may not have been consuming the same amount of cannabis (whether it be more or less than normal) and therefore, their cannabis use estimates may not have been completely characteristic. Although empirical investigation during the COVID-19 pandemic is a limitation, it can also be considered a strength, as it sheds light on SA and cannabis use during an unprecedented global pandemic. Future research should investigate whether an individual's levels of SA following the pandemic predicts an increase or decrease in cannabis problems, when individuals are transitioning back into normal, pre-pandemic, social situations. Second, the study was conducted primarily among anglophone women participants, and thus the results cannot be adequately generalized to Quebec's young adult population. Further research should be conducted with an adequate size of men and women to ensure that the sample is sufficiently powered and with French-translated questionnaires to include the large population of francophone young adults in Quebec. Third, the questionnaires used in this study rely on self-report responses from the participants, and thus, scores may not be as accurate as they would be if assessed by a professional. Future research would benefit from conducting a similar study in which SA and cannabis problems are assessed using semi-structured interviews conducted by trained professionals. Finally, future research should be conducted using a longitudinal design to assess the SA-cannabis use association, moderated by parental norms and gender, and see if the pattern of results remains stable or changes across the lifespan and as cannabis, which is now a legal drug, becomes less novel.

In conclusion, the current study aimed to assess the moderating role of gender and parental norms on the association between SA and cannabis problems. The results suggest that men who are high in SA tend to experience more cannabis problems, compared to women. Furthermore, men with elevated SA who perceive high parental approval of cannabis use tend to experience more cannabis problems than men with elevated SA who perceive low parental approval, or women with elevated SA regardless of their perceptions of parental approval. The results of the current study extend our understanding of the role of gender and parental approval on the SA-cannabis problems risk pathway. This study could help inform prevention and treatment strategies, given the important role that parents' approval of cannabis use behaviours has on their children's cannabis use. Therefore, prevention and treatment should include parents, especially for individuals with elevated SA at risk for cannabis problems.

References

- [1] Government of Canada (2018) Percentage of Canadians who used select illicit drugs in the past year as of 2017, by age. Statista.
- [2] Rotermann, M., & Macdonald, R. (2018). Analysis of trends in the prevalence of cannabis use in canada, 1985 to 2015 (Ser. Health reports). Statistics Canada.
- [3] Hall, W. (2009). The adverse health effects of cannabis use: what are they, and what are their implications for policy? *International Journal of Drug Policy*, 20(6), 458–466. <https://doi.org/10.1016/j.drugpo.2009.02.013>
- [4] Cheung, J. T. W., Mann, R. E., Ialomiteanu, A., Stoduto, G., Chan, V., Ala-Leppilampi, K., & Rehm Jurgen. (2010). Anxiety and mood disorders and cannabis use. *The American Journal of Drug and Alcohol Abuse*, 36(2), 118–122. <https://doi.org/10.3109/00952991003713784>
- [5] Buckner, J. D., Bonn-Miller, M. O., Zvolensky, M. J., & Schmidt, N. B. (2007). Marijuana use motives and social anxiety among marijuana-using young adults. *Addictive Behaviors*, 32(10), 2238–2252. <https://doi.org/10.1016/j.addbeh.2007.04.004>
- [6] Buckner, J. D., & Schmidt, N. B. (2008). Marijuana effect expectancies: relations to social anxiety and marijuana use problems. *Addictive Behaviors*, 33(11), 1477–1483. <https://doi.org/10.1016/j.addbeh.2008.06.017>
- [7] American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). Washington, DC: Publisher.
- [8] Stein, M. B., & Stein, D. J. (2008). Social anxiety disorder. *The Lancet*, 371(9618), 1115–1125. [https://doi.org/10.1016/S0140-6736\(08\)60488-2](https://doi.org/10.1016/S0140-6736(08)60488-2)
- [9] Weymouth, B. B., Fosco, G. M., & Feinberg, M. E. (2019). Nurturant-involved parenting and adolescent substance use: examining an internalizing pathway through adolescent social anxiety symptoms and substance refusal efficacy. *Development and Psychopathology*, 31(1), 247–260. <https://doi.org/10.1017/S0954579417001766>
- [10] Villarosa-Hurlocker, M. C., Bravo, A. J., & Pearson, M. R. (2019). The relationship between social anxiety and alcohol and marijuana use outcomes among concurrent users: a motivational model of substance use. *Alcoholism: Clinical and Experimental Research*, 43(4), 732–740. <https://doi.org/10.1111/acer.13966>
- [11] Nelemans, S. A., Hale, W. W., Raaijmakers, Q. A. W., Branje, S. J. T., van Lier, P. A. C., & Meeus, W. H. J. (2016). Longitudinal associations between social anxiety symptoms and cannabis use throughout adolescence : the role of peer involvement. *European Child & Adolescent Psychiatry*, 25(5).
- [12] Azjen, I. (1991). The theory of planned behavior. *Organizational Behavior and Human Decision Processes*, 50, 179 –211.
- [13] Buckner, J. D. (2013). College cannabis use: the unique roles of social norms, motives, and

expectancies. *Journal of Studies on Alcohol and Drugs*, 74(5), 720–6.

[14] Ecker, A. H., Richter, A. A., & Buckner, J. D. (2014). Cannabis-related impairment: the impacts of social anxiety and misconceptions of friends' cannabis-related problems. *Addictive Behaviors*, 39(12), 1746–1749.

[15] Neighbors, C., Geisner, I. M., & Lee, C. M. (2008). Perceived marijuana norms and social expectancies among entering college student marijuana users. *Psychology of addictive behaviors : journal of the Society of Psychologists in Addictive Behaviors*, 22(3), 433–438. <https://doi.org/10.1037/0893-164X.22.3.433>

[16] Foster, D. W., Garey, L., Buckner, J. D., & Zvolensky, M. J. (2016). Social anxiety and cannabis-related impairment: the synergistic influences of peer and parent descriptive and injunctive normative perceptions. *Substance Use & Misuse*, 51(7), 912–921. <https://doi.org/10.3109/10826084.2016.1156701>

[17] Ecker, A. H., & Buckner, J. D. (2014). Cannabis use behaviors and social anxiety: The roles of perceived descriptive and injunctive peer norms. *Journal of Studies on Alcohol and Drugs*, 75(1), 74–82.

[18] Buckner, J. D., Mallott, M. A., Schmidt, N. B., & Taylor, J. (2006). Peer influence and gender differences in problematic cannabis use among individuals with social anxiety. *Journal of anxiety disorders*, 20(8), 1087–1102. <https://doi.org/10.1016/j.janxdis.2006.03.002>

[19] Buckner, J. D., Heimberg, R. G., & Schmidt, N. B. (2011). Social anxiety and marijuana-related problems: The role of social avoidance. *Addictive Behaviors*, 36, 129–132.

[20] Liebowitz, M. R. (1987). Social phobia. *Modern Problems of Pharmacopsychiatry*, 22, 141-173.

[21] Heimberg, R. G., Horner, K. J., Juster, H. R., Safren, S. A., Brown, E. J., Schneier, F. R., & Liebowitz, M. R. (1999). Psychometric properties of the liebowitz social anxiety scale. *Psychological Medicine*, 29(1), 199–212.

[22] Baer J. S., Stacy A., & Larimer M (1991). Biases in the perception of drinking norms among college students. *Journal of Studies on Alcohol*, 52(6), 580-586.

[23] Broadwater, K., Curtin, L., Martz, D. M., & Zrull, M. C. (2006). College student drinking: perception of the norm and behavioral intentions. *Addictive Behaviors*, 31(4), 632–640. <https://doi.org/10.1016/j.addbeh.2005.05.041>

[24] Hines, S. A., & O'Connor, R. M. (2018, November). The Perceived Approval of Risky Drinking Inventory: Empirical development of an injunctive drinking norms measure. Presented at the symposium on "Advances in the use of social norms in risky drinking research: From conceptualization to intervention" (Co-Chairs: Sarah A. Hines & Roisin M. O'Connor) at the 52nd Annual Convention of the Association for Behavioral and Cognitive Therapies (ABCT), Washington, DC.

[25] Bashford, J., Flett, R., & Copeland, J. (2010). The Cannabis Use Problems Identification Test (CUPIT): Development, reliability, concurrent and predictive validity among adolescents and adults. *Addiction*, 105(4), 615-625. <http://dx.doi.org/10.1111/j.1360-0443.2009.02859.x>

- [26] Field, A. P. (2009). Discovering statistics using SPSS. Sage Publications.
- [27] Aiken, L., & West, S. (1991). Multiple Regression: Testing and interpreting interactions. Sage Publications.
- [28] Cohen, J. (1988). Statistical power analysis for the behavioral sciences. 2. Lawrence Erlbaum Associates.
- [29] Government of Canada (2021). Cannabis legalization and regulation. Retrieved March 13, 2021, from <https://www.justice.gc.ca/eng/cj-jp/cannabis/>
- [30] Buckner, J. D., Zvolensky, M. J., & Schmidt, N. B. (2012). Cannabis-related impairment and social anxiety: the roles of gender and cannabis use motives. *Addictive Behaviors*, 37(11), 1294–1297. <https://doi.org/10.1016/j.addbeh.2012.06.013>
- [31] Statistics Canada (2019). National Cannabis Survey, second quarter 2019. Retrieved from <https://www150.statcan.gc.ca/n1/daily-quotidien/190815/dq190815a-eng.htm>
- [32] Davis, J. P., Christie, N. C., Pakdaman, S., Hummer, J. F., DeLeon, J., Clapp, J. D., & Pedersen, E. R. (2020). Multifaceted impulsivity as a moderator of social anxiety and cannabis use during pregaming. *Journal of Anxiety Disorders*, 76, 102320–102320. <https://doi.org/10.1016/j.janxdis.2020.102320>

Social Anxiety and Coping Motivated Cannabis Use: The Moderating Effect of Negative Urgency *

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According to self-medication hypothesis, those who score high in social anxiety (SA) may use cannabis/marijuana for its anxiolytic effects (i.e., to cope with negative affect). Coping-motivated substance use has been linked to an increased risk for negative consequences such as sensations of paranoia. However, the SA-cannabis coping motive association is unclear, with mixed empirical support. Negative urgency (NU; the tendency to act impulsively when distressed) has been linked with substance misuse and may moderate this association. The current study tested NU as a moderator of the SA-cannabis coping motives relation and examined the correlation between cannabis coping motives and negative consequences. Psychology undergraduates ($N=241$) completed online self-reports of SA, NU, and cannabis use motives and negative consequences of cannabis use. Results of multiple regression analyses revealed support for a first order effect of NU only, such that elevated NU was associated with increased cannabis coping motives. Moderation of NU on cannabis use for the relief of SA was not supported. Cannabis coping motives was supported as a positive correlate of negative consequences. Findings may inform future interventions by highlighting motivation for use as an individual-level risk factor for negative consequences and NU as a risk factor for coping motives.

In 2019, more than 5.1 million Canadian's reported using cannabis. Of these, 33.3% were young adults aged 18 to 24 years that reported consuming cannabis within the last 3 months [1]. Further, approximately 6% of Canadians aged 15 years or older reported using cannabis daily. Daily cannabis consumption is associated with negative health outcomes including lung damage, cognitive impairment, altered brain development, and worsened mental health [1-3]. Coping motivated cannabis use as a primary reason for use – i.e., use of cannabis to regulate or reduce negative affect – has been associated with negative consequences, such as dependence and increased sensations of anxiety [4-7]. One population that may use cannabis to cope is young adults with social anxiety (SA) [8].

Undergraduates report using substances and known to experience SA. SA is characterized by marked fear of being negatively evaluated by others in social situations [9]. Strahan reported 22% of 248 undergraduates demonstrated clinical levels of SA [10]. Undergraduate students are also known users of cannabis [11]. A large survey from 2019, based on a sample of 58 Canadian post-secondary institutions, found that almost half (44.4%) of college students had used cannabis during their lifetime, and 20.9% of students reported using cannabis within the last 30 days [12]. Substance use disorder involves heavy or frequent substance use. The comorbidity of substance use disorders and anxiety disorders is well established in the literature [13]. The National Epidemiological Survey on Alcohol and Related Conditions (NESARC) revealed that 17.7% of respondents with diagnosed substance use disorder also met criteria for an anxiety disorder [14]. Researchers have also found that elevated SA is associated with problematic and elevated cannabis use [15-]

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18]. In one sample of 8098 American adults, for example, individuals with SA were 7 times more likely to experience marijuana related impairment (e.g., cognitive impairments) and dependence when compared to the general population of marijuana users [19]. Therefore, marijuana related impairment and dependence has a lifetime prevalence of approximately 4.2% in those with SA [19].

An individual's motives for cannabis use are based on expectancies for the effects of use, previous experiences with cannabis, and situational variables [8]. According to stress-coping models of addiction, individuals consume substances to cope with stress, reduce levels of negative affect, and/or increase levels of positive affect [20]. Similarly, the self-medication hypothesis states that substance use and dependence is a way to "self-soothe" by modulating affect and reducing negative feelings, which may stem from distressing psychological states [21]. On the basis of this theory, individuals with SA may use cannabis to reduce the negative affect experienced as a result of their SA symptoms (such as fear of judgement). Cannabis indeed is used in a wide variety of settings for its anxiolytic properties. Some individuals even use cannabis as a sleep aid, while others use cannabis as a medicinal drug, listing anxiety or depression as their reason for use [6, 22]. While cannabis does indeed have anxiety reducing properties [8], it also is associated with increased anxiety [5].

Those who list "coping with negative affect" as a primary reason for their cannabis use are found to be at highest risk for negative consequences of cannabis use, such as dependence [4]. Other negative consequences include: psychological distress; panic attacks; increased paranoia; increased sensations of anxiety; inter- and intra-personal conflict; and risk-taking behaviours [6,7]. According to stress-coping models of addiction, individuals use substances such as cannabis to reduce their negative affect, and would therefore, theoretically, be deterred from using substances that would induce or intensify such negative affect. In one sample of cannabis dependent participants, those who were aware of and expected the negative consequences associated with cannabis use were less likely to use than those who had positive expectations [23]. Despite the potential negative consequences associated with cannabis use, some individuals with SA continue to consume the drug to cope; thus, they discount the potential risks. SA is therefore a significant predictor of coping motives for marijuana use as opposed to the many other possible motives for use (such as enjoyment, conformity, experimentation). That is, individuals with high levels of SA are more likely to report coping with negative affect as a main reason for cannabis use as opposed to other reasons [8]. Although the underlying moderators linking SA to coping motivated marijuana use are complex, mixed, and not entirely understood, one possible moderator may involve impulsivity: more specifically, negative urgency (NU).

Individuals that are impulsive and emotionally driven, are more likely to engage in risk-taking behaviors, including problematic substance use, when they experience extremely positive or negative emotions [24]. NU is a trait tendency for individuals to act impulsively when experiencing strong negative emotions. It has been theorised that certain individuals impulsively use substances, such as cannabis, to cope with their strong emotions [25]. For example, individuals with diagnosed substance use disorders have been found to be more likely to score high in NU as opposed to other measures of impulsivity such as sensation seeking [26]. Another study found that NU was a positive predictor of problems related to cannabis use and dependence [26]. Individuals with SA who demonstrate high NU may engage in rash judgement when emotionally distressed, and thus may be at risk for using cannabis to cope. This coping motivated cannabis use is maladaptive because it may contribute to the negative affect and anxiety that users are trying

to alleviate in the first place. Conversely, individuals with SA who are low in NU may be more likely to consider the negative consequences associated with cannabis use and may be deterred from use.

The goal of the current study was to test NU as a moderator of the relation between SA and use of cannabis for coping motives in an undergraduate sample. Two separate models were run with SA-specific cannabis coping motives as the outcome in one model, and broad cannabis coping motives in another model. The association between coping motives and negative consequences of cannabis use was also investigated. It was hypothesized that elevated SA would be associated with increased use of cannabis for coping motives, including use to cope specifically with SA, but that this effect would only be evident for those high on NU. It was also hypothesized that cannabis coping motives (including SA coping motives) would be positively correlated with negative consequences of cannabis use.

Method

Participants

A total of 277 undergraduates living in the Montreal, QC area took part in the study, however, only those with data on the current study measures were retained for analyses. The final sample included 241 undergraduate students fluent in English and between the ages of 18–25 years ($M = 21.19$; $SD = 1.71$). Both individuals who consumed and did not consume cannabis were eligible to participate. The current study obtained ethical approval from Concordia's Ethical Research Board.

Procedure

Data for the current study were drawn from a larger online study investigating beliefs about marijuana use. Participants from this study were recruited through the Concordia Psychology Participant Pool, and flyers advertising the study were posted on Concordia's Undergraduate Psychology Association's (CUPA's) Facebook page. Individuals eligible for the Concordia Psychology Participant Pool were compensated with course credit; and those who were not eligible for course credit were entered into a draw for one of two \$50 cash prizes. Data collection began in June 2020 and is ongoing.

Measures

Participants were asked to complete a battery of self-report questionnaires; that took approximately 1 hour. The questionnaires were hosted on Checkbox Survey.

Marijuana Consequences Questionnaire (MACQ). The MACQ was used to assess cannabis-related negative consequences [29]. This 50 item self-report measure asks participants to respond yes (scored as 1) or no (scored as 0) to a series of statements pertaining to marijuana related problems and consequences, which occurred in the last 6 months (e.g., "I have passed out from marijuana use"). Marijuana related problems include risk taking behaviors, intra- and inter- personal problems, signs of physical dependence, and difficulty controlling use. The derived final score for each participant was a sum of yes responses. The Cronbach alpha coefficient in our sample was .93.

Comprehensive Marijuana Motives Questionnaire (CMMQ). The CMMQ is made up of 36 items and 8 subscales [5]. Each item describes different reasons to use marijuana. The coping

subscale (3 items; e.g., To forget your problems) and the SA-specific coping subscale (3 items; e.g., "Because it makes you more comfortable in an unfamiliar situation") were used to assess coping motives for cannabis use. The participants were asked to rate each item on a 5-point scale, ranging from almost never/never (scored as 1) to almost always/always (scored as 5), in terms of how often the item was their reason to use marijuana. The derived final scores for each subscale were summed scores of their responses. The Cronbach alpha coefficients in our sample were .92 for the both the coping subscale, and the SA subscale.

Liebowitz Social Anxiety Scale (LSAS). SA was assessed using the LSAS [30]. Each of the 24 items on the self-report questionnaire describes a different social situation (e.g., "Telephoning in public"). Participants were asked to rate each item on two different 4-point scales. On the first scale, participants indicated their level of fear or anxiety towards the situation ranging from none (scored as 0) to severe (scored as 3). Next, the participants were asked to indicate their level of avoidance for that same situation, ranging from never 0% (scored as 0) to usually 67-100% (scored as 3). The derived global score for each participant was a summed score of their responses. The Cronbach alpha coefficient in our sample was .96.

Urgency, Premeditation, Perseverance and Sensation Seeking Impulsive Behaviour Scale (UPPS-P). NU was measured using the NU subscale on the UPPS-P [31]. The UPPS-P consists of 59 items while the NU subscale is made up of 12 items. Each item describes a different impulsive behaviour or belief (e.g., "I often make matters worse because I act without thinking when I am upset"). Participants were asked to rate each item on a 4-point scale ranging from agree strongly (scored as 1) to disagree strongly (scored as 4). The derived final scores for each subscale were summed scores of their responses. The Cronbach alpha coefficient in our sample was .89.

Data Analysis

All statistical analyses were conducted using IBM SPSS Statistics 27.0 software. The statistical level at which we determined significance was $p < .05$. Effect sizes, as per Cohen (1992), were small at $R^2 = .02$, medium at $R^2 = .13$, and large at $R^2 = .26$ [32]. A multiple linear regression analysis was used to test the moderation hypothesis. Two separate models with SA-specific cannabis coping motives as the outcome in one model, and broad cannabis coping motives in another model were run. Coping motives were regressed on the first order effects of SA and NU, and the 2-way interaction between SA and NU. The predictor variable (SA) and moderator (NU) were centered to reduce multicollinearity [28]. Additionally, to test the relation between cannabis coping motives and the negative consequences of cannabis use, a Pearson correlational analysis was conducted. The strength and direction of the Pearson correlation was observed, and a 95% confidence interval was used.

The rate of missing data for the current sample was 14.94%. Those with complete data ($N = 241$) did not significantly differ from those with incomplete data ($n = 36$) in terms of age, $t(275) = 0.96$, $p = .34$ or gender $X^2 (3, n = 275) = 3.703$, $p = .30$. A single participant did not complete one response for one item on the LSAS and so that data point was imputed and replaced with that participant's mean score on that measure.

Results

Descriptive Statistic

At baseline, 202 (83.8%) participants identified as women, 31 (12.9%) as men, and 6 (2.5%) as

non-binary. The majority of the sample (63.1%) identified as Caucasian. Descriptive statistics are presented in Table 1. Our sample's mean scores of for NU ($M = 27.01$) and SA ($M = 60.63$) were considered to be "average" and in-line with the samples these measures were normed on [30-31]. Our sample's mean scores of coping motives ($M = 5.28$), SA coping motives ($M = 4.76$), and negative consequences ($M = 5.35$) were considered to be low [5,29].

Table 1

Correlations, Means, and Standard Deviations for All Analyses

Variables	1	2	3	4	5
1. Coping motives	—				
2. SA coping motives	.480	—			
3. Negative Urgency	.328	.220	—		
4. Social Anxiety	.138	.100	.273	—	
5. Negative Consequences	.600	.460	.316	.011	—
<i>M</i>	5.278	4.764	27.008	60.629	5.349
<i>SD</i>	3.152	3.102	7.467	27.865	6.985

Note. $N = 241$; SA = Social Anxiety; NU = Negative Urgency; M = Mean; SD = Standard Deviation.

Data Integrity

We tested the assumptions for multiple linear regression. The data set did not include multivariate outliers, as defined by z-scores exceeding $|3.29|$ standard deviations beyond the mean [33]. The assumption of linearity was met, which was assessed by visually inspecting partial regression plots and a plot of studentized residuals against the predicted values. Furthermore, the residuals were independent, as assessed by Durbin-Watson statistics of 2.008 (for the coping motives model) and 1.951 (for the SA coping motives model). Additionally, there was no evidence of multicollinearity. That is, bivariate correlations between variables were less than 0.9, tolerance values were higher than 0.1, and the conditioning indexes were less than 30, with two variance proportions less than .5 [33]. The assumption of homoscedasticity was also not met, as assessed by visual inspection of a plot of studentized residuals versus unstandardized predicted values. The data were normally distributed for SA and NU but were not for coping motives or for SA coping motives, as assessed by a Q-Q Plot. Coping motives and SA coping motives were right skewed and a floor effect was observed. No transformations were performed on the data. Given that the skewed variables were outcome variables and there were no issues with the predictor variables, we chose to continue with the analysis despite some assumptions being violated. We must therefore interpret findings with caution.

The assumptions for Pearson correlations were also tested. There were linear relationships between the variables and there were no outliers. The data were not normally distributed for coping

motives, for SA coping motives, or for negative consequences, as assessed by a Q-Q Plot. Variables were right skewed and a floor effect was observed in each one. We chose to move forward with the analysis in their original units of measurement despite some assumptions being violated. Therefore, caution must be taken when interpreting these findings.

Multiple Linear Regression Analysis of Predictors of Coping Motives

A multiple linear regression was run to predict cannabis coping motives. Entered into the model was the predictor variables NU, SA, and the interaction term NU \times SA. Results are presented in Table 2. The model significantly predicted cannabis coping motives, $F(3, 237) = 10.00, p < .001$. Further, 11.2% of the variation in cannabis coping motives was explained by the variation in the predictors, SA and NU, $R^2 = .11, 95\% \text{ CI } [.04 - .18]$. This effect size was considered to be small. The first order effect of SA ($p = .37$) and the interaction term ($p = .049$) on coping motives were not statistically significant. However, the first-order effect of NU emerged as a statistically significant predictor of cannabis coping motives. An increase by one unit in NU resulted in a .13 unit increase in cannabis coping motives ($p < .001$). As the interaction term's regression coefficient was below our predetermined cut-off of $p < .05$, we did not continue with simple slopes analysis and did not condition the model at high and low NU.

Table 2

Results of Multiple Linear Regression Analysis of Predictors of Coping Motives

Predictor	<i>B</i>	<i>SE B</i>	<u>95% CI for B</u>		β	<i>t</i> (238)	<i>p</i>
			<i>LL</i>	<i>UL</i>			
Constant	5.24*	.199	4.848	5.631		26.372	.000
NU	.129*	.027	.076	.183	.306	4.749	.000
SA	.006	.007	-.008	.021	.057	.892	.373
NU \times SA	.001	.001	-.001	.002	.049	.782	.435

Note. $N = 241; R^2 = .112, 95\% \text{ CI } [.042 - .182]; B = \text{unstandardized regression coefficient}; CI = \text{confidence interval}; LL = \text{lower limit}; UL = \text{upper limit}; SE B = \text{standard error of the coefficient}; \beta = \text{standard coefficient}$

* $p < .05$.

Multiple Linear Regression Analysis of Predictors of SA Coping Motives

A multiple linear regression was run to predict cannabis SA coping motives. Entered into the model was the predictor variables NU, SA, and the interaction term NU \times SA. Results are presented in Table 3. The model significantly predicted SA coping motives, $F(3, 237) = 4.24, p = .006$. Further, 5.1% of the variation in SA coping motives was explained by the variation in SA and NU, $R^2 = .051, 95\% \text{ CI } [.005 \text{ to } .11]$. This effect size was considered to be small.

Table 3*Results of Multiple Linear Regression Analysis of Predictors of SA Coping Motives*

Predictor	<i>B</i>	<i>SE B</i>	<u>95% CI for B</u>		β	<i>t</i> (238)	<i>p</i>
			<i>LL</i>	<i>UL</i>			
Constant	1.743*	.202	4.345	5.142		23.461	.000
NU	.085*	.028	.030	.139	.205	3.071	.002
SA	.005	.007	−.009	.020	.046	.691	.490
NU×SA	.000	.001	−.001	.002	.026	.402	.688

Note. $N = 241$; $R^2 = .051$, 95% CI [.005 – .105]; B = unstandardized regression coefficient; CI = confidence interval; LL = lower limit; UL = upper limit; $SE B$ = standard error of the coefficient; β = standard coefficient

* $p < .05$.

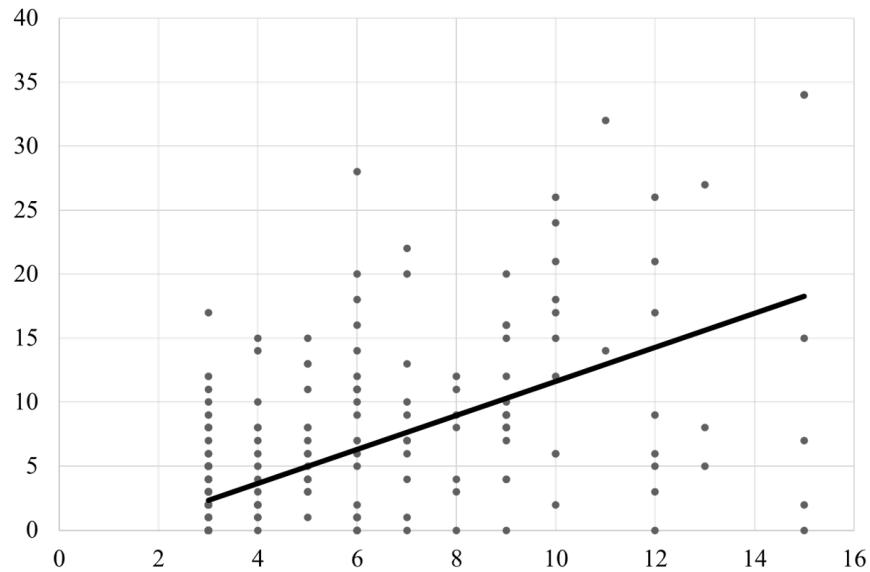
The first-order effect of SA ($p = .49$) and the interaction term ($p = .69$) were not statistically significant. However, the first-order effect of NU emerged as a significant predictor of SA cannabis coping motives. An increase by one unit in NU resulted in a .085 unit increase in SA cannabis coping motives ($p = .002$). As the interaction term's regression coefficient was below our predetermined cut-off of $p < .05$, we did not continue with simple slopes analysis and did not condition the model at high and low NU.

Correlations between Coping Motives and Negative Consequences

A Pearson correlation was run to examine the association between cannabis coping motives and the negative consequences of cannabis use. There was a significant positive correlation between the variables, $r(239) = .60$, $p < .001$, with cannabis coping motives explaining 36% of the variation in negative consequences of cannabis use. A scatterplot summarizes the results in Figure 1. Another Pearson correlation assessed the relation between cannabis SA coping motives and the negative consequences of cannabis use and there was a significant positive correlation between the variables, $r(239) = .46$, $p < .001$, with cannabis SA coping motives explaining 21.20% of the variation in negative consequences of cannabis use. A scatterplot summarizes the results in Figure 2.

Figure 1

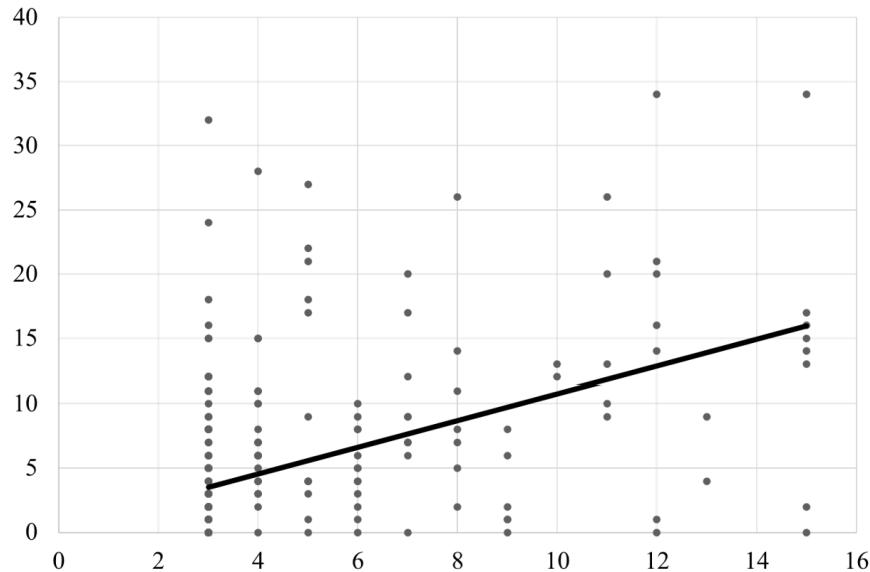
Scatterplot Depicting the Correlation between Coping Motives and Negative Consequences



Note. Scatterplot depicting association between total coping motives scores and total negative consequences scores; Pearson's $r(239) = .60$, $p < .001$

Figure 2

Scatterplot Depicting the Correlation between SA Cope Motives and Negative Consequences



Note. Scatterplot depicting association between total SA cope motives scores and total negative consequences scores; Pearson's $r(239) = .46$, $p < .001$

Discussion

Buckner and colleagues (2007) suggest that SA predicts coping motivated cannabis use [8]. Researchers have also reported that individuals who primarily use cannabis to cope are more likely to have negative consequences as a result of their cannabis use [4-7]. Despite the risk of these negative consequences, certain individuals with SA continue to use the substance. The mechanisms underlying this behaviour are not entirely understood, with mixed empirical support. Researchers have found that those high in NU are more likely to impulsively use substances to cope with their distress and may be discounting the potential negative consequences associated with their substance use [24-27]. We aimed to examine whether SA acts as a predictor in coping motivated cannabis use and whether NU was a moderator in this relation. We also investigated the relation between coping motivated cannabis use and the negative consequences associated with its use. SA was not found to significantly predict an individual's motivation to cope using cannabis nor predict an individual's motivation to cope using cannabis specifically for SA. There is mixed support for the link between SA and cannabis use in the literature. Previous literature suggests that those with SA are more likely to use cannabis to cope [8], however, our results did not support this association in undergraduate students. Those who use cannabis to cope are also more likely to experience the negative consequences associated with cannabis use [4]. While some individuals with SA might be deterred from coping motivated cannabis use when faced with potential negative consequences, others will continue to use cannabis to cope despite the risks.

NU significantly predicted both an individual's motivation to cope using cannabis, as well as an individual's motivation to cope using cannabis specifically for SA. This is in-line with what previous researchers have hypothesized, in that those high in NU may be impulsively using substances while they are distressed as a form of coping with negative emotions, such as fear of negative evaluation [24]. There was no observed interaction effect between SA and NU in predicting coping motives or SA coping motives in the regression models. Therefore, NU was not a significant moderator in the relation between SA and coping motives or SA and SA coping motives. Thus, suggesting there was no difference in the motivation to use cannabis to cope between anxious or non-anxious individuals, whether or not they were high or low in NU. Though this finding was not consistent with our hypothesis or expected results, there are several possible explanations for this outcome.

Typically, alcohol and drug expectancies can be dichotomized as positive or negative. Positive expectancies (e.g., I expect to feel joyful if I use cannabis) are thought to motivate substance use, while negative expectancies (e.g., I expect to feel embarrassed the day after using cannabis) may motivate individuals to restrict their substance use [34]. Previous research points to the role of positive marijuana expectancies when considering links between impulsivity (sensation seeking) and marijuana use disorder symptoms (such as urge to use) [35]. If individuals high in SA have negative expectancies towards cannabis use, they may not engage in coping motivated use. Those high in NU are also often impulsively engaging in poly-substance use (i.e., the use of multiple substances), such as alcohol or stimulants (e.g., Cocaine), which are influenced by different expectations [24]. That is, those high in SA and NU may not be drawn to cannabis, as they may not expect it to provide them with the social lubrication they desire or expect to get from other substances [5]. Future researchers may benefit from investigating more complex models that incorporate variables such as expectancies of use and poly-substance behaviours.

Lastly, consistent with our hypothesis and existent literature, cannabis coping motives as well

as SA cannabis coping motives were positively correlated with negative consequences associated with cannabis use. This means that the more an individual used cannabis to cope with negative affect, the more they experienced negative consequences associated with cannabis use (such as panic attacks). Although we cannot infer directionality or causation, one potential explanation for this may be that the individuals experiencing negative consequences may be led to use cannabis to cope because they lack alternative, more effective coping strategies or skills. Co-currently investigating other coping strategies of participants in regard to managing negative emotions may be beneficial. Alternatively, coping motivated use of cannabis may lead to increased frequency of use, which in itself can lead to negative consequences such as “passing out” or not remembering what happened while you were under the influence [4].

The results of the current study should be interpreted considering its limitations. First, convenience sampling was used. Participants were primarily recruited from Concordia University’s psychology participant pool, the majority being psychology students and therefore lacking generalizability to other students. The sample was also overwhelmingly WEIRD (western, educated, industrialized, rich, and democratic), female, and white. The findings of the study may not be generalizable to broader university populations and may not truly represent the cannabis coping motives of young adults more broadly. Future researchers may benefit from collecting data from more diverse samples, encouraging participation from more men and People of Colour especially. Second, this study was conducted during the COVID-19 global pandemic. There were decreased in-person social gatherings and interaction at varying points throughout data collection due to Montreal’s government enforced lockdown as a result of the pandemic. Thus, the experiences of SA of participants may have been impacted by these measures, as opportunities where participants may have felt socially anxious were somewhat limited. As such, the results from the present study may not be entirely representative of, or generalizable to, levels of SA and motivation for substance use pre- and post- pandemic. Conducting data collection post-COVID-19 may be beneficial. Comparisons of pre, during and post COVID-19 results could also be conducted to confirm if SA and substance use were, in fact, affected.

It should also be noted that the current study used a questionnaire to assess SA as opposed to participants reporting a diagnosis of Social Anxiety Disorder (SAD). This lack of formal diagnosis means results may not be reflective of those living with SAD. Lastly, the frequency of negative consequences and both coping motives and SA coping motives, had high rates of zero in the dataset. This made it more complicated to assess the association of our predictors to our outcomes. This may have been due to the fact we used a convenience sample of undergraduate students, and this population may, for example, experience fewer negative consequences as opposed to clinical populations. Future researchers may wish to perform statistical transformations or analyses better suited for such distributed data.

Despite the limitations of the current study, results still provide researchers insight into cannabis coping motives and the mechanisms, which underlie the associations between coping motives and NU. We found that NU may help predict coping motives and that coping motives and negative consequences were positively correlated with one another. Clinicians and intervention workers could potentially apply these results by screening undergraduates for their reasons for cannabis use and impulsivity. They could, for example, flag those who primarily use cannabis to cope as being at risk for experiencing negative consequences, and flag those with high scores in NU as at risk for using cannabis to cope. Such strategies could provide students with appropriate supports (e.g., counsellors) where they could learn alternative coping strategies, aimed at decreasing the

likelihood of experiencing negative consequences while using cannabis.

References

- [1] Rotermann, M. (2020). What has changed since cannabis was legalized? *Statistics Canada Health Reports*, 82(3). <https://www.doi.org/10.25318/82-003-x202000200002-eng>
- [2] Hall, W., & Degenhardt, L. (2009). Adverse health effects of non-medical cannabis use. *The Lancet*, 374(9698), 1383-1391. [https://doi.org/10.1016/S0140-6736\(09\)61037-0](https://doi.org/10.1016/S0140-6736(09)61037-0)
- [3] Volkow, N. D., Baler, R. D., Compton, W. M., & Weiss, S. R. (2014). Adverse health effects of marijuana use. *New England Journal of Medicine*, 370(23), 2219–2227. <https://doi.org/10.1056/NEJMra1402309>
- [4] Blevins, C. E., Banes, K. E., Stephens, R. S., Walker, D. D., & Roffman, R. A. (2016). Motives for marijuana use among heavy-using high school students: An analysis of structure and utility of the Comprehensive Marijuana Motives Questionnaire. *Addictive Behaviors*, 57, 42–47. <http://dx.doi.org/10.1016/j.addbeh.2016.02.005>
- [5] Lee, C. M., Neighbors, C., Hendershot, C. S., & Grossbard, J. R. (2009). Development and preliminary validation of a comprehensive marijuana motives questionnaire. *Journal of Studies on Alcohol and Drugs*, 70(2), 279–287. <https://doi.org/10.15288/jsad.2009.70.279>
- [6] Lee, C., Neighbors, C., & Woods, B. (2007). Marijuana motives: Young adults' reasons for using marijuana. *Addictive Behaviours*, 32(7), 1384–94. <https://doi.org/10.1016/j.addbeh.2006.09.010>
- [7] Simons, J., Gaher, R., Correia, C., Hansen, C., & Christopher, M. (2005). An affective-motivational model of marijuana and alcohol problems among college students. *Psychology of Addictive Behaviours*, 19(3), 326–34. <https://doi.org/10.1037/0893-164X.19.3.326>
- [8] Buckner, J., Bonn-Miller, M., Zvolensky, M., & Schmidt, N. (2007). Marijuana use motives and social anxiety among marijuana-using young adults. *Addictive Behaviors*, 32(10), 2238–2252. <https://doi.org/10.1016/j.addbeh.2007.04.004>
- [9] American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). American Psychiatric Pub.
- [10] Strahan, E. Y. (2003). The effects of social anxiety and social skills on academic performance. *Personality and Individual Differences*, 34(2), 347–366. [https://doi.org/10.1016/S0191-8869\(02\)00049-1](https://doi.org/10.1016/S0191-8869(02)00049-1)
- [11] Pinchevsky, G. M., Arria, A. M., Caldeira, K. M., Garnier-Dykstra, L. M., Vincent, K. B., & O'Grady, K. E. (2012). Marijuana exposure opportunity and initiation during college: parent and peer influences. *Prevention Science*, 13(1), 43–54. <https://doi.org/10.1007/s11121-011-0243-4>
- [12] American College Health Association. (2019). *National College Health Assessment II: Canadian reference group data report spring 2019*. ACHA.

- [13] Lai, H.M., Cleary, M., Sitharthan, T., & Hunt, G.E. (2015). Prevalence of comorbid substance use, anxiety and mood disorders in epidemiological surveys, 1990-2014: A systematic review and meta-analysis. *Drug and Alcohol Dependence*, 154, 1–13. <https://www.doi.org/10.1016/j.drugalcdep.2015.05.031>
- [14] Grant, B. F., Stinson, F. S., Dawson, D. A., Chou, S. P., Dufour, M. C., Compton, W. W., Pickering, R. P., & Kaplan, K. (2004). Prevalence and co-occurrence of substance use disorders and independent mood and anxiety disorders: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Archives of General Psychiatry*, 61(8), 807–816. <https://www.doi.org/10.1001/archpsyc.61.8.807>
- [15] Buckner, J. D., Heimberg, R. G., Schneier, F. R., Liu, S. M., Wang, S., & Blanco, C. (2012). The relationship between cannabis use disorders and social anxiety disorder in the National Epidemiological Study of Alcohol and Related Conditions (NESARC). *Drug and alcohol dependence*, 124(1-2), 128–134. <https://doi.org/10.1016/j.drugalcdep.2011.12.023>
- [16] Buckner, J. D., Silgado, J., & Schmidt, N. B. (2011). Marijuana craving during a public speaking challenge: Understanding marijuana use vulnerability among women and those with social anxiety disorder. *Journal of Behavior Therapy and Experimental Psychiatry*, 42(1), 104–110. <https://doi.org/10.1016/j.jbtep.2010.07.005>
- [17] Stapinski, L. A., Montgomery, A. A., & Araya, R. (2016). Anxiety, depression and risk of cannabis use: Examining the internalising pathway to use among Chilean adolescents. *Drug and alcohol dependence*, 166, 109–115. <https://doi.org/10.1016/j.drugalcdep.2016.06.032>
- [18] Buckner, J. D., Schmidt, N. B., Lang, A. R., Small, J. W., Schlauch, R. C., & Lewinsohn, P. M. (2008). Specificity of social anxiety disorder as a risk factor for alcohol and cannabis dependence. *Journal of Psychiatric Research*, 42(3), 230–239. <https://doi.org/10.1016/j.jpsychires.2007.01.002>
- [19] Agosti, V., Nunes, E., & Levin, F. (2002). Rates of psychiatric comorbidity among U.S. residents with lifetime cannabis dependence. *American Journal of Drug and Alcohol Abuse*, 28(4), 643–652. <https://doi.org/10.1081/ADA-120015873>
- [20] Wills, T. A., & Hirky, A. E. (1996). *Coping and substance abuse: A theoretical model and review of the evidence*. John Wiley & Sons.
- [21] Khantzian E. J. (1997). The self-medication hypothesis of substance use disorders: A reconsideration and recent applications. *Harvard Review of Psychiatry*, 4(5), 231–244. <https://doi.org/10.3109/10673229709030550>
- [22] Reinerman, C., Nunberg, H., Lanthier, F., & Heddleston, T. (2011). Who are medical marijuana patients? Population characteristics from nine California assessment clinics. *Journal of psychoactive drugs*, 43(2), 128–135. <https://doi.org/10.1080/02791072.2011.587700>
- [23] Boden, M. T., McKay, J. R., Long, W. R., & Bonn-Miller, M. O. (2013). The effects of cannabis use expectancies on self-initiated cannabis cessation. *Addiction*, 108(9), 1649–57. <http://doi.org/10.1111/add.12233>
- [24] Cyders, M. A., & Smith, G. T. (2008). Emotion-based dispositions to rash action: Positive and negative urgency. *Psychological Bulletin*, 134(6), 807–828. <https://doi.org/10.1037/a0013341>

- [25] Smith, G. T., & Cyders, M. A. (2016). Integrating affect and impulsivity: The role of positive and negative urgency in substance use risk. *Drug and Alcohol Dependence*, 163(1), S3–S12. <https://doi.org/10.1016/j.drugalcdep.2015.08.038>
- [26] Verdejo-García, A. J., Perales, J. C., & Pérez-García, M. (2007). Cognitive impulsivity in cocaine and heroin polysubstance abusers. *Addictive Behaviors*, 32(5), 950–966. <https://doi.org/10.1016/j.addbeh.2006.06.032>
- [27] Keough, M. T., Hendershot, C. S., Wardell, J. D., & Bagby, R. M. (2017). Investigating the mediational role of negative urgency in the anxiety sensitivity pathway to cannabis problems and dependence symptoms among postsecondary students. *Journal of American college health*, 66(2), 69–75. <https://doi.org/10.1080/07448481.2017.1369093>
- [28] Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Sage Publications, Inc.
- [29] Simons, J. S., Dvorak, R. D., Merrill, J. E., & Read, J. P. (2012). Dimensions and severity of marijuana consequences: Development and validation of the Marijuana Consequences Questionnaire (MACQ). *Addictive Behaviors*, 37(5), 613–21. <https://doi.org/10.1016/j.addbeh.2012.01.008>
- [30] Liebowitz, M. R. (1987). Social Phobia. *Modern Problems of Pharmacopsychiatry*, 22, 141–173. <https://doi.org/10.1159/000414022>
- [31] Cyders, M. A., Smith, G. T., Spillane, N. S., Fischer, S., Annus, A. M., & Peterson, C. (2007). Integration of impulsivity and positive mood to predict risky behavior: Development and validation of a measure of positive urgency. *Psychological Assessment*, 19(1), 107–118. <https://doi.org/10.1037/1040-3590.19.1.107>
- [32] Cohen, J. (1992). A power primer. *Psychological Bulletin*, 112(1), 155–159. <https://doi.org/10.1037/0033-295X.112.1.155>
- [33] Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics* (5th ed.). Pearson Education.
- [34] Oei, T. P. S., & Morawska, A. (2004). A cognitive model of binge drinking: The influence of alcohol expectancies and drinking refusal self-efficacy. *Addictive Behaviors*, 29(1), 159 – 179. [https://doi.org/10.1016/s0306-4603\(03\)00076-5](https://doi.org/10.1016/s0306-4603(03)00076-5)
- [35] Curry, I., Trim, R. S., Brown, S. A., Hopfer, C. J., Stallings, M. C., & Wall, T. L. (2018). Positive expectancies mediate the association between sensation seeking and marijuana outcomes in at-risk young adults: A test of the acquired preparedness model. *American Journal on Addictions*, 27(5), 419–424. <https://doi.org/10.1111/ajad.12754>

Intraindividual Variability of Proactive Control in Cognitive Aging *

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Executive functions are known to decline as a function of healthy aging. The Dual Mechanisms of Control model characterizes age-sensitive executive functions by positing two mechanisms: proactive and reactive control. Proactive control recruits cognitive resources such as maintenance of goal-relevant information in working memory, allowing for the anticipation of a forthcoming interference. Given the known age-related declines in working memory, I investigated the role of intraindividual variability of proactive control in relation to working memory processes in 25 young adults (18-30 years old, $M = 25.72$) and 26 older adults (60-80 years old, $M = 71.19$) using a computerized AX-CPT paradigm. A working memory index (Letter Number Sequencing) significantly predicted intraindividual variability of proactive control ($t = -3.01$, $p = .004$, 95 % CI for t [-17.49, -3.47], $s_r^2 = .167$). The present findings provide preliminary evidence that working memory processes are involved in age-related intraindividual variability of proactive control.

Healthy aging is often accompanied by declines in memory, attention, and inhibition [1, 2, 3]. These cognitive changes in older adults (OA) may be a result of normative age-related declines in executive functioning [4, 5]. Higher order cognitive control processes, such as executive functions, supervise and regulate incoming information to achieve goal-intended behaviors [6]. One classification of these executive control processes includes three divided components. The first includes working memory updating, which involves adding and deleting information from working memory. The second is response inhibition, which is defined as suppressing goal-irrelevant information. The third is task-switching which consists of being able to flexibly alternate between mental tasks [7]. Among all cognitive processes, executive functions are known to be especially age-sensitive [8]. For instance, deficits in working memory updating and response inhibition account for a significant portion of the age-related variance in working memory [9]. The current study was designed to examine the role of executive functions in relation to the variable nature of cognition in aging. Specifically, the influence of intraindividual variability of proactive control was investigated with regards to the maintenance of goal-relevant information in working memory.

One candidate model of executive functions that may be applicable to understanding age differences in cognitive control is Braver's Dual Mechanisms of Control (DMC) model [10]. Braver posited that when individuals are faced with goal-irrelevant information, they use two main modes of cognitive control to resolve the interference and accomplish their task. In proactive control, goal-relevant information is maintained early on in working memory, in order to anticipate and adjust for an incoming interference. Proactive control is highly demanding on cognitive resources due to the need to maintain goal-relevant cue information in working memory in anticipation of a later target event. At the same time, this cue maintenance also serves to suppress goal-irrelevant information. For example, if an individual is standing on a bus and sees a stop sign coming up ahead, they may use the goal-relevant information (the stop sign) to anticipate

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a change in movement, allowing them to adjust ahead of time. Conversely, reactive control is a late stimulus-driven reflexive response after the interference or stimulus is presented to correct and readjust for the goal-intended behavior. Reactive control is less cognitively demanding than proactive control, as there is not the same need to maintain goal-relevant information in working memory. In the same example, the individual may have failed to maintain the goal-relevant information (that there is a stop sign coming up ahead) in working memory and had to rely on a reflexive behavior. Researchers have found that older adults exhibit a greater use of reactive control compared to young adults (YA), as a result of age-related declines in executive functions, such as working memory. Young adults, on the other hand, exhibit a greater use of proactive control compared to older adults [11].

To measure proactive and reactive control in older and young adults, Braver used the AX-Continuous Performance Task (AX-CPT) [11]. This computerized reaction time task evaluates participant engagement in proactive and reactive control when faced with goal-irrelevant information. The AX represents a cue-stimulus pairing. In each trial, a letter cue (either A or B) is presented on a screen for a short period of time, followed by a cue-stimulus interval (CSI). This time delay engages the maintenance of goal-relevant information (the cue) in working memory. Following the CSI, a letter stimulus (X or Y) is presented for a limited duration and participants are asked to respond as quickly as possible. The presentation of an AX (cue-stimulus) pairing requires a target button response (left mouse click), while presentation of any of the three other possible pairings (AY, BX, and BY) requires a non-target button response (right mouse click).

The trial type distribution is as follows: 70% AX, 10% AY, 10% BX, and 10% BY. A greater proportion of AX trials creates a response bias towards the A cue over trials. On 70% of trials, an X stimulus follows an A cue, requiring a target response. The A cue becomes associated with the need to make a target response. Intact proactive control leads to the successful maintenance of the A cue in working memory and the anticipation of the target response. The presentation of a B cue, regardless of the stimulus type, elicits a non-target response. In contrast, reliance on reactive control may be caused by a failure to maintain the A cue in working memory. As a result, the presentation of an X stimulus is more likely to elicit a reflexive target response (false positive), regardless of the initial cue type.

Due to the A cue response bias eliciting an X stimulus expectancy, participants utilizing proactive control are more prone to making false target response errors on AY trials. This tendency is greater in young adults as a consequence of their greater engagement in proactive control [11]. Conversely, participants engaging in reactive control are more susceptible to false target response errors on BX trials, as a result of reactive responding upon the presentation of an X stimulus. This greater tendency observed in older adults is attributed to an increased use of reactive control [11]. Lastly, BY trials are considered to be a control trial type as there is no A cue to trigger a response bias, nor an X stimulus to engage a reactive response.

Age-related differences have been observed in the use of proactive control in a cognitive setting and in working memory capacity [9, 11]. However, there is little research addressing the role of intraindividual differences (within-person change) in the cognitive applications of the DMC theory. There is experimental evidence suggesting that individual differences in working memory may lead to differences in the ability to maintain consistent cognitive control [12]. Specifically, young adults with a lower working memory capacity exhibit greater intraindividual variability of proactive control, resulting in more AX errors on the AX-CPT, compared to those with a higher

working memory capacity [13]. However, Weimers and Redick defined intraindividual variability, in the context of the DMC framework, as a general inconsistency in responding on the overall task, rather than the variable application of both cognitive control processes (proactive and reactive control). In their study, variability was specifically calculated as a function of mean differences in accuracy scores across testing blocks [13]. Previous research also suggests an age-related increase in general intraindividual variability [14, 15, 16]. The nature of this variability has also been suggested to be both adaptive and maladaptive [17]. Increased variability offers more opportunity for practice-related gains, which becomes an adaptive learning tool. Alternatively, intraindividual variability can be maladaptive in nature due to the overall inconsistency of responding [17]. The work of Weimers and Redick [13] identifies working memory as a key influence but does not explore whether working memory is linked to any age-related differences in the intraindividual variability of the specific application of proactive control on the AX-CPT. These questions are warranted, given the known age-related declines in working memory capacity in healthy aging, and the importance of cognitive control in the aging population. The present study was designed to examine the role of intraindividual variability in a cognitive application of the DMC model. Specifically, cognitive age-related differences in intraindividual variability of proactive control were investigated using the computerized AX-CPT. Given the known age-related differences in general intraindividual variability and working memory capacity, it was hypothesized that older adults would exhibit greater cognitive intraindividual variability of proactive control compared to young adults.

Method

Participants

The current study used archival data collected from May to October 2018 in the Li Lab at Concordia University. The study was approved by the Human Research Ethics Committee of Concordia University. An a priori power analysis using G Power revealed that 25 participants were needed per age group to achieve a power of .80, at a moderate effect size ($\eta p^2 = .15$) [18]. This sample size requirement was met for the current analyses. Participants were comprised of 26 community-dwelling older adults, between the ages of 60 and 80 years old, and 25 young adults between the ages of 18 and 30 years old (see Table 1 for sample characteristics). Older adults were recruited through advertisements placed around the Montreal community and young adults were recruited through Concordia University's Psychology participant pool. The inclusion criteria required all participants to be English-speaking, with no prior history of cognitive impairment. Participants also had to be free of any self-reported visual and auditory impairments. All participants were screened for exclusion criteria by telephone prior to testing. Further screening was conducted upon arrival at the lab. Older adults were excluded if they scored less than or equal to 26 out of 30 on the Montreal Cognitive Assessment (MoCA), which screens for mild cognitive impairment in combination with falling below age normative ranges on one or more neuropsychological measure [19]. All participants provided written and verbal consent. As shown in Table 1, participants did not significantly differ in terms of the years of education they possessed. Older adults' mean MoCA score was above the cutoff of 26 for potential mild cognitive impairment. Most notably, older adults did not significantly differ from young adults on the Letter Number Sequencing task, which assesses auditory working memory, $t(52) = 1.79, p = .079, 95\% \text{ CI} = [-0.12, 2.17], d = 0.49$.

Table 1*Sample Characteristics on Background Measures*

	Young Adults		Older Adults		<i>p</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
Age (years)	25.72	5.50	71.19	4.35	-
Education (years)	16.48	2.22	15.90	3.21	.449
MoCA (max.30)	-	-	27.39	2.14	-
LNS (max.30)	19.79	1.89	18.77	2.24	0.79

Note. Sample characteristics of participant age in years, number of years of education, performance on the Montreal Cognitive Assessment (MoCA) scored out of 30, and performance on the Letter Number Sequencing (LNS) task scored out of 30. Independent sample *t*-tests were conducted to examine group differences between young and older adults.

**p* < .05

Measures

Participants completed a demographic questionnaire that included information such as age, gender, medical history, and level of education to gain more knowledge on the characteristics of the sample. Additional background measures were also administered for a second separate study. Relevant to the current research, the MoCA was used to screen for potential cognitive impairment in older adults [19]. This measure had good internal reliability with a Cronbach's α of .79, comparable to the reported norms (Cronbach's α = .83) [19]. To examine participants' auditory working memory, Letter Number Sequencing was used [20]. Assessors auditorily presented a random list of letters and numbers and asked participants to recall them in alpha-numeric order. A total Letter Number Sequencing score was derived by averaging out the number of correct responses in each item. Measures of internal consistency revealed good reliability with a Cronbach's α of .76, falling slightly below the reported norms (Cronbach's α = .87) [20]. Finally, the Computerized AX-CPT was used as a measure of cognitive control processes from the DMC model [11].

Computerized AX-CPT. The computerized AX-CPT followed a similar experimental design as the one previously employed in Braver's study [11]. Participants focused their attention on a computer screen. A letter cue (either A or B) was presented for a duration of 300 milliseconds (ms), followed by a CSI of 4900 ms. This maintenance period was designed to be lengthy to engage working memory processes. Following the delay, participants were presented with a stimulus (either X or Y) for 300 ms and subsequently given 1300 ms to respond with either a target or non-target button click. To indicate the end of a trial, a screen with the text "Trial is over, get ready for next one" was displayed for 1000 ms (see Appendix A). Participants were told in advance that when an AX pair was presented, they should press the target button (left mouse click). When any other cue-stimulus pairing (AY, BX, or BY) was presented, they should press the non-target button (right mouse click). Accuracy scores (probability score out of 1.00) and reaction times (ms) were recorded for each trial. The experiment was made up of four blocks containing 50 trials each, for

a total of 200 trials. Trial type frequencies were consistent with Braver et al.'s [11] study: 70% AX, 10% AY, 10% BX, and 10% BY. All letter cues and stimuli were presented in a white bolded uppercase font, using 36-point Helvetica, on a black background [21]. Inquisit 4 software was used to present the AX-CPT (Millisecond Software, Seattle).

Procedure

The study was comprised of one experimental session. This session took place in Concordia University's Psychology Department and lasted an average of 1.5 hours. Participants signed the consent form upon arrival. Participants then completed the demographic questionnaire, followed by the background measures and neuropsychological assessments (MoCA, Computerized AX-CPT, Letter-Number Sequencing). Older adults were compensated with a \$20 honorarium, while young adults were solely compensated with participant pool credits.

Analyses

The hypothesis relating to intraindividual variability of proactive control was analyzed using Weimers and Redick's [13] measure of proactive shift in the computerized AX-CPT paradigm. Mean accuracy and reaction time of the second half of trial blocks (3 and 4) was subtracted from the first half of trial blocks (1 and 2), to obtain measures of change in accuracy and reaction time for trial types AY and BX. These trial types were most relevant in evaluating the use of proactive control, as they reveal behavioral patterns indicative of each mode of cognitive control. For accuracy, a positive value for mean change is indicative of an increased shift to proactive control over time. A negative value indicates a shift away from proactive control across blocks. For reaction time, the inverse is true. A positive value is indicative of decreased proactive control, while a negative value indicates increased proactive control over time. These analyses capture global intraindividual variability on the AX-CPT. However, Weimers & Redick fail to give a complete representation of proactive control as their analyses were originally designed to measure an overall inconsistency of responding in the task, in combination with measures of hits and false alarms for AX and BX trials, rather than variability specific to proactive control. Going beyond the Weimers and Redick analyses, a Proactive Behavior Index (PBI) was also derived for each participant, with the equation $(AY-BX)/(AY+BX)$ [21]. The Weimers & Redick [13] and Braver et al. [21] analyses were considered as they both offered a unique measure of intraindividual variability. The PBI represents a quantitative measure of interference between AY and BX trials. A positive PBI is indicative of proactive control, while a negative PBI is indicative of reactive control. The PBI was obtained for reaction time and error rate averages on AY and BX trials. For error rate computations, the log-linear correction (number of errors + 0.5) / (number of trials + 1) was applied to correct for trials where the error rate was 0 [22]. Intraindividual variability was obtained by subtracting the PBI of the first two blocks from the last two blocks, in a similar manner to the Weimer's and Redick analyses. Greater intraindividual variability indicates a greater inconsistency in the use of cognitive control, as predicted for older adults.

Independent samples t-tests were conducted to compare young and older adults on mean accuracy change for AY and BX trials, as well as on the intraindividual variability of PBI measures [13, 21]. A one-way repeated measures ANOVA was used to assess participants' general patterns across of responding all four blocks on the computerized AX-CPT based on their reaction times. Finally, a multiple linear regression was used to assess the relation between intraindividual variability of proactive control and cognitive processes of working memory.

Results

Data Integrity

Preliminary data screening measures were conducted. Cases in which participants scored below the age-normative range on two or more neuropsychological tests were excluded from the analyses. These criteria resulted in the exclusion of three older adults. Additionally, one older adult's MoCA score exceeded the cutoff of +/- 3 standard deviation units and was thus winsorized. For the computerized AX-CPT reaction time analyses, only correct trials were processed, eliminating incorrect trials and trials where participants were too slow to respond within the 1300 ms response window. Two reaction time outliers within the correct responses were not removed, as the original authors suggested that the response window in the AX-CPT paradigm is short enough as is [11]. For error rate exclusions, a chance level of 50% on AX trials in any given block, was used as the threshold level. No participants fell below this threshold.

Computerized AX-CPT reaction time and error rate distributions were assessed for skewness and kurtosis using a z-score value of +/- 1.96. As expected, reaction time distributions were right skewed by young adults. Data normality checks for error distributions also revealed skewness and kurtosis. Both distributions remained skewed after log transformations were applied. Alternative non-parametric tests, such as the independent-samples Mann-Whitney U tests, did not yield different results in the analyses. Therefore, the results from parametric tests are reported in the current study, to be consistent with reporting methods from previous literature [11, 23].

Computerized AX-CPT: Overall Performance

Before testing the hypothesized age difference in cognitive intraindividual variability, basic analyses were carried out to describe the computerized AX-CPT data set more generally. To investigate the general patterns of responding in young adults, a one-way repeated measures ANOVA was conducted using reaction time on all four trial types (see Appendix B for mean reaction times over all four blocks). The assumption of sphericity was violated for trial type according to Mauchly's Test, $\chi^2(5) = 24.02, p < .001$. Greenhouse-Geisser estimates were considered to reduce Type I error ($\epsilon = .62$). There was a statistically significant main effect of trial type, $F(1, 39) = 119.03, p < .001, \eta^2 = .85$. Bonferroni post-hoc corrections revealed that young adults exhibited significantly slower reaction times on AY trials than on AX trials, MDIFF = 69.68, $p < .001$, 95% CI [93.17, 162.85], $d = -0.86$. However, young adults performed significantly faster on BX trials compared to AX trials, MDIFF = 71.72, $p < .001$, 95% CI [-113.32, -42.04], $d = 1.31$. Therefore, there was evidence of young adults utilizing proactive control in the computerized AX-CPT.

To investigate the general patterns of responding in older adults, a one-way repeated measures ANOVA was conducted, examining reaction time on all four trial types (see Appendix B for mean reaction times over all four blocks). Mauchly's Test revealed that sphericity was violated for trial type, $\chi^2(5) = 14.69, p = .012$. Greenhouse-Geisser estimates were applied ($\epsilon = .74$). A statistically significant main effect of trial type was found, $F(2, 55) = 101.30, p < .001, \eta^2 = .80$. Bonferroni post-hoc corrections revealed that older adults exhibited significantly slower reaction times on AY trials than on AX trials, MDIFF = 127.71, $p < .001$, 95% CI [164.08, 291.79], $d = -1.44$. However, older adults also performed significantly faster on BX trials compared to AX trials, MDIFF = 134.74, $p < .001$, 95% CI [-349.55, -214.81], $d = 1.79$, therefore, showing evidence that older adults were also engaging in proactive control in the computerized AX-CPT.

To further compare young and older adults' engagement in proactive control, an independent

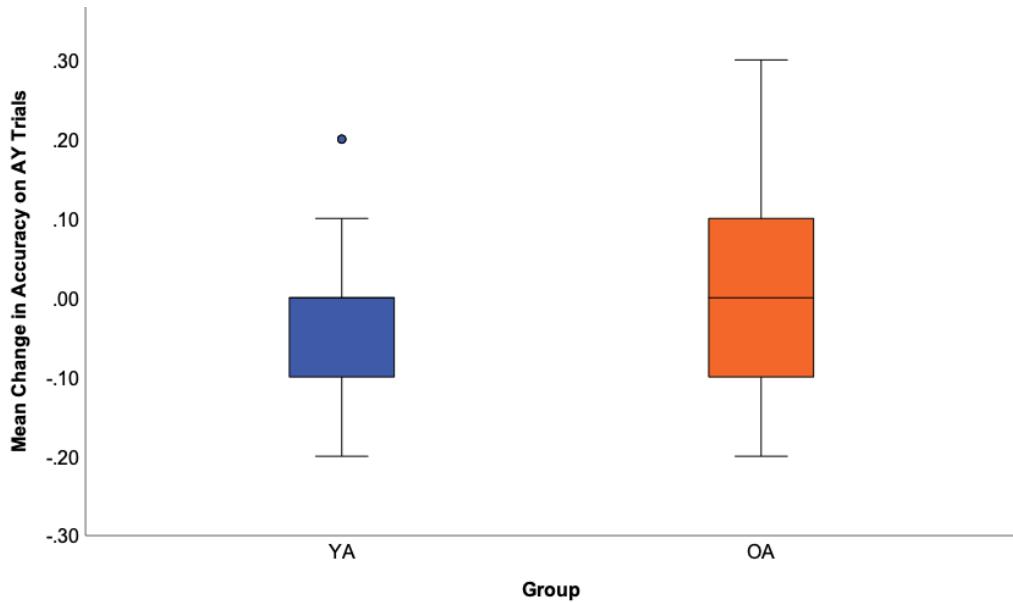
samples t-test was conducted using the more robust mean PBI scores across all blocks, as a function of reaction time, between young adults ($M = 0.197$, $SD = 0.07$) and older adults ($M = 0.199$, $SD = 0.08$). No statistically significant difference was revealed, $t(46) = -0.09$, $p = .926$, 95% CI for t [-0.05, 0.04], $d = -0.03$. The same pattern of results was found using PBI scores derived from correct error rates, $t(48) = 0.40$, $p = .695$, 95% CI for t [-0.10, 0.15], $d = 0.11$. Therefore, it was concluded that young and older adults did not significantly differ in their overall use of proactive control.

Hypothesis: Intraindividual Variability in AX-CPT Performance

To test the first hypothesis, that older adults exhibit greater intraindividual variability of proactive control than young adults, Weimers and Redick's [13]proactive shift scores were first considered. An independent samples t-test revealed no statistically significant difference between young adults ($M = -0.016$, $SD = 0.11$) and older adults ($M = -0.056$, $SD = 0.22$) in their mean accuracy change on AY trials across earlier and later blocks, $t(50) = 0.82$, $p = .418$, 95% CI for t [-0.06, 0.14], $d = 0.23$ (see Figure 1). The same pattern was found in mean accuracy change on BX trials, $t(50) = -1.82$, $p = .075$, 95% CI for t [-0.14, 0.01], $d = -0.51$, between young ($M = -0.040$, $SD = 0.13$) and older adults ($M = 0.026$, $SD = 0.14$). Therefore, using Weimers and Redick's measure of proactive shift, it was concluded that there were no group differences in variability of proactive control on the computerized AX-CPT.

Figure 1

Mean change in accuracy on AY trials in older and young adults

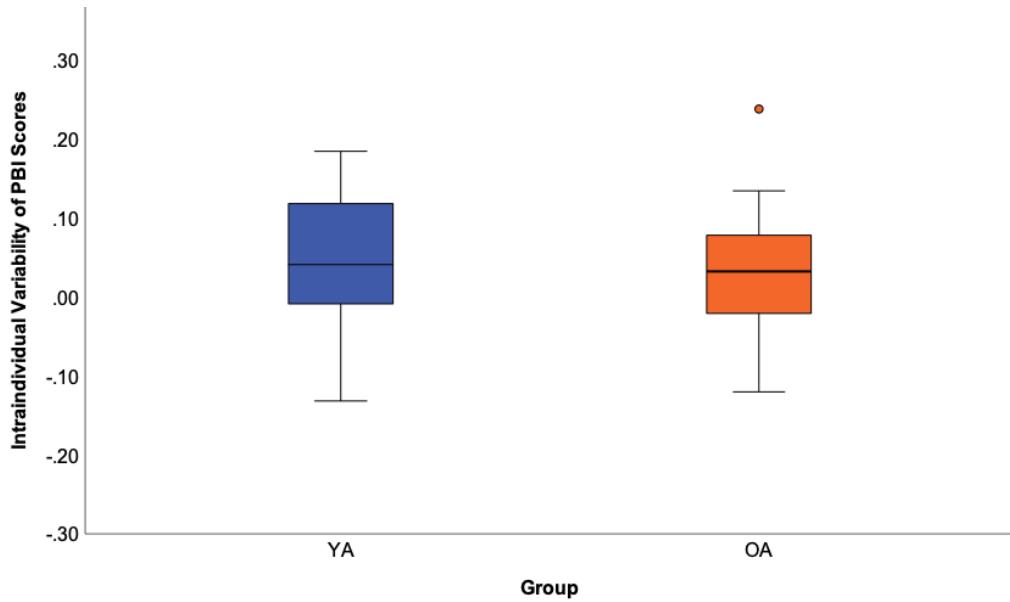


Note. Comparing young adults (YA) and older adults (OA) on the computerized AX-CPT as a function of mean change in accuracy (probability out of 1.00) on AY trials, calculated by subtracting mean accuracy from the first two blocks from mean accuracy of the last two blocks. Error bars represent 95% confidence intervals. Dots falling outside of the intervals represent outliers (less than 50% accuracy on AX trials overall).

A supplementary analysis using Braver's more robust measure of PBI also replicated the findings. There was no statistically significant difference between young adults ($M = 0.044$, $SD = 0.09$) and older adults ($M = 0.036$, $SD = 0.08$) in their intraindividual variability of PBI scores, based on reaction time, $t(49) = .31$, $p = .759$, 95% CI for t [-0.04, 0.05], $d = 0.09$ (see Figure 2). The same result was found for intraindividual variability of PBI scores, based on corrected error rates, $t(50) = -1.43$, $p = .160$, 95% CI for t [-0.42, 0.07], $d = -0.39$, between young ($M = -0.041$, $SD = 0.28$) and older adults ($M = 0.132$, $SD = 0.56$). Therefore, the null hypothesis was retained given that there was no statistically significant difference between young and older adults in their intraindividual variability of proactive control based on their reaction times and error rates on the computerized AX-CPT.

Figure 2

Intraindividual Variability of PBI Scores in older and young adults



Note. Comparing young adults (YA) and older adults (OA) on the computerized AX-CPT as a function of intraindividual variability of Proactive Behavioral Index (PBI) scores, calculated using mean reaction times on AY and BX trials with the equation $[AY-BX]/[AY+BX]$. Error bars represent 95% confidence intervals. Dots falling outside of the intervals represent outliers (+/- 3 standard deviations from the mean).

The previous measures of intraindividual variability did not take into consideration AX trials, which make up 70% of all trials. An independent samples t-test revealed a statistically significant difference between young and older adults in mean reaction times on AX trials overall, $t(48) = -4.13$, $p < .001$, 95% CI for t [-159.51, -55.00], $d = -1.17$. However, an additional independent samples t-test revealed no statistically significant difference between older adults ($M = 1.46$, $SD = 2.27$) and young adults ($M = 0.84$, $SD = 2.27$) in their mean number of errors on AX trials overall, $t(48) = -0.97$, $p = .336$, 95% CI for t [-1.92, 0.67], $d = -0.28$, indicating that older adults were slower to respond on 70% of trials, but that had no impact on their overall accuracy on AX trials. A lack of differences in error rates on AX trials, further solidifies the similarity in performance between the

two groups.

Working Memory and Intraindividual Variability. An exploratory analysis was conducted to determine whether the different measures of intraindividual variability could predict Letter Number Sequencing scores. The reason for this prediction was to investigate the association between working memory processes and cognitive intraindividual variability. A multiple linear regression was conducted with Weimers' and Redick's [13] measures of mean change in accuracy (on AY and BX trials) and measures of intraindividual variability using Braver et al.'s [21] PBI scores. These variables were set as predictors for Letter Number Sequencing total scores (see Appendix C for descriptive statistics). Young and older adults were pooled together, given that they showed no statistically significant difference in performance on the Letter Number Sequencing task (see Table 2). The overall model was not statistically significant, $F(4, 45) = 2.28, p = .076, R^2 = .17$. However, the following variables were revealed to be statistically significant predictors of Letter Number Sequencing total scores: mean change in accuracy on AY trials ($t = 2.17, p = .035, 95\% \text{ CI for } t [0.44, 11.90], sr^2 = .087$), mean change in accuracy on BX trials ($t = -3.01, p = .004, 95\% \text{ CI for } t [-17.49, -3.47], sr^2 = .167$), and intraindividual variability of PBI based on error rate ($t = 2.38, p = .021, 95\% \text{ CI for } t [.44, 5.17], sr^2 = .105$). Therefore, intraindividual variability of proactive control within the computerized AX-CPT was associated with working memory capacity, as measured with the Letter Number Sequencing task.

Table 2

Results of Multiple Regression Analysis of Predictors of Letter Number Sequencing Scores

Predictor	Unstandardized coefficients		Standardized coefficients	$t (45)$	p	s_r^2
	B	$95\% \text{ CI for } B$				
AY Change in Accuracy	6.17	[.44, 11.90]	2.85	.525	2.169	.035*
BX Change in Accuracy	-10.48	[-17.49, -3.47]	3.48	-.679	-3.012	.004*
IIV of PBI (reaction time)	-1.95	[-9.15, 5.25]	3.57	-.076	-.546	.588
IIV of PBI (error rate)	2.80	[.435, 5.171]	1.18	.612	2.384	.021*

Note. $N = 50, R = .410, R^2 = .168$, adjusted $R^2 = .094$. Predictors of Letter Number Sequencing scores were set as the mean change in accuracy on AY trials, the mean change in accuracy on BX trials, the intraindividual variability (IIV) of Proactive Behavioral Index (PBI) as a function of reaction time (ms), and the intraindividual variability (IIV) of Proactive Behavioral Index (PBI) as a function of corrected error rates. * $p < .05$

Discussion

The current study was designed to investigate the role of intraindividual variability of cognitive control in relation to maintaining goal-relevant information in working memory on the computerized AX-CPT paradigm. Age differences in intraindividual variability were examined separately for both older and young adults. The variable nature of proactive control and its association with working memory in older and young adults was the primary focus of the present study. As a function of normative age-related declines in executive functions, it was hypothesized that older adults would exhibit greater intraindividual variability of proactive control in the computerized AX-CPT.

The current findings suggest that both young and older adults engage in proactive control, when goal relevant information is available. Intraindividual variability of proactive control in older adults was comparable to that of young adults, failing to support the hypothesis. In a supplementary analysis, an independent indicator of working memory (the Letter Number Sequencing task) was found to be associated with the measures of intraindividual variability of proactive control, showing partial evidence for a link between working memory processes and individual differences of cognitive strategies.

Computerized AX-CPT

The computerized AX-CPT assessed modes of proactive and reactive control in older and young adults. Measures of reaction time revealed that young adults were slower to respond on AY trials compared to AX trials, indicating a use of proactive control. The trial type distribution in the AX-CPT favors an X stimulus expectancy response following an A cue, as a result of the high occurrence of AX trials (70% of trials). Therefore, slower reaction times on AY trials is indicative of anticipatory modes of cognitive control, as more time is required to resolve the occurrence of an unexpected Y stimulus following an A cue.

Young adults also performed faster on BX trials compared to AX trials, suggesting a lack of reliance on reactive control brought on by the presentation of an X stimulus. Reactive control involves a potential failure to maintain goal relevant information in working memory (the B cue). Therefore, an uncertainty in the preceding cue type would provoke a form of response conflict on BX trials. Reactive control would thus result in slower reaction times and an increased rate of false alarms on BX trials. As a result, the young adult pattern of responding is suggestive of an engagement in proactive control rather than reactive control on the computerized AX-CPT. These findings have been consistently supported in the DMC literature [11, 21].

Inconsistent with the literature, older adults were also found to engage in proactive control on the computerized AX-CPT. Previous studies have reflected age-related differences in modes of proactive and reactive control in older and young adults [11, 21, 23]. Like the young adults, for older adults, slower reaction times were observed on AY trials compared to AX trials, as well as faster reaction times on BX trials compared to AX trials. Further, the Proactive Behavioral Index provides an alternative way to quantify the use of proactive control and revealed statistically comparable levels in young and older adults. A previous study conducted using a smaller subset of the current sample revealed similar findings to the present study; both young and older adults engaged in proactive control in the computerized AX-CPT [18].

Inconsistencies between the present findings and the literature may be attributable to the char-

acteristics of this sample. For example, young and older adults performed similarly on the Letter Number Sequencing task, an indicator of working memory. However, age-related working memory deficits have been reported widely [9, 24, 25], specifically on the Letter Number Sequencing task [26, 27]. Therefore, older adults in this sample may possess a higher level of cognitive functioning. As noted previously, three older adults scored higher on the Letter Number Sequencing task than any of the young adults. The same analyses, with these participants excluded, yielded the same results, suggesting that the overall sample of older adults may be higher functioning than average. Comparing the standardized Letter Number Sequencing scores to the relative age-norms provided further evidence of a high-functioning older adult sample. A total of 10 older adults scored 11 or higher, indicating that they fell in the 69th or higher percentile. Therefore, the sample of older adults would be deemed high functioning in accordance with the published norms [20].

Intraindividual Variability of Proactive Control

Given the high-functioning nature of older adults in this sample, comparable performance was revealed on measures of intraindividual variability of proactive control between the two age groups. Although not statistically significant, the operationalization of intraindividual variability of proactive control by Weimers and Redick [13] using the change in mean accuracy scores on BX trials, produced a moderate effect size ($d = -0.51$). This pattern was moderately supported by Braver et al.'s [21] more robust Proactive Behavioral Index measures of variability based on corrected error rates ($d = -0.39$). However, these patterns were not consistent across AX trials or measures of PBI based on reaction time. Previous literature fails to report consistent effect sizes in the field, leaving interpretation to be cautioned. Contextualizing these findings within the literature, the results demonstrate a lack of support for Weimers and Redick's [13] previous findings suggesting individual differences in intraindividual variability of proactive control. However, the work of Weimers and Redick focused on high and low working memory capacity young adult groups and did not examine the effects of aging by including older adults. Additionally, the authors went beyond the scope of proactive control when defining intraindividual variability in the AX-CPT. Weimers and Redick examined AX errors and proactive variability as a function of more time-on-task effects, using two distinct design manipulations, which was not possible for the current study given the archival nature of the data. The current findings are also inconsistent with the overall effect of an age-related increase in intraindividual variability. Specifically, it has been well-documented in the literature that older adults exhibit a greater level of variability in their reaction times compared to young adults [15, 16, 28, 29]. Therefore, the alternative pattern of results in the current study may have been a product of the characteristics of the sample, such as the high-functioning capacity of older adults.

Working Memory and Intraindividual Variability

Letter Number Sequencing was used as an independent indicator of working memory. Performance on this task was found to be predicted by the measures of intraindividual variability of proactive control. Both of Weimers and Redick's [13] measures of proactive shift, using AY and BX trials, were revealed to be statistically significant predictors of working memory performance, uniquely accounting for 8.7% and 16.73% of the variance in intraindividual variability respectively. In addition, Braver et al.'s [21] more robust PBI measure, calculated using error rates, replicated these findings and was also a statistically significant predictor of Letter Number Sequencing scores, uniquely accounting for 10.5% of the variance. The involvement of working

memory processes in intraindividual variability of proactive control strengthens the validity of the computerized AX-CPT. This paradigm was designed to capture executive functions involved in cognitive control, such as working memory. The association between working memory and an individuals' preferred mode of cognitive control has been previously studied [30]. Other research using anti-saccade tasks, which require the maintenance of a task rule in working memory, has revealed increased intraindividual variability of cognitive control in individuals with a low working memory capacity [31, 32, 33, 34]. However, the current findings are the first to reveal an association between intraindividual variability of proactive control and working memory in the computerized AX-CPT. The novelty of this finding contributes to solidifying the DMC as a model of executive functions, and the implication of working memory capacity therein.

Implications

Given that young and older adults did not differ in the variability with which they engaged in proactive control in the computerized AX-CPT, it is suggested that they are not reaping the potential adaptive benefits of increased variability, while simultaneously not being affected by any maladaptive effects of increased variability. As proposed by Allaire and Marsiske [17], greater variability in responding offers more opportunities for learning the correct and optimal form of response, thus proving to be adaptive in the long run. Conversely, increased variability is defined by a general inconsistency in responding, resulting in more overall errors on a given task, as opposed to employing the optimal form of response throughout. Therefore, older adults were neither at a loss nor at a gain, as their level of intraindividual variability was comparable to that of young adults on the computerized AX-CPT. An independent index of working memory (the Letter Number Sequencing task) was associated with the measures of intraindividual variability of proactive control. Therefore, the current findings tentatively support the involvement of working memory processes in age-related declines in cognitive control. A greater overall understanding of fall risk can allow for the implementation of prevention measures to minimize such risks.

Limitations and Future Directions

Limitations within the current study also offer potential future avenues for research. Characteristics of the sample may have played a role in the comparable performance of young and older adults. A form of self-selection bias may have been introduced, resulting in a subset of high-functioning older adults choosing to participate in the study. The AX-CPT designs themselves also offered potential limitations to the study. For instance, the number of AY and BX trials was fairly limited throughout the paradigm. These trials are essential to determining the use of proactive control, yet they only occur 10% of the time, amounting to a total of only 20 trials across all four blocks. One error on AY and BX trials can thus have a large impact on a participant's PBI. The current design was similar to Paxton et al.'s study involving the same number of trials [35]. Weimers and Redick [13] improved upon this design by doubling the number of blocks, resulting in double the amount of AY and BX trials. This increase allowed for a greater representation of intraindividual variability within the different blocks. However, given the archival nature of the data, adjustments to the design for the specific hypotheses was not possible.

Future research should consider the role of other executive functions, such as inhibition, task switching, and processing speed. Age differences in processing speed have been previously investigated at length and could offer a new perspective into the DMC model, both in terms of understanding the variability of proactive and reactive control in the computerized AX-CPT [36].

The current study only examined one type of executive function, that is working memory. To the best of my knowledge, different executive functions could be involved in relation to intraindividual variability in cognitive aging. Therefore, it would be interesting to consider the novelty of further examining executive functions through the DMC lens.

Conclusion

The present study investigated the role of intraindividual variability as a function of cognitive aging. A standard computerized AX-CPT was used to measure proactive control in a cued-stimulus presentation paradigm. The focus of the present study was the fluctuation with which older and young adults engaged in different modes of cognitive control and how this variability relates to processes of working memory. Overall, young and older adults performed similarly in their level of intraindividual variability of proactive control in the computerized AX-CPT. However, an independent test of working memory (the Letter Number Sequencing task) revealed to be statistically associated with the measures of intraindividual variability of proactive control. The present findings overall provide preliminary evidence that working memory processes are involved in age-related declines in cognitive control.

References

- [1] Hasher, L., & Zacks, R. T. (1988). Working Memory, Comprehension, and Aging: A Review and a New View. *Psychology of Learning and Motivation*, 193–225. [https://doi.org/10.1016/s0079-7421\(08\)60041-9](https://doi.org/10.1016/s0079-7421(08)60041-9)
- [2] Moscovitch, M., & Winocur, G. (1995). Frontal lobes, memory, and aging. *Annals of the New York Academy of Sciences*, 769(1), 119–150. <https://doi.org/10.1111/j.1749-6632.1995.tb38135.x>
- [3] Salthouse, T. A. (1990). Working memory as a processing resource in cognitive aging. *Developmental Review*, 10(1), 101–124. [https://doi.org/10.1016/0273-2297\(90\)90006-p](https://doi.org/10.1016/0273-2297(90)90006-p)
- [4] Herman, T., Mirelman, A., Giladi, N., Schweiger, A., & Hausdorff, J. M. (2010). Executive control deficits as a prodrome to falls in healthy older adults: A prospective study linking thinking, walking, and falling. *The Journals of Gerontology: Series A: Biological Sciences and Medical Sciences*, 65(10), 1086–1092. <https://doi-org.lib-ezproxy.concordia.ca/10.1093/gerona/glp077>
- [5] Studenski, S. (2011). Gait Speed and Survival in Older Adults. *JAMA*, 305(1), 50. <https://doi.org/10.1001/jama.2010.1923>
- [6] Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., & Howerter, A. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology*, 41(1), 49–100. <https://doi-org.lib-ezproxy.concordia.ca/10.1006/cogp.1999.0734>
- [7] Friedman, N. P., & Miyake, A. (2017). Unity and diversity of executive functions: Individual differences as a window on cognitive structure. *Cortex*, 86(1), 186–204. <https://doi.org/10.1016/j.cortex.2016.04.023>
- [8] Glisky, E. L., Alexander, G. E., Hou, M., Kawa, K., Woolverton, C. B., Zigman, E. K., Nguyen,

L. A., Haws, K., Figueiredo, A. J., & Ryan, L. (2020). Differences between young and older adults in unity and diversity of executive functions. *Aging, Neuropsychology, and Cognition*, 1–26. <https://doi.org/10.1080/13825585.2020.1830936>

[9] Zuber, S., Ihle, A., Loaiza, V. M., Schnitzspahn, K. M., Stahl, C., Phillips, L. H., Kaller, C. P., & Kliegel, M. (2019). Explaining age differences in working memory: The role of updating, inhibition, and shifting. *Psychology & Neuroscience*, 12(2), 191–208. <https://doi-org.lib-ezproxy.concordia.ca/10.1037/pne0000151>

[10] Braver, T. S. (2012). The variable nature of cognitive control: a dual mechanisms framework. *Trends in Cognitive Sciences*, 16(2), 106–113. <https://doi.org/10.1016/j.tics.2011.12.010>

[11] Braver, T. S., Barch, D. M., Keys, B. A., Carter, C. S., Cohen, J. D., Kaye, J. A., Janowsky, J. S., Taylor, S. F., Yesavage, J. A., Mumenthaler, M. S., Jagust, W. J., & Reed, B. R. (2001). Context processing in older adults: Evidence for a theory relating cognitive control to neurobiology in healthy aging. *Journal of Experimental Psychology: General*, 130(4), 746–763. <https://doi.org/10.1037/0096-3445.130.4.746>

[12] Engle, R. W., & Kane, M. J. (2004). Executive Attention, Working Memory Capacity, and a Two-Factor Theory of Cognitive Control. *The psychology of learning and motivation: Advances in research and theory*, 44(5), 145–199.

[13] Wiemers, E. A., & Redick, T. S. (2017). Working memory capacity and intra-individual variability of proactive control. *Acta Psychologica*, 182(1), 21–31. <https://doi.org/10.1016/j.actpsy.2017.11.002>

[14] Mella, N., Fagot, D., Lecerf, T., & de Ribaupierre, A. (2015). Working memory and intraindividual variability in processing speed: A lifespan developmental and individual-differences study. *Memory & Cognition*, 43(3), 340–356. <https://doi-org.lib-ezproxy.concordia.ca/10.3758/s13421-014-0491-1>

[15] Myerson, J., & Hale, S. (1993). General slowing and age invariance in cognitive processing: The other side of the coin. *Adult information processing: Limits on loss*, 115–141.

[16] Salthouse, T. A. (1993). Attentional blocks are not responsible for age-related slowing. *Journal of Gerontology*, 48(6), 263–270. <https://doi.org/10.1093/geronj/48.6.p263>

[17] Allaire, J. C., & Marsiske, M. (2005). Intraindividual variability may not always indicate vulnerability in elders' cognitive performance. *Psychology and Aging*, 20(3), 390–401. <https://doi-org.lib-ezproxy.concordia.ca/10.1037/0882-7974.20.3.390>

[18] Kandasamy, K. (2018). A Postural Paradigm for the Dual Mechanisms of Control Model (Unpublished bachelor's thesis). Concordia University, Montreal, Canada.

[19] Nasreddine, Z. S., Phillips, N. A., Badirian, V., Charbonneau, S., Whitehead, V., Collin, I., Cummings, J. L., & Chertkow, H. (2005). The Montreal Cognitive Assessment, MoCA: a brief screening tool for mild cognitive impairment. *Journal of the American Geriatrics Society*, 53(4), 695–699. <https://doi.org/10.1111/j.1532-5415.2005.53221.x>

[20] Wechsler, D. (2008). Wechsler Adult Intelligence Scale: WAIS-IV Technical and Interpretive

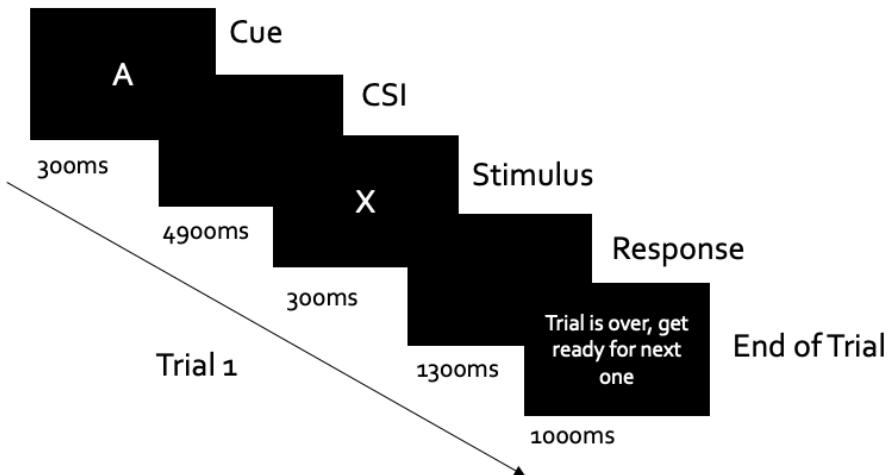
Manual. San Antonio, TX: The Psychological Corporation.

- [21] Braver, T. S., Paxton, J. L., Locke, H. S., & Barch, D. M. (2009). Flexible neural mechanisms of cognitive control within human prefrontal cortex. *Proceedings of the National Academy of Sciences*, 106(18), 7351–7356. <https://doi.org/10.1073/pnas.0808187106>
- [22] Gonthier, C., Macnamara, B. N., Chow, M., Conway, A. R. A., & Braver, T. S. (2016). Inducing Proactive Control Shifts in the AX-CPT. *Frontiers in Psychology*, 7(1822), 1–12. <https://doi.org/10.3389/fpsyg.2016.01822>
- [23] Paxton, J. L., Barch, D. M., Racine, C. A., & Braver, T. S. (2008). Cognitive control, goal maintenance, and prefrontal function in healthy aging. *Cerebral Cortex*, 18(5), 1010–1028. <https://doi.org.lib-ezproxy.concordia.ca/10.1093/cercor/bhm135>
- [24] Moscovitch, M., & Winocur, G. (1992). The neuropsychology of memory and aging. *The handbook of aging and cognition*, 315–372.
- [25] Raz, N. (2000). Aging of the brain and its impact on cognitive performance: Integration of structural and functional findings. *The handbook of aging and cognition*, 1–90.
- [26] Emery, L., Myerson, J., & Hale, S. (2007). Age differences in item manipulation span: The case of letter-number sequencing. *Psychology and Aging*, 22(1), 75–83. <https://doi.org/10.1037/0882-7974.22.1.75>
- [27] Ryan, J. (2000). Age effects on Wechsler Adult Intelligence Scale-III subtests. *Archives of Clinical Neuropsychology*, 15(4), 311–317. [https://doi.org/10.1016/s0887-6177\(99\)00019-0](https://doi.org/10.1016/s0887-6177(99)00019-0)
- [28] Rabbitt, P. (1979). How old and young subjects monitor and control responses for accuracy and speed. *British Journal of Psychology*, 70(2), 305–311. <https://doi.org/10.1111/j.2044-8295.1979.tb01687.x>
- [29] Smith, G. A., Poon, L. W., Hale, S., & Myerson, J. (1988). A regular relationship between old and young adults' latencies on their best, average and worst trials. *Australian Journal of Psychology*, 40(2), 195–210. <https://doi.org/10.1080/00049538808259082>
- [30] Redick, T. S. (2014). Cognitive control in context: Working memory capacity and proactive control. *Acta Psychologica*, 145(1), 1–9. <https://doi.org/10.1016/j.actpsy.2013.10.010>
- [31] McVay, J. C., & Kane, M. J. (2009). Conducting the train of thought: Working memory capacity, goal neglect, and mind wandering in an executive-control task. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 35(1), 196–204. <https://doi.org/10.1037/a0014104>
- [32] Redick, T. S., & Engle, R. W. (2011). Rapid communication: Integrating working memory capacity and context-processing views of cognitive control. *Quarterly Journal of Experimental Psychology*, 64(6), 1048–1055. <https://doi.org/10.1080/17470218.2011.577226>
- [33] Unsworth, N., Redick, T. S., Spillers, G. J., & Brewer, G. A. (2012). Variation in working memory capacity and cognitive control: Goal maintenance and microadjustments of control. *Quarterly Journal of Experimental Psychology*, 65(2), 326–355. <https://doi.org/10.1080/17470218.2011.597865>

- [34] Unsworth, N., Redick, T. S., Lakey, C. E., & Young, D. L. (2010). Lapses in sustained attention and their relation to executive control and fluid abilities: An individual differences investigation. *Intelligence*, 38(1), 111–122. <https://doi.org/10.1016/j.intell.2009.08.002>
- [35] Paxton, J. L., Barch, D. M., Storandt, M., & Braver, T. S. (2006). Effects of environmental support and strategy training on older adults' use of context. *Psychology and Aging*, 21(3), 499–509. <https://doi.org/10.1037/0882-7974.21.3.499>
- [36] Salthouse, T.A. (1986). The processing-speed theory of adult differences in cognition. *Psychological Review*, 103(3), 403-428. <https://doi-org.lib-ezproxy.concordia.ca/10.1037/0033-295X.103.3.403>

Appendix A

Computerized AX-CPT Paradigm



Note. Experimental paradigm of an AX trial in the computerized AX-CPT design. An “A” cue is presented for 300 ms, followed by a Cue-Stimulus Interval (CSI) of 4900 ms. An “X” stimulus is then presented for 300 ms, and a response window of 1300ms is given to participants. A screen for 1000 ms with the text “Trial is over, get ready for next one” marks the end of the AX trial.

Appendix B

Computerized AX-CPT Mean Reaction Times

Trial Type	AX		AY		BX		BY	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Young Adults	531.00	98.59	646.00	89.46	459.00	129.23	452.00	79.69
Older Adults	638.00	85.22	864.00	113.97	584.00	99.94	581.00	109.80
Overall	589.00	105.52	766.00	150.00	525.00	129.76	523.00	116.26

Average reaction time (ms) across all trial types as a function of age group on the computerized AX-CPT.

Appendix C

Correlations, Means, and Standard Deviations for Working Memory Regression Analyses

Variables	1	2	3	4	5
1. LNS Total Score	—				
2. AX Change in Accuracy	-.018	—			
3. BX Change in Accuracy	-.242	.308	—		
4. IIV of PBI (reaction time)	.007	-.120	-.178	—	
5. IIV of PBI (error rate)	.023	-.563	.427	.042	—
	<i>M</i>	19.26	-.04	-.01	.04
	<i>SD</i>	2.08	.18	.14	.08
					.45

Note. $N = 50$. Predictors of Letter Number Sequencing (LNS) total scores (out of 30) were set as the mean change in accuracy on AY trials, the mean change in accuracy on BX trials, the intraindividual variability (IIV) of Proactive Behavioral Index (PBI) as a function of reaction time (ms), and the intraindividual variability (IIV) of Proactive Behavioral Index (PBI) as a function of corrected error rates.

The Impact of Multisensory and Cognitive Load on Older Adults' Complex Balance Performance *

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With age, older adults are increasingly subject to sensory, cognitive, and motor declines, placing them at a greater risk of falls. The impact of sensory impairment and increased cognitive load on older adults' complex balance performance has remained understudied. The purpose of the current study was to determine the impact that sensory loss and increased cognitive load have on older adults' single and dual-task balance performance. A total of 27 participants (11 healthy older adults (HOA), 16 age-related hearing loss (ARHL) between the ages of 56 to 90 were recruited. The participants underwent auditory, visual, and cognitive assessments, and their balance performance was tested under both single and dual-task conditions. A statistically significant main effect of balance task complexity was found, in which anterior-posterior sway increased with the addition of a sensory challenge and a cognitive load. There was no statistically significant interaction between hearing status and task complexity, thus indicating that individuals with ARHL performed similarly to HOA across balance conditions. Additionally, older adults with ARHL demonstrated a cognitive facilitation effect when considering cognitive dual-task costs. Those with ARHL had better cognitive performance in the dual-task in comparison to the single-task condition. An ineffective dual-task strategy of prioritizing cognition over balance may increase older adult's risk of falling.

Between 2015 and 2050, the world population aged 60 and over is expected to nearly double, reaching approximately 2 billion older adults [1]. With an increasingly older population, understanding the typical aging process is vital. As a function of age, older adults are subject to changes within their sensory [2, 3], cognitive [4, 5, 6], and balance [7] processes. Researchers have found that decreases in overall physical functioning are linked to an increased risk of falls, with approximately 46% of adults over the age of 85 having self-reported increases in instability [8]. Alarmingly, nearly 20 to 30% of older adults fall each year, and falls remain the leading cause of injury-related hospitalizations among Canadian seniors [9]. Understanding the functions that decline with increasing age and how they affect one another is essential to research regarding healthy aging. The current research was therefore designed to determine how older adults' balance and cognitive performance is impacted by hearing loss and the addition of a sensory challenge and cognitive load.

Sensory Loss and Aging

In Canada, an estimated 78% of older adults between the ages of 60 to 79 have at least slight hearing loss [9]. By age 80, nearly 90% of older adults have some form of hearing impairment [3]. Brennan et al. (2005) found that nearly one fifth of older adults reported hearing and vision loss and that both the number and the severity of dual-sensory impairment influenced participants'

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performance on a series of activities of daily living. Additionally, researchers have highlighted that an individual's risk of mortality increases as a result of dual-sensory loss [10]. Dual-sensory loss may therefore exacerbate the decline in functioning in older adults, as they can no longer compensate for the loss through greater recruitment of other senses [11]. In contrast, Alfaro and colleagues found that older adults with dual-sensory loss fared just as well on measures of cognition and activities of daily living, compared to individuals with sensory impairment in only one modality. These contradictory results highlight the importance of determining the impact of dual-sensory loss on cognitive performance [12].

Sensory Loss and Cognition

Hearing loss is negatively associated with cognition, since older adults with greater hearing loss have been found to perform more poorly on measures of cognitive functioning (i.e., working memory, inhibition, processing speed) [13, 14]. Hearing loss has been found to add a significant cognitive load and independently increase the rate of cognitive decline and cognitive impairment. More specifically, older adults presenting with greater hearing loss demonstrate greater annual decreases in cognitive performance [15, 16]. One interpretation of these associations between hearing and cognition is the Cognitive Load Hypothesis, which posits that older adults with hearing loss allocate greater cognitive resources to compensate for a degraded auditory signal, which diverts resources away from other cognitive tasks, potentially resulting in cognitive reserve depletion [17].

Dual-sensory loss is also seen to negatively impact cognitive functioning, in that with the additional sensory impairment, there is a notable decrease in measures of cognitive performance, such as short-term memory and decision making [18]. Cognitive impairment is therefore more prevalent in older adults with multiple sensory impairments [19]. For example, Davidson and Guthrie discovered that among their sample of older adults, the poorest cognitive performance was found in individuals with significant hearing and vision loss in comparison to those with either a single sensory impairment, or no sensory impairment [20]. Additionally, dual-sensory loss is found to increase an individual's risk of developing dementia [21].

Importantly, cognitive processes and frontal regions of the brain are more often utilized in older adults to compensate for sensory loss. For example, Du and colleagues' fMRI study highlighted that older adults show increased activation in frontal speech motor areas of the brain during a syllable identification task, to compensate for hearing loss [22]. Additionally, older adults with age-related vision or hearing loss demonstrate an increased reliance on cognitive resources for everyday functioning [23]. Thus, due to their increased utilization of cognitive processes and recruitment of frontal regions, older adults may have fewer cognitive resources to distribute efficiently to motor, or dual-task conditions.

Sensory Loss and Balance

In addition to the impact of sensory loss on cognition, hearing and vision loss are also positively associated with several measures of postural control. An individual's ability to balance efficiently is heavily influenced by the contribution of multiple sensory systems (i.e., proprioceptive, vestibular, auditory [24]. Among these sensory systems, which are subject to decline gradually with healthy aging, hearing loss has been shown to increase fall risk and instability [15]. Older adults with hearing loss are more likely to demonstrate poor postural performance, and this effect appears to be stronger for greater levels of hearing loss [25]. For example, Lin and Ferrucci found

that with every 10-decibel (dB) increase in hearing loss, an individual was 1.4 times more likely to have reported a fall within the 12 preceding months [15]. Older adults with hearing loss will often struggle more in regaining their balance when performing a secondary task in comparison to their normal hearing counterparts [26].

Older adults with significant vision impairment also demonstrate increased postural instability and a greater risk of falling [27]. Hallot and colleagues found greater postural instability, specifically anterior-posterior changes (i.e., greater forward and backward sway) when using stimulated vision impairment goggles. Such changes are often determined using balance platforms that measure centre of pressure displacement [28]. Additionally, a more extreme eyes-closed visual manipulation has been found to increase centre of pressure displacements [29]. This signals greater postural sway and unsteadiness among the older adults. Thus, sensory loss as a result of typical aging, negatively impacts both cognition and balance performance and may influence the utilization of cognitive resources and recruitment of frontal brain regions.

Cognition and Aging

In addition to sensory and motoric changes, typical aging is accompanied by neurobiological changes which in turn affect cognitive processes [4]. Consequently, cognitive performance on tests of memory and attentional control decreases as a function of age [6]. Older adults also experience a decline in executive functioning with increasing age. Executive functions can be defined as a set of cognitive skills required for planning, monitoring, and executing a sequence of goal-directed complex actions [5]. These processes include inhibition, attentional switching or divided attention, and working memory capacity. In particular, older adults' decline in processing speed ability creates a greater difficulty in tasks requiring divided attention (e.g., having a conversation while walking) [30].

Interesting however, is the idea that older adults may use compensatory mechanisms to reduce the age-related gap in cognitive performance. When comparing the performance of younger and older adults in a cognitive task where younger adults are seen to recruit regions of one hemisphere, older adults may utilize additional frontal brain regions and demonstrate bilateral activation [31]. Older adults who show bilateral activation may perform more similarly to their younger aged counterparts on cognitive tests.

Individuals with mild cognitive impairment may also demonstrate this pattern of activation [32] Approximately 19% of older adults over the age of 65 will be affected by Mild Cognitive Impairment (MCI), which is an intermediate state between normal aging and dementia [33]. Nearly 50% of adults with MCI will progress to dementia within five years of the initial MCI diagnosis [34]. Characterized by greater cognitive decline than would be typically expected for an individual's age and level of education, MCI does not appear to largely interfere with activities of everyday life in comparison to dementia, which affects daily functioning [34]. Importantly, greater decline in cognitive ability (i.e., dementia or severe cognitive impairment) can lead to increased difficulty in performing everyday activities, and an increased risk of falling, thus highlighting the importance of maintaining healthy cognitive and motor functioning [35].

Cognition and Balance

Additionally, there is a relationship between changes in older adults' cognitive functioning and subsequent balance performance [36]. Older adults demonstrate a reduced ability in allo-

cating their limited cognitive resources among multiple tasks [37]. Impaired performance on tasks involving divided attention can be determined using a dual-task paradigm [38]. A dual-task paradigm is used to assess how attentional resources are allocated among particular tasks of interest [39]. The paradigm allows for the comparison of the performance of two tasks completed individually with the performance of both tasks completed simultaneously. Dual-task performance can be estimated by considering dual-task costs, which are determined by subtracting the performance on the dual-task condition by performance on the single-task condition, and further dividing it by the single-task performance [38]. A positive dual-task cost value indicates reduced dual-task ability or maladaptive performance when performing both tasks. A negative dual-task value would be indicative of an improvement in dual-task performance compared to single-task performance. Participants with a negative dual-task cost would therefore be demonstrating a facilitative performance [38]. Older adults are more likely to demonstrate greater dual-task costs (i.e., positive dual-task cost value) in comparison to their younger-aged counterparts. Brustio and colleagues highlighted a significant main effect of age, where older adults demonstrated greater dual-task costs in the cognitive domain, compared to younger adults, when undergoing both motor and cognitive tasks simultaneously [40]. Huxhold and colleagues found the greatest dual-task costs in the motor domain, in older adults undergoing a balance and working memory load paradigm [41].

While most research has highlighted the idea that postural stability is compromised by the addition of a cognitive load or cognitive impairment [41, 42], performance on a dual task may change as a result of postural prioritization. The posture-first principle states that when undergoing a cognitive-motor dual task, older adults will allocate more resources to the balance task to avoid falling, which as a result would impair their performance on the cognitive task [43]. Contradictory evidence where postural performance declines as function of a dual-task paradigm may be explained through alterations in the complexity of both the balance and cognitive tasks [29, 44].

Cognitive decline is negatively associated with complex balance performance, where older adults presenting with greater cognitive decline are more likely to demonstrate increased postural instability [36]. For example, Shin and colleagues found that older adults with MCI demonstrated greater mediolateral sway than their cognitively normal counterparts. Furthermore, increases in cognitive load also negatively impact older adults balance performance [45]. Older individuals will typically demonstrate greater postural instability with the addition of a cognitive load [41, 42]. For example, researchers using a Wii balance board to measure cognitive-motor dual-task performance found greater sway with increased cognitive load (i.e., serial subtraction task) compared to single-task balance conditions [46].

To summarize the relevant literature, there is abundance of evidence that sensory modalities (i.e., vision and hearing) [21], cognition, [4], and balance [7] decline as a function of increasing age. Additionally, these three functions appear to be highly interrelated. Sensory loss is negatively related to cognition, where increases in hearing loss or multiple sensory impairments are associated with worsened performance in multiple tests of cognitive functioning [16]. Further, sensory loss is positively related to balance performance where greater levels of hearing loss are associated with increases in postural instability [15]. Lastly, greater cognitive decline and added cognitive loads appear to negatively impact older adults' balance performance [36, 42]. However, less is known about the impact of multisensory impairments and cognitive load on older adults' complex balance performance.

Present Study

The purpose of the current study was to determine the impact that sensory losses and increased cognitive load have on older adults' single- and dual-task balance performance. More specifically, we were interested in the impact of hearing loss and simulated visual impairment on older adults' cognitive functioning and complex balance performance. We therefore hypothesized that with the addition of both a cognitive load and visual challenge, complex balance performance would decrease. Next, we hypothesized that individuals with age-related hearing loss ARHL would demonstrate worsened complex balance performance with increasing complexity relative to healthy older adults HOA. Regarding postural dual-task costs, we hypothesized that under dual-task conditions, all individuals would demonstrate decreased balance performance. Additionally, we expected that postural dual-task costs would be greater in the ARHL group than in the HOA group. Lastly, regarding cognitive dual-task costs, we hypothesized that the presence of hearing loss and increased cognitive load while balancing would result in reduced cognitive performance in the dual-task condition in comparison to the single-task condition. We therefore hypothesized that when undergoing dual-task conditions individuals would demonstrate both postural and dual-task costs, however likely not to the same degree.

Method

Participants

The present sample was drawn from an existing data set of 27 older adults between the ages of 56 and 90 ($M = 74.74$, $SD = 9.51$) Among the participants 16 were female and 11 were male. Within the sample, 11 participants were HOA and 16 had ARHL. Based on an a priori G*POWER [47] analysis, a minimum of 26 participants were needed to achieve a medium effect size of 0.5 with a power of 0.8, thus the present sample size was deemed adequate.

Older adults were recruited from the engAGE Living Lab, located in the Cote Saint-Luc Cavendish Mall. Data collection occurred during a 3.5-week residency, which was part of a larger funded project aiming to provide an interactive space to help older adults combat social isolation and participate in collaborative research. Older adults interested in the research would simply walk into the centre and sign up to be a part of the study. The residency allowed for data to be collected in a natural setting, allowing for a more diverse aging population to be recruited. Participants were included if they were 50 years or older and had normal or corrected-to-normal vision. The cut-off of 50 years was chosen as many individuals begin to undergo hearing loss screening as of that age [48]. Participants were excluded if they had any vestibular disorders, artificial limbs, or any neurodegenerative diseases. Such exclusions were determined through self-report measures.

Measures

Cognition

The Montreal Cognitive Assessment (MoCA) was used as a measure of global cognition [49]. The MoCA is comprised of eight sections, each assessing a different domain of cognitive functioning. More specifically the test includes the categories of visuospatial/executive naming, attention, language, abstraction, delayed recall, and orientation. A total of 30 points were available, and participants were given an extra point if they had less than 12 years of education. A lower score on the MoCA indicates poorer cognitive performance. More specifically, a score of less than 26 is suggestive of the presence of MCI. The MoCA has good internal consistency (Cronbach's $\alpha =$

.83) [49]. The duration of the test was approximately 10 minutes. Participants with MoCA scores indicative of Mild Cognitive Impairment remained eligible.

The serial 7s subtraction task was used as a measure of working memory [50]. The same task was also given concurrently within the dual task balancing conditions. Participants completed this task in a seated position wearing control clear goggles, while fixating their view on a target icon on the wall in front of them (single-task cognition). Participants were required to count backwards by 7s from 175 for 30 seconds. Participants gave their answers verbally and the percentage of correct responses was recorded and used as an indicator of cognitive performance. In the case of a calculation error, participants were permitted to continue and the correct subtractions from that point onward were counted. The internal consistency of a sample of older adults was found to be a Cronbach's α of 0.764 [50].

Audition

ShoeBOX Audiometry (Clearwater Clinical Limited) was used to measure hearing acuity. ShoeBOX Audiometry is clinically validated for auditory assessment outside of a sound-attenuated booth [51]. Specially calibrated DD 450 Headphones were used to deliver pure tones, which ranged in frequency (e.g., 200, 500, 1000, 2000, 3000, 4000, and 8000 Hz) and were presented to the left followed by the right ear. Participants responded using a touch-sensitive tablet and were instructed to click on a blue disk to begin each trial. Depending on whether or not they heard a presented tone, they were told to drag the blue disk to the green (heard) speaker or the red (not heard) speaker. ShoeBOX Audiometry has been validated against standardized pure tone testing methods and was chosen to facilitate the unconventional setting of our testing locale.

Participants were classified according to grades of hearing acuity which were calculated using pure tone averages (PTA) for the better ear as an average of four frequencies (e.g., 500, 1000, 2000, 4000 hertz (Hz)). A higher PTA would indicate poorer hearing acuity; that is a greater sound intensity (dB HL) would be needed to detect that tone [52]. As recruitment for this project did not involve actively seeking out individuals with hearing loss, we created two broad categories of hearing status by pooling all individuals with PTAs above 25 dB hearing loss (HL) in the better ear (i.e., ARHL: > 25 dB HL, HOA: < 25 dB HL) in keeping with the World Health Organization classification scheme for hearing impairment [1].

Complex Balance Performance

To assess postural stability, a Nintendo Wii Balance Board (Nintendo, Kioto Japan) was paired with custom software (RombergLab) that recorded centre of pressure displacements [53]. The Wii Balance Board is similar to typical force plates as it contains four gauge-based load sensors. To measure postural instability, the following centre of pressure displacements were considered. Medial-lateral sway amplitude (mm), a distance between the farthest point leftward and rightward. Anterior-posterior sway amplitude (mm), the distance between the most forward and backward point. Lastly total path length was considered. Total path length included both medial-lateral and anterior-posterior amplitude sway measures and is the total distance travelled in millimetres. Data for center of pressure displacements are shown in Appendix A.

During the balance conditions involving the addition of a sensory challenge, 20/80 vision impairment goggles were used. The vision impairment goggles were designed to mimic the visual acuity of an individual with vision impairment and could be worn over glasses if relevant. A simu-

lated visual acuity of 20/80 was chosen as it is the level of visual impairment needed to qualify for vision rehabilitation in Quebec. As a more extreme manipulation of visual impairment, an eyes-closed condition was included. For balance conditions not involving a visual manipulation, clear control goggles were used, which did not impair vision. All participants underwent five balance conditions: balance eyes closed, balance and control goggles, balance control goggles and serial 7s task, balance and 20/80 vision impairment goggles, balance 20/80 vision impairment goggles and serial 7s task. The five conditions were used to determine how both a sensory challenge and a cognitive load influence the centre of displacement measures (i.e., total path length, medial-lateral (ML) amplitude, anterior-posterior (AP) amplitude). To account for potential learning and fatigue effects, the order of the balance test conditions was counterbalanced.

General Procedure

Prior to participating, individuals were required to sign a consent form Once having completed the measures of global cognition (MoCA), single cognitive task (serial 7s subtraction), and hearing acuity (ShoeBOX), participants underwent each of the five balance conditions. Once all of the measures were completed, participants were debriefed, and contact information was provided in the case of follow up questions from the participants.

Results

Data Integrity

Prior to conducting the statistical analyses, the data were examined for normality, outliers, skewness, and kurtosis. Specifically, descriptive statistics were assessed (see Table 1). The scores from hearing, cognitive, and balance measures were converted to z-scores and inspected to ensure that they fell within 3 standard deviations of the mean. One participant's scores consistently did not meet this cut-off for balance and therefore their case was removed from the analyses. Additionally, there were no missing values within the dataset. The final sample size was 27.

Table 1

Means (SD) for Participant Characteristics

	Total Sample (N=27)	HOA (n=11)	ARHL (n=16)	t	p
Age (years)	74.74 (9.51) ^a	71.27 (11.30) ^a	77.13 (7.53) ^a	-1.62	.118
Gender (male, female)	11, 16	3, 8	8, 8		
MoCA (max. = 30)	22.96 (3.79) ^a	24.00 (3.29) ^a	22.25 (4.04) ^a	1.19	.246
Hearing Acuity (PTA)	30.46 (12.25) ^a	19.20 (5.82) ^a	38.20 (8.99) ^a	-6.16	.000**

Note. ^a Mean (standard deviation). MoCA= Montreal Cognitive Assessment. MoCA scores of < 26 are suggestive of MCI. PTA= pure tone average.

A higher pure tone average value indicates poorer hearing acuity. Independent samples *t*-tests were used to compare HOA and ARHL.

p* < .05 *p* < .001

The assumptions needing to be met to conduct a mixed analysis of variance (ANOVA) and independent sample t-tests were also considered. The assumption of independence was met, indicating that the sample was both randomly and independently sampled. Regarding the repeated measures ANOVA, Mauchly's test of sphericity was not significant, therefore we interpreted the results using a Greenhouse-Geisser correction. Regarding independent samples t-tests, Levene's test for equal variances was not significant, and we therefore interpreted the results using equal variances assumed.

Main Results

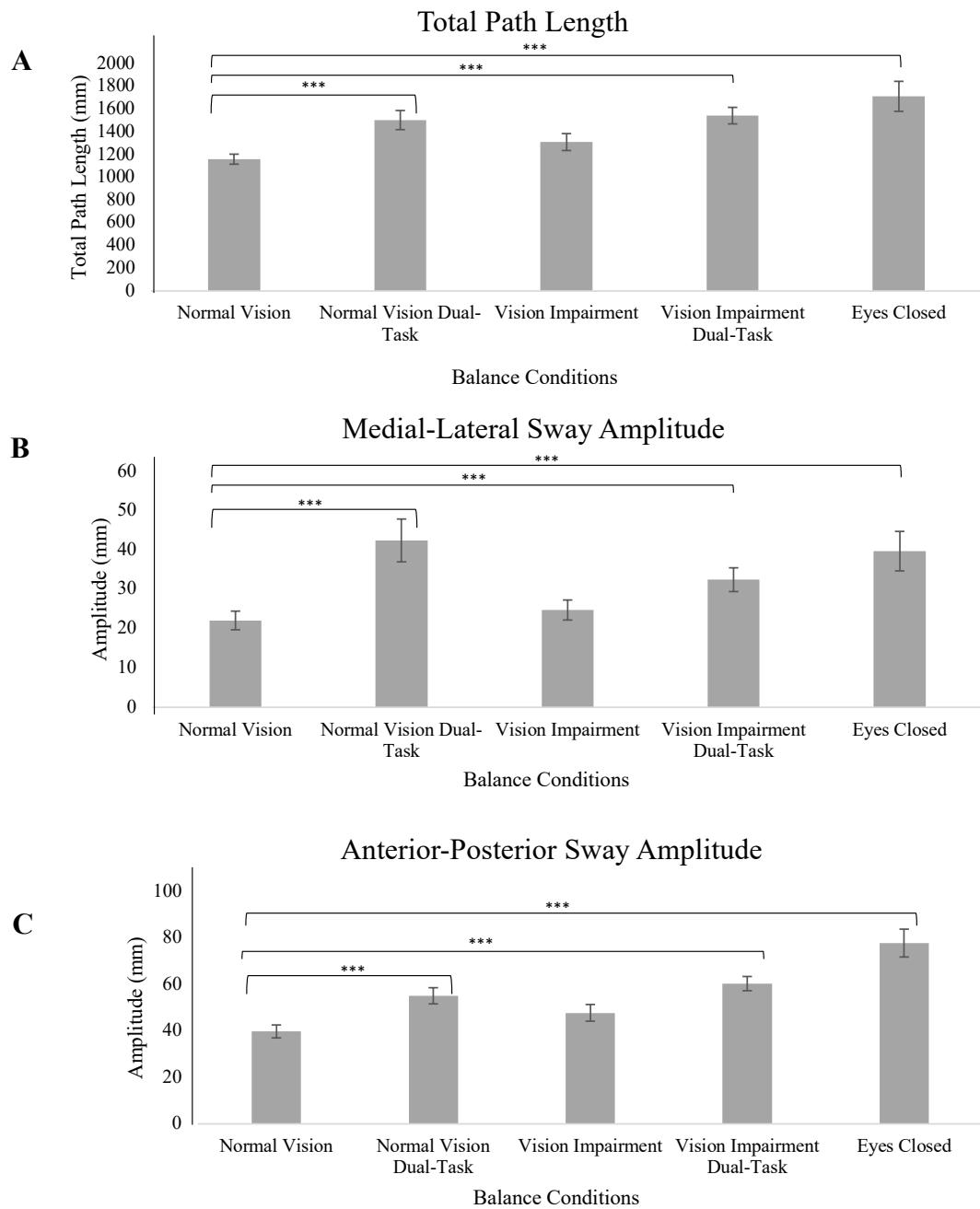
Prior to conducting hypotheses driven analyses, independent samples t-tests were used to assess group differences in cognitive performance. Regarding MoCA scores, 9 participants had scores of less than 26 ($M = 20.94, SD = 2.92$) and 18 participants had scores greater than 26 ($M = 27.00, SD = 1.00$). MoCA Scores were lower in the ARHL group ($M = 22.25, SD = 4.04$) in comparison to the HOA group ($M = 24.00, SD = 3.29$). The difference in MoCA scores between groups was not found to be statistically significant ($t(25) = 1.19, p = .246$). Participant characteristics are shown in Table 1.

Hypothesis 1

First, we hypothesized that with the addition of both a sensory challenge and cognitive load, complex balance performance would decrease. To evaluate the effect of increasing task complexity on balance performance, we conducted a 2×5 mixed analysis of variance ANOVA. Specifically, the within-subjects factor was Task Complexity with 5 levels: normal vision, normal vision & serial 7s, vision impairment, vision impairment & serial 7s, eyes closed. Additionally, Hearing Status was used as a between-subjects factor. A statistically significant main effect of task complexity was observed in anterior-posterior sway amplitude, $F(2.23, 55.82) = 6.396, p = .002, \eta^2 = .204$. Total path length did not show a significant complexity effect, $F(1.80, 44.89) = 1.96, p = .157, \eta^2 = .073$, but the same task complexity factor approached significance for medial-lateral amplitude, $F(2.07, 51.84) = 2.626, p = .080, \eta^2 = .095$. Data for all complexity conditions are shown per balance parameter in Figure 1. Post-hoc pairwise comparisons using Bonferroni corrections were used to identify which balance conditions differed specifically from one another. We found that when using the normal vision single-task balance condition as a reference point, total path length increased with the addition of a cognitive load (i.e., normal vision dual-task; $MD = -344.05, SE = 60.86, 95\% CI [-531.38, -156.72], p < .001$), with a cognitive and sensory challenge (i.e., vision impairment + serial 7s: $MD = -383.126, SE = 53.75, 95\% CI [-548.57, -217.68], p < .001$), and with the complete removal of a visual input (i.e., eyes closed; $MD = -552.83, SE = 113.91, 95\% CI [-903.46, -202.20], p = .001$). No statistically significant difference was observed when comparing the normal vision condition to the vision impairment condition ($MD = -150.50, SE = 54.40, 95\% CI [-317.94, 16.95], p = .105$). We concluded the addition of a visual challenge alone did not impair balance performance in comparison to the normal vision baseline condition, possibly because there were adequate cognitive resources to compensate for the visual challenge.

Figure 1

Centre of Pressure Displacements for the Total Sample



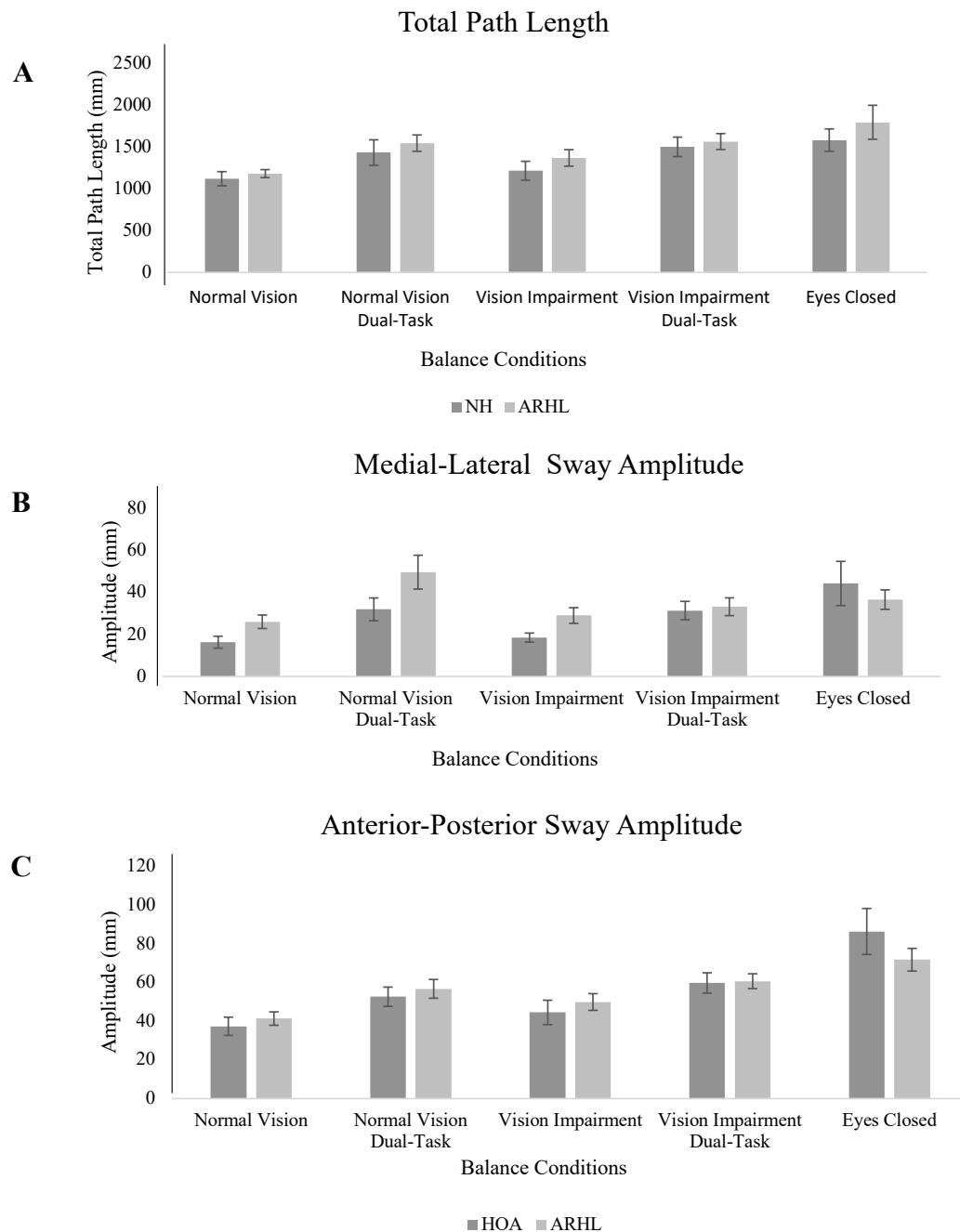
Hypothesis 2

Second, we hypothesized individuals with ARHL would demonstrate worsened complex balance performance with increasing task complexity. No statistically significant main effect of hearing status group was observed for total path length ($F(1, 25) = .43, p = .518, \eta^2 = .017$), medial-lateral amplitude ($F(1, 25) = 2.24, p = .147, \eta^2 = .082$), and anterior-posterior amplitude ($F(1, 25) = .03, p =$

.876, $\eta^2 = .01$). No statistically significant interaction between task complexity and hearing status was observed for total path length ($F(1.80, 44.89) = .32, p = .706, \eta^2 = .024$), medial-lateral amplitude ($F(2.07, 51.84) = .70, p = .506, \eta^2 = .027$), and anterior-posterior amplitude ($F(2.23, 55.82) = 1.24, p = .299, \eta^2 = .047$). Balance performance data separated by hearing status group are shown in Figure 2. We therefore concluded that individuals with hearing impairment did not have poorer balance performance with increased task complexity, as we had originally hypothesized.

Figure 2

Centre of Pressure Displacements by Hearing Status

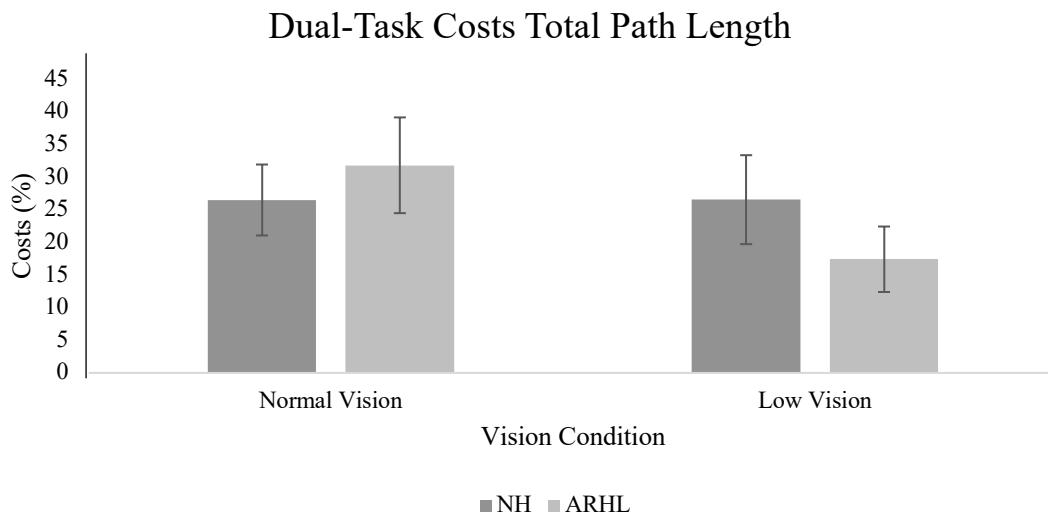


Hypothesis 3

Third, regarding postural dual-task costs, we hypothesized that under dual-task conditions, individuals would demonstrate decreased balance performance. Additionally, we expected that postural dual-task costs would be greater in the ARHL group in comparison to the HOA group. In partial support of our hypothesis, participants did demonstrate postural dual-task costs. Older adults with ARHL presented with 31.84% dual-task costs in the normal vision condition and 17.42% dual-task costs within the low vision condition. For HOA, dual-task costs were 26.52% and 26.56% for normal and low vision conditions respectively. Additionally, upon visual inspection, in the normal vision condition, the postural dual-task costs appear greater in the ARHL group in comparison to the HOA group (see Figure 3). However, results from an independent t-test revealed that the difference in dual-task costs within the normal vision condition when comparing the between the ARHL group and HOA groups was not statistically significant ($t(25) = -.52, p = .611$). The dual-task costs within the low vision condition did not support our hypothesis. With inspection alone, individuals in the ARHL group appeared to demonstrate fewer postural dual-task costs in comparison to the HOA group. This result however was not found to be statistically significant ($t(25) = 1.06, p = .299$). Within dual-task conditions, those with ARHL appear to allocate more attention to their balance in the low vision condition in comparison to the normal vision condition. A paired sample t-test revealed this result to approach significance ($t(15) = 2.03, p = .06$).

Figure 3

Postural Dual-task Costs by Vision Condition and Hearing Status



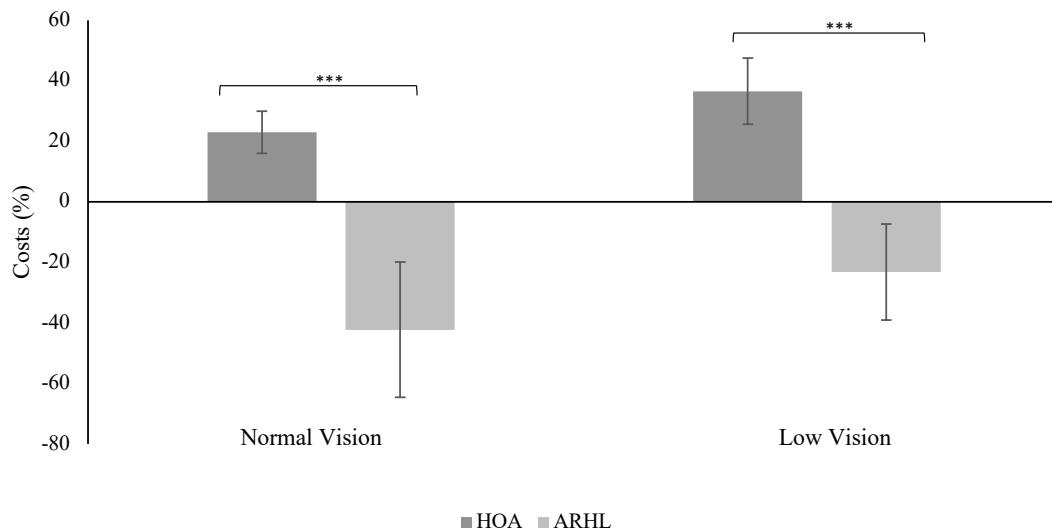
Hypothesis 4

Finally, regarding cognitive dual-task costs, we hypothesized that the presence of hearing loss and a cognitive load while balancing would result in significant cognitive dual-task costs. Contrary to what was hypothesized, not only did those with ARHL not demonstrate greater cognitive dual-task costs in comparison to HOA, but they appeared to demonstrate dual-task facilitation.

That is, ARHL participants showed superior cognitive performance (i.e., serial 7s subtraction) in the dual-task, compared to the single-task condition. This facilitation effect was also observed, however to a lesser degree in the low vision condition. Our hypothesis was therefore not supported. An independent samples t-test was conducted to compare the cognitive dual-task costs of participants with ARHL and HOA. Within the normal vision condition, we observed a statistically significant difference between the two groups in the magnitude of their cognitive dual-task facilitation ($t(25) = 2.23, p = .032, d = 0.68$) such that the ARHL group exhibited facilitation whereas the HOA group exhibited dual-task costs. Additionally, a statistically significant difference between ARHL and HOA groupings was observed in the low vision condition ($t(25) = 2.72, p = .012, d = 0.86$) again with the HOA group exhibiting costs and the ARHL group exhibiting facilitation (see Figure 4).

Figure 4

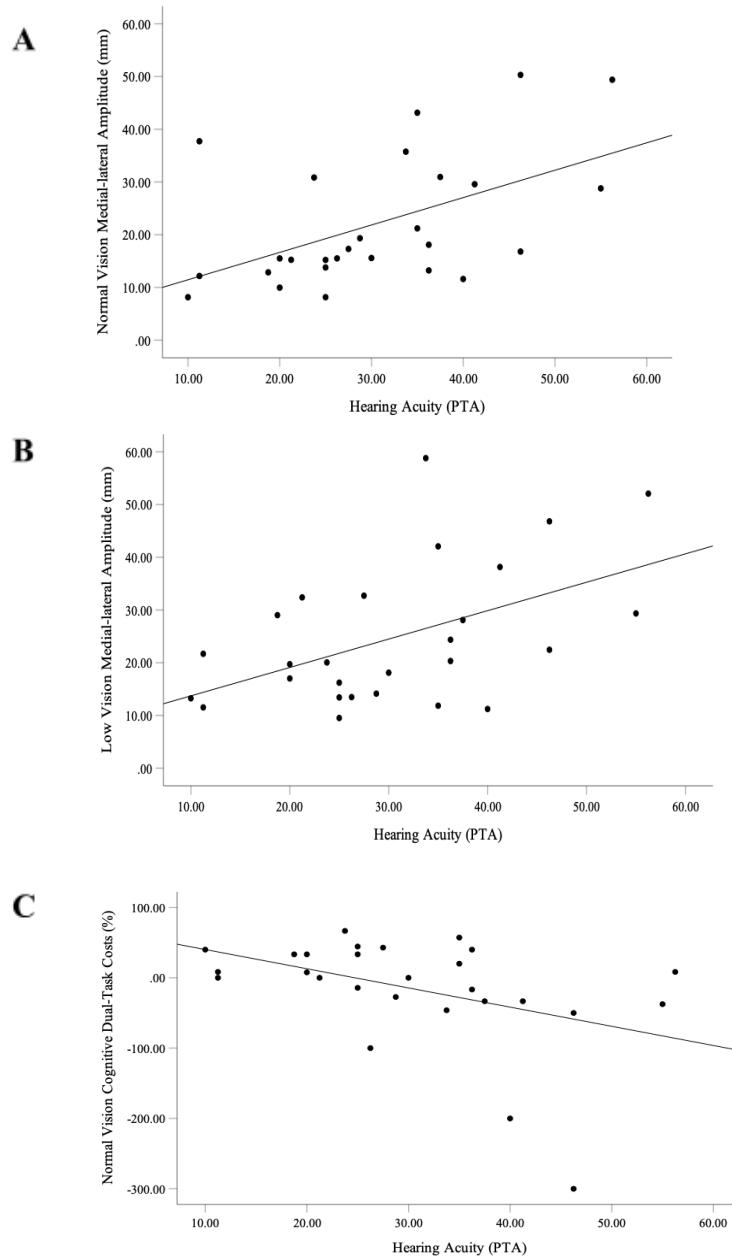
Cognitive Dual-task Costs by Vision Condition and Hearing Status



An additional series of correlations were conducted to examine the relationship between hearing loss and balance task complexity, and hearing loss and cognitive dual-task costs. Specifically, we aimed to determine the direction of the relationships. Regarding hearing status and task complexity, we found a statistically significant positive correlation between hearing acuity (PTA) and single-task low vision ($r(26) = .498, p = .008$) and single-task normal vision in the medial-lateral amplitude parameter ($r(26) = .517, p = .006$). A negative correlation was observed between hearing acuity (PTA) and cognitive dual-task costs in the normal vision condition ($r(26) = -.424, p = .027$; see Figure 5). Thus, indicating that participants with greater hearing loss demonstrated more greater facilitation in their cognitive performance when comparing normal vision single-task to dual-task. Additionally, we aimed to determine if MoCA scores influenced either hearing status or cognitive performance. MoCA scores were negatively correlated with age ($r(26) = -.428, p = .026$), indicating poorer cognitive functioning with increasing age. However, no statistically significant correlations were found between MoCA scores and hearing acuity (PTA), or with cognitive performance. Therefore, the participants degree of cognitive functioning did not appear to influence their auditory performance, nor their performance on the serial 7s task.

Figure 5

Associations Between Hearing Acuity and Medial-lateral Amplitude for Normal and Low Vision Conditions and Hearing Acuity and Normal Vision Dual-task Costs



Note. PTA = pure tone average. Positive dual-task costs indicate a maladaptive performance. Negative dual-task costs indicate a facilitative performance

Discussion

The aim of the current research was to determine the impact that sensory loss and cognitive load have on older adults' balance performance. Given that many of our analyses either did not reach or only approached significance, we must be careful when interpreting the results.

First, to determine whether there was an effect of task complexity on balance performance, we considered centre of pressure displacements for the entire sample. Overall, the whole sample appeared to demonstrate worsened postural performance with increased complexity of the balance conditions. Interestingly, adding a visual challenge alone did not appear to impact balance performance in comparison to the normal vision baseline condition. Performance instead was relatively similar among the two conditions. This may be due to the amount of time spent wearing the vision impairment goggles. The vision impairment goggles were only worn for 30 seconds at a time, which may not have been long enough to see an effect of sensory load alone. Additionally, the vision goggles only partially obscured vision, thus older adults may still have the cognitive capacity to compensate for the impairment. By contrast, with the addition of a cognitive or both a sensory challenge and a cognitive load, the participants centre of pressure displacements (i.e., total path length, AP amplitude, ML amplitude) increased. In support of our findings, researchers have highlighted that with increasing balance task complexity, or the addition of cognitive and sensory loads, individuals demonstrate poorer balance performance [46]. In contrast, Deviterne and colleagues found that with the addition of a cognitive load, participants demonstrated improved postural performance, highlighting how they may allocate more resources toward the postural task rather than the cognitive task [54]. Contradictory evidence to what we had found in our study further highlights the need for future replication by other researchers.

To address Hypothesis 2, the main effect of hearing status and the interaction of hearing status and task complexity was considered. We compared centre of pressure displacements among HOA individuals and those with ARHL. There was no statistically significant main effect of hearing status on task complexity. Thereby, the increase in task complexity did not appear to be impacted by hearing loss. Additionally, a statistically significant interaction was not observed, in that both normal hearing older adults and those with age-related hearing loss were similarly affected by the complexity manipulation. This finding was not in support of the second hypothesis, which predicted that those with ARHL would be more negatively affected by increased balance task complexity. This may be due to the distribution of hearing loss severity in the current ARHL sample, which was largely in the mild range. More specifically, only a few individuals had moderate hearing loss and no individuals had severe hearing loss. Perhaps having more individuals with a greater degree of hearing loss, as well as a more even distribution among hearing loss categories would have allowed for the discovery of an effect of hearing status on balance performance.

To address postural dual-task costs, Hypothesis 3 predicted that under dual-task conditions, participants would demonstrate significant dual-task costs in their balance performance. Additionally, we expected that these costs would be greater among those with ARHL. Within the normal vision condition, we observed postural dual-task costs in both groups, however the magnitude of these costs did not differ between HOA and ARHL groups. In the low vision condition, we found that those with age-related hearing loss demonstrated numerically fewer postural dual-task costs than those without hearing loss, however this result did not reach significance. When considering the normal and low vision conditions among ARHL individuals, postural dual-task costs decreased with the addition of the visual challenge. This finding suggests that there was some-

thing about the addition of the vision impairment goggles among those with ARHL that caused these individuals to allocate more attention towards the preservation of their balance. Similarly, Bruce and colleagues found that older adults with ARHL demonstrated postural prioritization (i.e., preserved balance performance) when undergoing dual-task conditions [55]. In contrast, other researchers found that with the addition of a cognitive load, balance performance was impaired in comparison to the single-task balance condition [46]. Essentially, within the low vision condition, those with ARHL are comparable to individuals with dual-sensory loss. Dual-sensory impairment has been linked to poorer balance performance and an increased risk of falls [56].

Lastly, to address cognitive dual-task costs, Hypothesis 4 predicted that individuals with age-related hearing loss would demonstrate greater cognitive dual-task costs in comparison to HOA. Greater cognitive dual-task costs have been found in studies demonstrating preserved postural performance at the expense of the cognitive task [42]. Contrary to expectation, we found a facilitation effect among those with ARHL, meaning that the participants improved in performance on the serial 7s subtraction task from single- to dual-task conditions. Interestingly, a study conducted by Hazamy and colleagues found that with a concurrent motor task, participants prioritized their cognitive performance, thus demonstrating improved N-back performance in the dual-task condition in comparison to the single-task condition. N-back tests are used as a way to measure working memory capacity [57]. While not as strong as in the normal vision condition, this facilitation effect was also observed in the low vision condition. This may have been due to a potential cohort effect. The mean age of the ARHL group was 5.86 years older than in the HOA group. Therefore, we believe that individuals with ARHL may feel as if they had more to prove cognitively, due to their increasing age. In the future, it would be beneficial to add a self-report questionnaire to evaluate whether or not they fear conforming to age stereotypes and how they believe their cognitive performance varies among multiple tasks. This would help to address whether age-related stereotypes of cognitive decline influence participants degree of dedication toward the cognitive task. The cognitive facilitation effect however is an ineffective dual-task strategy because older adults were allocating more of their attentional resources toward cognition and as a result less toward their balance performance. Sometimes termed the posture-second strategy this pattern is considered maladaptive and may lead to an increased risk of falling [58].

The present study had several limitations, which should be considered when interpreting the findings and proposing future directions. First, the participants' years or level of education was not recorded. This information may have helped explain the cognitive facilitation effect among the participants with ARHL. Perhaps these individuals were performing well cognitively because of a greater level of education in comparison to those without hearing loss. There may also be greater expertise with mental arithmetic in older generations [59]. Thus, future studies may benefit from matching by chronological age across groups, to consider whether those in older generations are more likely to perform better on arithmetic tasks. Additionally, the balance task itself (i.e., standing on the Wii Balance Board) may not have been challenging enough to see the dual-task costs we had originally anticipated. More specifically, the balance task required participants to stand similarly to how they would on a bathroom scale. To address the difficulty of the balance task itself, in the future it would be interesting to see how the participants balance performance may differ with the addition of a more threatening balance task, and whether we would observe worsened dual-task costs among ARHL individuals. For example, many studies have used a moving platform to evaluate balance performance [60].

Furthermore, the sample size was relatively small with an uneven distribution of individuals

with either mild or moderate hearing impairment, and no individuals with severe hearing impairment. This may explain why we were unable to find an effect of hearing status on balance performance. Finally, we only considered individuals with hearing loss and added the sensory challenge (i.e., vision impairment goggles). We did not consider individuals with actual vision loss or with dual-sensory loss and how their impact on balance performance may differ. In the future, it would be beneficial to consider individuals with vision loss while adding a hearing impairment, as well as individuals with dual-sensory loss to see how the different impairments affect balance performance independently and in conjunction.

There were also several strengths to this research project. Having the study conducted in a mall setting allowed for an inclusive representation of a general population. Additionally, the environment allowed for a relatively stress-free experiment experience.

In conclusion, this study aimed to determine the impact that sensory losses and increased cognitive load had on older adults' single-and-dual task balance performance. We discovered that with increased balance task complexity, participants demonstrated poorer balance performance. In addition, under dual-task conditions, individuals demonstrated increased postural dual-task costs and cognitive facilitation.

As previously mentioned, approximately 78% of Canadians between the ages of 60 to 79 have some form of hearing loss [9]. Within the 60 to 69 age group, only 9% of those with hearing loss were hearing aid users. Among the 70 to 79 age group, only 24% of individuals with hearing loss were hearing aid users. Thus, among a population greatly affected by hearing loss, very few individuals choose to correct their hearing. Interestingly, within our sample only one participant wore a hearing aid. Despite unexpectedly finding decreased postural and cognitive dual-task costs among our ARHL group, our results highlight how untreated hearing impairment may impact both cognitive and motor abilities.

In the low vision condition, the normal hearing individuals demonstrated dual-task costs in both postural and cognitive domains. It is reported than among individuals 80 and over, approximately 8% have uncorrected vision [9]. This finding further highlights the importance of correcting vision impairment when it occurs, as it can affect both older adult's balance as well as cognitive performance.

Ultimately, it is important to consider how both uncorrected hearing, vision, or both may impact older adults balance, cognitive functioning, and dual-task ability. Furthering the understanding the influence of visual and hearing aids can be pivotal in decreasing life-threatening injuries and falls among older adults

References

- [1] World Health Organization. (2018). Ageing, https://www.who.int/health-topics/ageing#tab=tab_1
- [2] Brennan, M., Horowitz, A., & Su, Y. (2005). Dual sensory loss and its importance in everyday competence. *The Gerontologist*, 45(3), 337-346. <https://doi.org/10.1093/geront/45.3.337>

- [3] Lacerda, C.F., Silva, L. O., De Tavares Canto, R. S., & Cheik, N.C. (2012). Effects of hearing aids in the balance, quality of life and fear to fall in elderly people with sensorineural hearing loss. *International Archives of Otorhinolaryngology*, 16(2), 156-162. <http://www.arquivosdeorl.org.br/conteudo/pdfForl/16-02-02-eng.pdf>
- [4] Cabeza, R., Anderson, N.D., Locantore, J.K., & McIntosh, A. R. (2002). Aging gracefully: compensatory brain activity in high performing older adults. *NeuroImage*, 17, 1394-1402. <https://doi.org/10.1006/nimg.2002.1280>
- [5] Diamond, A. (2013). Executive functions. *Annual Review of Psychology*, 64, 135-168. <https://doi.org/10.1146/annurev-psych-113011-143750>
- [6] Rogers, W.A. (2012). Attention and aging. In D.C. Park & N. Schwarz (Eds), *Cognitive aging: A prime* (pp. 57-71) Psychology Press
- [7] Borah, D., Wadhwa, S., Singh, U., Yadav, S., Bhattacharjee, M., & Sindhu, V. (2007). Age related changes in postural stability. *Indian J Physiol Pharmacol*, 51(4), 395-404. https://www.researchgate.net/profile/Upinderpal_Singh/publication/5375990_Age_related_changes_in_postural_stability/links/0912f50c6aad0bce7a000000.pdf
- [8] Osoba, M.Y., Rao, A.K., Agrawal, S.K., & Lalwani, A.K. (2019). Balance and gait in the elderly: A contemporary review. *Investigative Otolaryngology*, 4(1), 143-153. <https://doi.org/10.1002/lio2.252>
- [9] Statistics Canada. (2016). *Hearing loss of Canadians*, 2012 to 2015, <https://www150.statcan.gc.ca/n1/pub/82-625-x/2016001/article/14658-eng.htm>
- [10] Gopinath, B., Scheider, J., McMahon, C.M., Burlutsky, G., Leeder, S.R., & Mitchell, P. (2013). Dual sensory impairment in older adults increases the risk of mortality: A population-based study. *PLOS ONE*. <https://doi.org/10.1371/journal.pone.0055054>
- [11] Saunders, G.H., & Echt, V. (2007). An overview of dual sensory impairment in older adults: perspectives for rehabilitation. *Trends in Hearing*, 11(4) 243-258. <https://doi.org/10.1177/1084713807308365>
- [12] Alfaro, A.U., Guthrie, D.M., McGraw, C., & Wittich, W.(2020). Older adults with dual sensory loss in rehabilitation show high functioning and may fare better than those with single sensory loss. *PLOS ONE*, 15(8). <https://doi.org/10.1371/journal.pone.0237152>
- [13] Guerreiro, M.J., & Van Gerven, P.W.M. (2017). Disregarding hearing loss leads to overestimation of age-related cognitive decline. *Neurobiology of Aging*, 56, 180-189. <https://doi.org/10.1016/j.neurobiolaging.2017.05.001>
- [14] Liu, C., & Lee, C.T. (2019). Association of hearing loss with dementia. *Neurology*, 2(7). <https://jamanetwork.com/journals/jamanetworkopen/article-abstract/2740068>
- [15] Lin, F. R., & Ferrucci, L. (2012). Hearing loss and falls among older adults in the United States. *JAMA Internal Medicine*, 172(4), 369-372. <https://jamanetwork.com/journals/jamainternalmedicine/article-abstract/1108740>
- [16] Lin, F.R., Yaffe, K., Xia, J., Xue, Q., Harris, T.B., Purchase-Helzner, E., Statterfield, S., Ay-

onayon, H.N., Ferrucci, L., & Simonsick, E.M. (2013). Hearing loss and cognitive decline in older adults. *JAMA Internal Medicine*, 173(4), 293-299. <https://jamanetwork.com/journals/jamainternalmedicine/article-abstract/1558452>

[17] Uchida, Y., Sugiura, S., Nishita, Y., Saji, N., Sone, M., & Ueda, H. (2019). Age-related hearing loss and cognitive decline-The potential mechanisms linking the two. *Auris Nasus Larynx*, 46(1), 1-9. <https://doi.org/10.1016/j.anl.2018.08.010>

[18] Yamada, Y., Denkinger, M.D., Onder, G., Henrard, J.C., Van der Roest, H.G., Finne-Soveri, H., Richter, T., Vlachova, M., Bernabei, R., & Topinkova, E. (2016). Dual sensory impairment and cognitive decline: The results from the shelter study. *Journals of Gerontology: Medical Sciences*, 76(1), 117-123. <https://doi.org/10.1093/gerona/glv036>

[19] Mitoku, K., Masaki, N., Ogata, Y., & Okamoto, K. (2016). Vision and hearing impairments, cognitive impairment and mortality among long-term care recipients: a population-based cohort study. *BMC Geriatrics*, 16(112). <https://doi.org/10.1186/s12877-016-0286-2>

[20] Davidson, J. G., & Guthrie, D. M. (2019). Older adults with a combination of vision and hearing impairment experience higher rates of cognitive impairment, functional dependence, and worse outcomes across a set of quality indicators. *Journal of Aging and Health*, 31(1), 85-108. <https://doi.org/10.1177/0898264317723407>

[21] Brenowitz, W., Kaup, A., Lin, F.R., & Yaffe, K. Multiple sensory impairment is associated with increased risk of dementia among black and white older adults. *The Journals of Gerontology*, 74(6), 890-896. <https://doi.org/10.1093/gerona/gly264>

[22] Du, Y., Buchsbaum, B. R., Grady, C. L., & Alain, C. (2015). Increased activity in frontal motor cortex compensates impaired speech perception in older adults. *Nature Communications*, 7, 12241. <https://doi.org/10.1038/ncomms12241>

[23] Heyl, V., & Wahl, H. W. (2012). Managing daily life with age-related sensory loss: Cognitive resources gain in importance. *Psychology and Aging*, 27(2), 510-521. <https://doi.org/10.1037/a0025471>

[24] Seidler, R.D., Bernard, J.A., Burutolu, T.B., Fling, B.W., Gordon, M.T., Gwin, J.T., Kwak, Y., & Lipps,D.B. (2020). Motor control and aging: links to age-related brain structural, functional, and biochemical effects. *Neuroscience and Biobehavioural reviews*, 34(5), 721-733. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2838968/>

[25] Agmon, M., Lavie, L., & Doumas, M. (2017). The association between hearing loss, postural control, and mobility in older adults: A systematic review. *Journal of American Academy of Audiology*, 28(6), 575-588. <https://insights.ovid.com/american-academy-audiology/jaaa/2017/06/000/association-hearing-loss-postural-control-mobility/9/00001818>

[26] Kowalewski, V., Patterson, R., Hartos, J., & Bugnariu, N. (2018). Hearing loss contributes to balance difficulties in both younger and older adults. *Journal of Preventative Medicine*, 3(2). <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6017998/>

[27] Ray, C.T., Horvat, M., Croce, R., Mason, R.C., & Wolf, S.L. (2008). The impact of vision loss on postural stability and balance strategies in individuals with profound vision loss. *Gait & Posture*,

28(1), 58-61. <https://doi.org/10.1016/j.gaitpost.2007.09.010>

[28] Hallot, S., Pietrangelo, S., Aubin, G., Sader, E., Murphy, C.E., Li, K., & Johnson, A.P. (2020). Establishing baseline centre of pressure measures in older adults with and without simulated vision impairment using the Nintendo Wii Balance Board. *Invest. Ophthalmol. Vis Sci*, 61(7). <https://iovs.arvojournals.org/article.aspx?articleid=2766790>

[29] Teasdale, N., Bard, C., Larue, J., & Fleury, M. (1993). On the cognitive penetrability. *Experimental Aging Research*, 19(1), 1-13. <https://doi.org/10.1080/03610739308253919>

[30] Ponds, R.W., Brouwer, W.H., & Wolffelaar, P.C. (1988). Age differences in divided attention in a simulated driving task. *Journal of Gerontology*, 43(6), 151-156. <https://doi.org/10.1093/geronj/43.6.P151>

[31] Reuter-Lorenz, P.A., & Cappell, K.A. (2008). Neurocognitive aging and the compensation hypothesis. *Sage Journals*, 17(3), 177-182. <https://doi.org/10.1111/j.1467-8721.2008.00570.x>

[32] Farias, S., Giovannetti, T., Payne, B.R., Marsiske, M., Rebok, G.W., Schaie, W., Thomas, K.R., Willis, S.L., Dzierzewski, J.M., Unverzagt, F., & Gross, A.L. (2018) Self-perceived difficulties in everyday function precede cognitive decline among older adults in the ACTIVE study. *Journal of the International Neuropsychological Society*, 24(1), 104-112. <https://www.cambridge.org/core/journals/journal-of-the-international-neuropsychological-society/article/abs/selfperceived-difficulties-in-everyday-function-precede-cognitive-decline-among-older-adults-in-the-active-study/4D45186B504590DEA145932A3635AD52>

[33] Chen, P., Cheng, S.J., Lin, H.C., Lee, C.Y., & Chou, C. (2018). Risk factors for the progression of mild cognitive impairment in different types of neurodegenerative disorders. *Behavioural Neurology*, 2018, 6929732. <https://doi.org/10.1155/2018/6929732>

[34] Gauthier, S., Reisberg, B., Zaudig, M., Peterson, R.C., Ritchie, K., Broich, K., Belleville, S., Brodaty, H., Bennett, D., Chertkow, H., Cummings, J.L., Leon, M., Feldman, H., Ganguli, M., Hampel, H., Scheltens, P., Tierney, M.C., Whitehouse, P., & Winbald, B. (2006). Mild cognitive impairment. *The Lancet*, 376(9518), 1260-1270. [https://doi.org/10.1016/S0140-6736\(06\)68542-5](https://doi.org/10.1016/S0140-6736(06)68542-5)

[35] Shaw, F.E., Bond, J., Richardson, D.A., Dawson, P., Steen, N., McKeith, I., & Kenny, R. (2003). Multifactorial intervention after a fall in older people with cognitive impairment and dementia presenting to the accident and emergency department: randomised controlled trial. *BMJ*, 326(7380). <https://doi.org/10.1136/bmj.326.7380.73>

[36] Tangen, G.G., Engedal, K., Bergland, A., Moger, T.A., & Mengshoel, A.M. (2014). Relationships between balance and cognition in patients with subjective cognitive impairment, mild cognitive impairment, and Alzheimer's disease. *Physical Therapy*, 94(8), 1123-1134. <https://doi.org/10.2522/ptj.20130298>

[37] Malcolm, B.R., Foxe, J.J., Butler, J.S., & De Sanctis, P. (2015). The aging brain shows less flexible reallocation of cognitive resources during dual-task walking: A mobile brain/ body imaging study. *NeuroImage*, 117, 230-242. <https://doi.org/10.1016/j.neuroimage.2015.05.028>

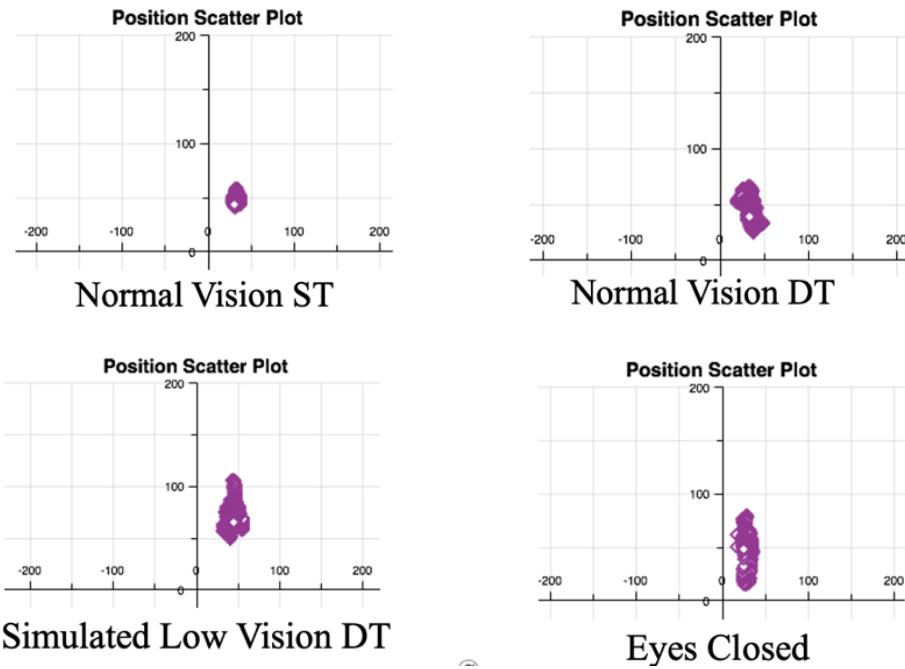
[38] Plummer, P., & Eskes, G. (2014). Measuring treatment effects on dual-task performance: a framework for research and clinical practice. *Frontiers in Human Neuroscience*. <https://doi.org/10.3389/fnhum.2014.00338>

- [39] Boisgontier, M., Beets, I.A., Duysens, J., Nieuwboer, A., Krampe, R.T., & Swinn, S.P. (2013). Age-related differences in attentional cost associated with postural dual tasks: Increased recruitment of generic cognitive resources in adults. *Neuroscience & Biobehavioural Reviews*, 37(8), 1824-1837. <https://doi.org/10.1016/j.neubiorev.2013.07.014>
- [40] Brustio, P. R., Magistro, D., Zecca, M., Rabaglietti, E., & Liubicich, M.E. (2017). Age related decrements in dual-task performance: Comparison of different mobility and cognitive tasks. A cross sectional study. *PLOS ONE*, 12(7). <https://doi.org/10.1371/journal.pone.0181698>
- [41] Huxhold, O., Li, S., Schmiedek, F., Lindenberger, U. (2006). Dual-tasking postural control: Aging and the effects of cognitive demand in conjunction with focus of attention. *Brain Research Bulletin*, 69(3), 294-305. <https://doi.org/10.1016/j.brainresbull.2006.01.002>
- [42] Doumas, M., Smolders, C., & Krampe, R.T. (2008). Task prioritization in aging: effects of sensory information on concurrent posture and memory performance. *Experimental Brain Research*, 187, 275-281. <https://doi.org/10.1007/s00221-008-1302-3>
- [43] Brown, L.A., Sleik, R.J., Polych, M.A., & Gage, W.H. (2002). Is the prioritization of postural control altered in conditions of postural threat in younger and older adults? *Journal of Gerontology*, 57(12) 785-792. <https://doi.org/10.1093/gerona/57.12.M785>
- [44] Berger, L., & Bernard-Demanze, L. (2010). Age-related effects of a memorizing spatial task in the adults and elderly postural control. *Gait & Posture*, 33(2), 300-302. <https://doi.org/10.1016/j.gaitpost.2010.10.082>
- [45] Shin, B.M., Han, S.J., Jung, J.H., Kim, J.E., & Fregni, F. (2011). Effect of mild cognitive impairment on balance. *Journal of Neurological Sciences*, 2305(1), 121-125. <https://doi.org/10.1016/j.jns.2011.02.031>
- [46] De Mello Alves Rodrigues, A.C., Reis Tinini, R.C., Gatica-Rojas, V., Deslandes, A.C., Pereira, E.L., De Rezende, L.F., Maillot, P., Cassilhas, R.C., & Monteiro-Junior, R.S. (2019). Motor-cognitive dual-task performance of older women evaluated using Wii Balance Board. *Aging Clinical and Experimental Research*, 32, 907-912. <https://doi.org/10.1007/s40520-019-01270-y>
- [47] Erdfelder, E., Faul, F., Buchner, A. (1996). Gpower: A general power analysis program. *Behavioural Research Methods Instruments & Computers*, 28(1), 1-11. https://www.researchgate.net/profile/Franz_Faul/publication/226660676_GPOWER_a_general_power_analysis_program/links/577d102508aef26c3b809c68/GPOWER-a-general-power-analysis-program.pdf
- [48] Chou, R., Dana, T., Bougatsos, C., & Beil, T. (2011). Screening for hearing loss in adults ages 50 years and older. *A Review of the Evidence for the U.S Preventive Services Task Force*, 83. Retrieved from <https://www.ncbi.nlm.nih.gov/books/NBK53864/toc/?report=reader>
- [49] Nasreddine, Z. S., Phillips, N. A., Bédirian, V., Charbonneau, S., Whitehead, V., Collin, I., Cummings, J. L., & Chertkow, H. (2005). The Montreal Cognitive Assessment, MoCA: A brief screening tool for mild cognitive impairment. *Journal of the American Geriatrics Society*, 53(4), 695-699. <https://doi.org/10.1111/j.1532-5415.2005.53221.x>

- [50] Lopez, M. N., Charter, R.A., Mostafavi, B. Nitbut, L., & Smith, W. E. (2005). Psychometric Properties of the Folstein Mini-Mental State Examination. *Assessment*, 12(2),137-144. <https://doi.org/10.1177/1073191105275412>
- [51] Bastianelli, M., Mark, A. E., McAfee, A., Schramm, D., Lefrançois, R., & Bromwich, M. (2019). Adult validation of a self-administered tablet audiometer. *Journal of Otolaryngology – Head & Neck*, 48, 59. <https://doi.org/10.1186/s40463-019-0385-0>
- [52] Yeung, J. C., Heley, S., Beauregard, Y., Champagne, S., & Bromwich, M. A. (2015). Self-administered hearing loss screening using an interactive, tablet play audiometer with ear bud headphones. *International Journal of Pediatric Otorhinolaryngology*, 79(8), 1248-1252. <https://doi.org/10.1016/j.ijporl.2015.05.021>
- [53] Martinez, J. R., & Fernandez, N. (2016). Open source posturography. *Acta Oto-Laryngologica*, 136(12), 1225-1229. <https://doi.org/10.1080/00016489.2016.1204665>
- [54] Deviterne, D., Gauchard, G. C., Jamet, M., Vançon, G., & Perrin, P. P. (2005). Added cognitive load through rotary auditory stimulation can improve the quality of postural control in the elderly. *Brain Research Bulletin*, 64(6), 487-492. <https://doi.org/10.1016/j.brainresbull.2004.10.007>
- [55] Bruce, H., Aponte, D., St-Ongle, N., Phillips, N., Gagné, J., Li, K. Z. H. (2019). The effects of age and hearing loss on dual-task balance and listening. *The Journals of Gerontology: Series B*, 74(2), 275-283. <https://doi.org/10.1093/geronb/gbx047>
- [56] Wilson, S. J., Garner, J. C., & Loprinzi, P. D. (2016). The influence of multiple sensory impairments on functional balance and difficulty with falls among U.S adults. *Preventive Medicine*, 87, 41-46. <https://doi.org/10.1016/j.ypmed.2016.02.023>
- [57] Hazamy, A. A., Altmann, L. J. P., Stegemoller, E., Bowers, D., Lee, H. K., Wilson, J., Okun, M. S., Haas, C. J. (2017). Improved cognition while cycling in Parkinson's disease patients and healthy adults. *Brain and Cognition*, 113, 23-31. <https://doi.org/10.1016/j.bandc.2017.01.002>
- [58] Bloem, B. R., Grimbergen, Y. A., Gert van Dijk, J., & Munneke, M. (2006). The "posture second" strategy: a review of wrong priorities in Parkinson's disease. *Journal of Neurological Science*, 24(1-2), 196-204. <https://doi.org/10.1016/j.jns.2006.05.010>
- [59] Oberauer, K., Demmrich, A., Mayr, U., & Kliegl, R. (2001). Dissociating retention and access in working memory: An ag-comparative study of mental arithmetic. *Memory and Cognition*, 29, 18-33. <https://doi.org/10.3758/BF03195737>
- [60] Dunsky, A., Zeev, A., & Netz, Y. (2017). Balance performance is task specific in older adults. *Biomed Research International*. <https://doi.org/10.1155/2017/6987017>

Appendix A

Centre of Pressure Displacements Scatter Plots



Note. Normal Vision ST= normal vision single task. Normal Vision DT= normal vision dual task. Simulated Low Vision DT= simulated low vision dual task.

Appendix B

Source Tables for Repeated Measures Analysis of Variance

Table B1

Analysis of Variance for Total Path Length Across Task Complexity

Source	SS	df	MS	F	p	η^2
Task Complexity	8313.43.65	1.80	462967.795	1.96	.157	.073
Task Complexity *	134960.53	1.80	75157.303	.32	.706	.013
Hearing Status						
Error	10607402.80	44.89	236286.688	Error		

Table B2

Analysis of Variance for Medial-lateral Sway Amplitude Across Task Complexity

Source	SS	df	MS	F	p	η^2
Task Complexity	3826.82	2.07	1845.462	2.63	.08	.095
Task Complexity *	1020.70	2.07	492.23	.70	.506	.027
Hearing Status						
Error	36435.40	51.84	702.83			

Table B3

Analysis of Variance for Anterior-posterior Sway Amplitude Across Task Complexity

Source	SS	df	MS	F	p	η^2
Task Complexity	7830.64	2.23	3507.23	6.40	.002	.204
Task Complexity *	1521.65	2.23	681.53	1.24	.299	.047
Hearing Status						
Error	30605.94	55.82	548.32			

Appendix C

Correlations between Hearing Acuity and Medial-Lateral Amplitude Across Task Complexity

Variables	1	2	3	4	5	6
1. Hearing Acuity (PTA)	-					
2. Normal Vision		.517**	-			
3. Normal Vision & serial 7s		.065	-.024	-		
4. Vision Impairment		.498**	.764**	-.137	-	
5. Vision Impairment & Serial 7s		.093	.242	.462*	-.007	-
6. Eyes Closed		-.014	.489**	-.144	.242	.146

Note. Hearing Acuity was determined using pure tone averages (PTA). A higher value of pure tone average indicates poorer hearing acuity.

* $p < .05$, ** $p < .01$

Evaluating the Relationship between Interpersonal Mindfulness and Social Connectedness with Emotion Regulation as a Mediator *

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Interpersonal mindfulness is a newly conceptualized construct that aims to measure mindful awareness during interpersonal interactions. Considering not much research has yet been conducted on how standard mindfulness and interpersonal mindfulness relate differently towards social outcomes, this study aimed to further explore how interpersonal mindfulness relates to social connectedness (SC), and if emotion regulation (ER) mediated the relationship between the two variables. A dataset of 438 participants (females = 89.9%; M age = 35.31) was procured from a previous scale validation study during which participants completed measures of mindfulness, interpersonal mindfulness, social connectedness and emotion regulation. Hierarchical multiple regression analyses revealed that interpersonal mindfulness was significantly positively associated with SC after controlling for standard mindfulness. Mediation analyses revealed that ER partially positively mediated the relationship between interpersonal mindfulness and SC. Considering these preliminary findings, future studies should conduct longitudinal research comparing the associations of standard mindfulness and interpersonal mindfulness to SC and other social outcomes.

Interpersonal Mindfulness and Social Connectedness

Research on mindfulness has flourished in the last decade [1], and multiple meta-analyses indicate mental health benefits that extend to clinical and non-clinical populations [2-4]. Often defined as "paying attention in a particular way; on purpose, in the present moment, and nonjudgmentally" [5], mindfulness is typically cultivated by a variety of meditation practices to bring greater clarity towards our patterns of thinking, feeling and behaviour. These practices result in greater mental flexibility, which in turn tends to promote a further sense of care for oneself and others [6]. Part of its popularity may be attributed to the fact that it has gained considerable support of evidence in a wide range of academic fields, most considerably in medicine and psychology [7, 8]. In therapy, this standing remains robust even when comparing mindfulness-based interventions to other current evidence-based treatments, such as cognitive-behavioural therapy. For example, a meta-analysis by Goldberg and colleagues investigated comparative effects of mindfulness-based interventions to evidence-based treatments on a variety of psychiatric conditions (e.g., Schizophrenia, Major Depressive Disorder, etc.) and showed both interventions to be of similar effectiveness, regardless of disorders [9]. Similar results were found in non-clinical populations as well [10].

Given the amount of evidence supporting the effectiveness of mindfulness in the last decades [3, 11], certain researchers have directed their attention to other subfields, such as bringing mindful awareness during social exchanges [12]. Indeed, the development of a mindful practice en-

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hances the capacity to be receptively aware of one's own present moment experiences, such as thoughts, emotions, bodily sensations, sounds, smells, etc., but research suggests this awareness may also be extended to interpersonal interactions [13]. In romantic couples, a meta-analysis by McGill et al. of 10 studies suggested that the positive association between mindfulness and relationship satisfaction was statistically significant with a small-medium effect size of $d = .27$ [14]. Another area of interest has been regarding parenting and child behaviour. According to a study by Fuller & Fitter, "Mindful parenting has aided in the decrease of maladaptive behaviours by increasing parent attention to the present moment, increasing engagement with the child, and decreasing judgment of the situation" [15].

Past groundwork has mostly focused on developing more generalized mindfulness measures, which we will address as standard mindfulness. The arguably most common standard mindfulness scales seemingly measure mindful awareness towards intrapersonal events, such as thoughts, or sensory experiences, such as sounds, but few of their items involve applying mindfulness specifically during social interactions. For example, the Mindful Attention Awareness Scale [16] offers one item out of fifteen ("I find myself listening to someone with one ear, doing something else at the same time.")

The Freiburg Mindfulness Inventory [17] offers one item out of thirty ("I am impatient with myself and others") and the Five Facet Mindfulness Questionnaire [18] and the Kentucky Inventory of Mindfulness Skills [19] offer none. In consequence, such scales may not be best suited to measure mindful awareness during interpersonal interactions. Indeed, different complexities come into play during social exchanges. Social interactions shape our mind through the practice of verbal communication, nonverbal cues such as gestures, facial expressions, and postures [20]. Comprehension of verbal messages is associated with enhanced synchrony between the brains of the speakers and listeners [21] and even our behaviour automatically adapts to match that of others in one's current social environment [22].

Some researchers have even suggested that interpersonal mindfulness may be a distinct construct from standard mindfulness [12, 23, 13]. Interpersonal mindfulness is defined as: "paying attention in the present moment while with another person/s, including being aware of internal experiences (bodily sensations, thoughts, reactions, mood, etc.) and external experiences (verbal and nonverbal communication, apparent mood, etc.)" [13]. Although standard and interpersonal mindfulness have similarities, being interpersonally mindful consists of being simultaneously aware of one's own experience while also attending to the experience of another person. According to the authors, this may allow a loosening of limiting cognitive-affective patterns such as: (1) ingrained emotional responses based on past interpersonal experiences, (2) not listening to others deeply because we focus on what we want to say next, or (3) misinterpretation of emotional signals from others because of a distortive internal narrative [13]. Given not only the added challenge of remaining attentive to oneself and to another person simultaneously, but of the possible loosening of the consequences mentioned, interpersonal mindfulness may draw upon different skills than those assessed in standard mindfulness scales.

To measure the social aspects of mindfulness, Pratscher et al. developed the Interpersonal Mindfulness Scale (IMS) and found that interpersonal mindfulness was associated with romantic relationship satisfaction above and beyond correlations with standard mindfulness [13]. Additionally, although standard and interpersonal mindfulness constructs did unsurprisingly correlate with the IMS ($r = .71$), the association was not as strong as the one found between various

standard mindfulness questionnaires ($r = .88$). Considering the recent development of the scale, little is still known about ways interpersonal mindfulness may uniquely relate to countless mental health constructs, particularly those pertaining to our social needs.

Social Connectedness

In a parallel fashion, destabilization following the start of the COVID-19 global crisis is also bringing a shift of focus in multiple academic fields, including psychology. Many countries have enforced numerous restrictions on daily living including social distancing, isolation, and home confinement. With the addition of in person interactions being replaced by virtual ones, the way we conceive human nature's need for social connection is being conscientiously reassessed. Social connectedness (SC), as originally drawn from Kohut's Self Psychology [24], is defined by Lee and Robbins as "an attribute of the self that reflects cognitions of enduring interpersonal closeness with the social world in toto" [25]. Closeness to others is experienced at a macro level, encompassing closer relationships with family and friends to further ones with strangers, community, or society. This denotes an internal subjective sense of belongingness with others at large, rather than to a certain group or to specific peer affiliations [26].

According to Lee & Robbins, SC emerges in early childhood years through parent-child attachment styles and continues to develop in adolescence as we start building rapport and securing bonds with people who share similar values, interest, or appearances [25]. In adulthood, these experiences slowly integrate into a relatively stable sense of self that persists amidst common social vacillations. A lack of SC indicates a persistent struggle towards feeling connected to others, which often results in a relative sense of loneliness [25]. Although related, it differs from loneliness, isolation, as well as loss of social support, the latter which tends to be impacted by specific events (e.g., the loss of a friendship, moving to a different city, romantic breakups; [27, 28]. People with higher SC tend to have higher self-esteem, social competence, and hope [28]. They also tend to have fewer interpersonal problems, fewer depression and anxiety symptoms, less rejection sensitivity, loneliness, and social avoidance [29]. Standard mindfulness, on the other hand, has shown to ease levels of depression, anxiety, and loneliness, as well as augment relationship satisfaction and secure attachment [30, 31], which suggests it could also play a role in regard to SC.

Emotion regulation

To shed further clarity on the possible mechanisms explaining the relationship between interpersonal mindfulness and SC, past evidence points to the potential role of emotion regulation (ER) as a mediator. ER is defined as follows: A process involving the (a) awareness and understanding of emotions, (b) acceptance of emotions, (c) ability to control impulsive behaviours and behave in accordance with desired goals when experiencing negative emotions, and (d) ability to use situationally-appropriate emotion regulation strategies flexibly to modulate emotional responses as desired in order to meet individual goals and situational demands [32].

There is a clear correlation between standard mindfulness and ER as currently defined, and the two have even been found to share neurobiological correlates [33, 34], although they remain distinct constructs that only share moderate amounts of variance [35]. Higher levels of self-reported standard mindfulness related to lower scores on the Difficulties in Emotion Regulation Scale (DERS) [24], even after controlling for symptoms of stress, anxiety, and depression [36].

On the other hand, greater ER enhances the development of social skills [37] and relationship

satisfaction [38]. This is in line with previous studies, considering understanding and differentiating one's emotions permits to understand others [39]. Finally, a correlational study by Winter, Moriarty, and Short, found that impairments in emotion regulation experienced by patients with traumatic brain injury negatively associated with various sub-aspects of social connectedness, such as interpersonal functioning [40]. Considering these findings altogether, interpersonal mindfulness may also facilitate ER in social contexts, perhaps even beyond standard mindfulness.

In light of the above, more research is needed to explore the potential of interpersonal mindfulness, especially in relationship with SC and ER. Findings may enlighten new interventions more effective at targeting interpersonal outcomes. The objective of this study would be to explore if interpersonal mindfulness positively relates to SC, and whether this association is mediated by ER. We hypothesize that (1) even when controlling for standard mindfulness, higher levels of interpersonal mindfulness will be associated with higher levels of SC and that (2) higher ER will mediate the relationship between interpersonal mindfulness and SC.

Method

Participants

A dataset was obtained from a sample of 1191 individuals who completed 27 scales in the context of a scale development and validation study conducted between 2018 and 2019. Half of the sample recruited were undergraduates from McGill University, whereas the other half were recruited from the general Montreal community. No exclusion criteria regarding participation were applied. After excluding individuals who did not complete the four scales of interest for this study, our final sample consisted of 438 participants (females = 89.9%; M age = 35.31; 74% White, 21.9% Asian, 2.1% Black, 2.5% as Latin American, 1.8% as Arab, and 4.8% from Indigenous descent). Since 67.1% of participants had at least a college degree, and 83% indicated that they always or mostly had enough money, the sample may be qualified as WEIRD. This acronym was term coined by Henrich, Heine and Norenzayan to define samples whose participants were mainly white, educated, industrialized, rich and from democratic countries [41].

Procedure

Compensation was of either 2 participant pool credits for the undergraduates, or 25\$ for individuals from the Montreal community. Participants who signed up completed the questionnaires online via LimeSurvey. The total duration of the study was around 2 hours.

Measures

Social Connectedness Scale- Revised (SCoS-R). The SCoS-R consists of 20 items and measures interpersonal closeness that individuals feel between themselves and others at a macro level [25]. For example, "I don't feel related to anyone." It has shown to have good test-retest ($\alpha = .96$) and internal reliability ($\alpha = .91$; [25, 29]. Items are placed on a 6-point Likert Scale (1 = strongly disagree to 6 = strongly agree), and higher scores represent a stronger sense of belonging.

Difficulties in Emotion Regulation Scale (DERS). The DERS [24] is a 36-item questionnaire that measures several elements of emotion regulation, including awareness, understanding, acceptance of emotions, as well as ability to regulate them adaptively to situations. However, it is conceptualized as a reverse scale. An example item is "I experience my emotions as overwhelming and out of control". Authors reported internal consistency of $\alpha = .93$, test-retest reliability of α

$\alpha = .88$ during a 4- to 8-week interval. Items are placed on a 5-point Likert Scale (1 = almost never to 5 = almost always). Higher scores indicate greater difficulties in emotion regulation [24].

Five Facet Mindfulness Questionnaire (FFMQ). The FFMQ [18] is a 39-item measure comprising five subscales of trait standard mindfulness: describing, observing, acting with awareness, non-reactivity, and non-judgmental acceptance. Items are placed on a 6-point Likert Scale (1 = strongly disagree to 6 = strongly agree) and include "I watch my feelings without getting carried away by them" Higher scores on subscales indicate higher trait standard mindfulness. Repeated administration pointed at high test-retest reliability and internal consistency of the assessment [42].

Interpersonal Mindfulness Scale (IMS). The IMS [13] is a 27-item questionnaire that consists of four subscales: presence, awareness of self and others, nonjudgmental acceptance, and non-reactivity. Items are rated on a 5-point Likert Scale (1 = Almost never, 5 = Almost always) and include items such as "When I am interacting with another person, I get a sense of how they are feeling". Higher scores indicate higher interpersonal mindfulness. Good test-retest reliability ($\alpha = .86$) and internal consistency was reported ($\alpha = .89$).

Results

Statistical analyses were conducted using R Studio, version 1.3.1073 [43]. Hierarchical multiple regression analysis was used to examine if interpersonal mindfulness contributes to SC total scores above and beyond the effect of standard mindfulness. In step 1, any confounding variables were entered in order to serve as control. In step 2, FFMQ total scores were entered into the regression equation. In step 3, IMS total scores were entered into the regression equation. Effect sizes in the regression were measured with Cohen's f^2 , as it is one of the most informative standardized measures of effect size for hierarchical linear modeling [44]. A single mediator analysis was conducted to evaluate if emotion regulation mediates the relationship between interpersonal mindfulness and social connectedness using the bootstrapping procedure (1000 resamples).

Prior to inferential analyses, correlation analyses were conducted. The first confirmed that all four variables of interest (FFMQ, IMS, DERS, SCoS-R) were significantly positively associated with each other (see Table 1). The second was conducted to determine if any demographic variables were significantly associated with SC (see Table 2). One variable, the perception that participants had of having enough money (MONEY), was significantly associated with SC and was entered in Step 1 as a control variable. Assumptions were met for normality, homogeneity, additivity and linearity. The residuals were normally distributed along an oval shape. Independent variables were assessed for collinearity as well. Results on the variance inflation factor (all <5.0) and collinearity tolerance (all greater than 0.2) indicated that the estimated β s were well established in the regression model.

Table 1*(Zero Order) Correlation Matrix of Variables of Interest*

Variables	1	2	3	4
FFMQ	—			
IMS	.68**	—		
DERS	-.82**	-.59**	—	
ScoS-R	.56**	.19**	-.62**	—
<i>M</i>	122.9	98.56	88.71	76.72
<i>SD</i>	23.69	14.81	27.56	16.79

Note. ** $p < 0.01$.**Table 2***Correlation Matrix of Demographic Variables and Social Connectedness*

Variable	1	2	3	4	5	6	7	8	9
Age	—								
Sex Orientation	-.20	—							
Relationship Status	.10	.12	—						
Living Situation	-.02	.05	-.16	—					
Employed	-.12	.06	.11	-.01	—				
Enough Money	-.07	.10	.08	-.09	.25	—			
Education	-.12	.05	.04	-.14	.09	.02	—		
Meditation Experience	-.18	-.01	-.03	.07	.10	.05	.02	—	
SC Total	.03	-.1	-.10	-.03	-.03	-.29*	-.08	-.10	—

Note. * $p < 0.05$.

The results from step 1 of the regression analysis suggested that the variable MONEY had a negative statistically significant association with SC (adjusted $R^2 = 0.08223$, $F(1, 436) = 40.15$, $p < 0.001$, $f^2 = .09$) with a medium effect size. The results from step 2 suggested that the added independent variable (FFMQ) had a statistically significant association with SC (adjusted $R^2 = 0.3592$, $F(2, 435) = 123.5$, $p < 0.001$, $f^2 = .44$) with a large effect size. The results from step 3 suggested that added independent variable (IMS) had a statistically significant effect on SC (adjusted $R^2 = 0.3652$,

$F(3, 434) = 84.8, p < 0.001, f^2 = .01$ with a small effect size.

Eight percent of variance in SC was explained in Model 1, $F(1, 436) = 40.15, p < 0.001$. There was a significant negative association between MONEY and SC ($B = -5.2574, SE = 0.8297, t(436) = -6.337, p < .001$). Thirty-six percent of variance in SC was explained in Model 2, $F(2, 435) = 123.5, p < 0.001$, which indicates that the FFMQ accounted for 28% additional variance. There was a statistically significant association between standard mindfulness and SC ($B = 0.37, SE = 0.0274, t(435) = 13.76, p < 0.001$). Thirty-seven percent of variance in SC was explained by the variables in Model 3, $F(2, 434) = 123.5, p < 0.001$, which indicates that the IMS accounted for less than 1% additional variance. There was a significant association between interpersonal mindfulness and SC ($B = 0.13, SE = 0.05903, t(434) = 2.266, p < 0.001$). Results from the hierarchical regression analysis are presented in Table 3.

Table 3

Hierarchical Multiple Regression Analysis on the Effect of Standard and Interpersonal Mindfulness on Social Connectedness

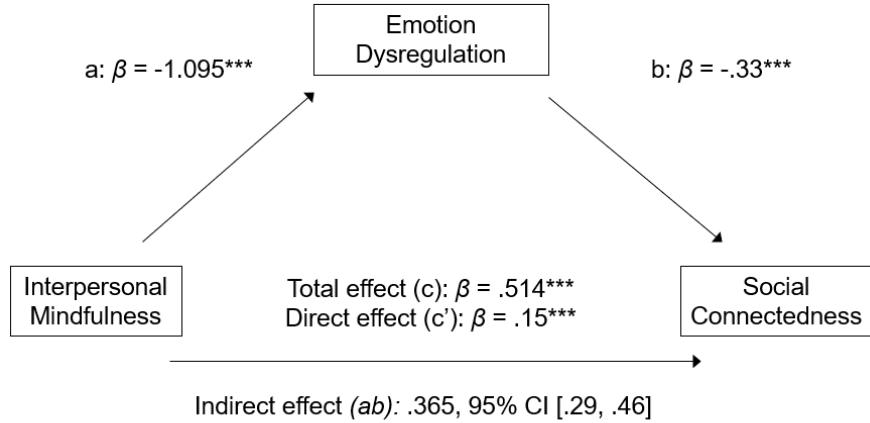
Variable	Step 1				Step 2				Step 3			
	<i>B</i>	<i>SE</i>	<i>b</i>	<i>t</i>	<i>B</i>	<i>SE</i>	<i>b</i>	<i>t</i>	<i>B</i>	<i>SE</i>	<i>b</i>	<i>t</i>
Money	-5.257	.830	-.290	-6.34	-3.93	3.79	-.217	9.82	-3.85	4.71	-.213	6.56
FFMQ					.37	.027	.532	13.76	.321	.037	.452	8.68
IMS									.13	.059	.118	2.66
R^2_{adj}			.082				.359				.365	

Note. All results = $p < 0.001$

A single mediation analysis was conducted to test the mediational role of emotion regulation in the relationship between interpersonal mindfulness and SC (see Figure 1 for model illustration). Results revealed that emotion regulation partially positively mediated the relationship between interpersonal mindfulness and SC. The estimated fully standardised indirect effect was .365 ($SE = 0.30; p < .001; 95\% CI = [.29, .46]$). This indicates that an increase of one standard deviation in IMS yields an increase of 0.30 standard deviation on DV SC through changes in the mediator (emotion regulation), which can be interpreted as a small-to-medium indirect effect.

Figure 1

Model illustration of the relationship between interpersonal mindfulness and social connectedness, mediated by emotion dysregulation.



Discussion

The present study aimed to explore the relationship of interpersonal mindfulness with social connectedness and emotion regulation. The objective was to determine if interpersonal mindfulness positively related to SC above and beyond standard mindfulness, and if this association was mediated by ER. Results supported our first hypothesis. Although the relationship was small, when controlling for standard mindfulness, interpersonal mindfulness was significantly positively associated with SC beyond standard mindfulness. Results also supported our second hypothesis as ER partially positively mediated the relationship between interpersonal mindfulness and SC.

Hypothesis 1

In regard to the first hypothesis, the present findings lend support to previous studies highlighting the relationship between standard mindfulness and SC [45, 46]. A study by Trautwein, Naranjo, and Schmidt found a causal relationship between Loving-Kindness Meditation, a form of mindfulness meditation, and “self-other connectedness”, a sense which is fundamental to empathy, social bonding and compassion [47]. Additionally, results from the study also showed a simultaneous decrease in self-centeredness during the increase in self-other connectedness. This suggests that perhaps SC works like a balance: the more socially connected we feel, the less self-centered we become. Previous research also supports that standard mindfulness relates to other social outcomes.

Interestingly, interpersonal mindfulness also related to SC beyond standard mindfulness. Interpersonal mindfulness especially brings one's attention to the social exchanges they're having, enhancing the presence they bring to their conversation partner [12]. Findings from research

suggest that social presence promotes the growth of social interaction quality amongst students, which in turn promoted the development of a sense of community [48]. Higher presence also augments interpersonal interaction quality [49]. This is in par with the current literature supporting that, while controlling for standard mindfulness, higher interpersonal mindfulness scores were related to higher friendship quality [50] and relationships quality [13], two variables that are also influenced by interpersonal interaction quality [51, 52, 53]. This indicates that future research may want to explore if interpersonal mindfulness would have a stronger association towards social variables that are more influenced by social interaction quality.

Results that the IMS only added a small contribution to SC when controlling for the FFMQ could be interpreted as unsurprising considering the similarities between standard mindfulness and interpersonal mindfulness. Indeed, standard mindfulness scales such as the FFMQ typically measure general mindfulness skills that may serve as stepping stones to other more specialized ways of using mindfulness. For example, certain items in the FFMQ include: "I think before reacting under stressful situations." or "I can easily talk about my thoughts and opinions". Such behaviours may not be satisfactory direct measurements of mindfulness during interpersonal exchanges, but they may serve as some form of prerequisites, as non-reactivity, self-awareness and honesty generally also benefit interpersonal outcomes such as relationship quality [54, 55].

Results may also support low practical implications of interpersonal mindfulness to increase social connectedness in comparison to standard mindfulness. This would mean that to increase social connectedness, training people through techniques that cultivate standard mindfulness (such as breathing meditations, body scans, etc.) [5] would have a similar effect than training them in interpersonal mindfulness techniques (such as mindful listening) [12]. However, experimental research supporting that increases interpersonal mindfulness and standard mindfulness similarly increase SC would be needed to support this interpretation.

Hypothesis 2

In regard to the second hypothesis, results are consistent with previous research indicating positive relationships between standard mindfulness and ER, as well as between ER and SC. Since no previous research has yet studied the association between interpersonal mindfulness and ER or SC, findings with standard mindfulness were used to elaborate our hypothesis instead. As previously mentioned, the literature strongly supports an association between standard mindfulness and ER. Findings of this study supported that ER and interpersonal mindfulness are also separate constructs considering (1) no multicollinearity was found between the IMS and the DERS (2) the indirect effect suggested a partial but not full mediation between interpersonal mindfulness and SC through ER.

Additionally, research supports that higher levels of standard mindfulness related to lower scores on the DERS, even after controlling for symptoms of stress, anxiety, and depression [36]. Considering interpersonal mindfulness may make use of abilities measured in standard mindfulness scales, the positive relationship it has with ER is consistent with the literature. Interpersonal mindfulness may relate to SC through ER through the similar scales dimensions it shares with ER. ER involves an awareness of and acceptance of emotional response, as well as an impulse control when experiencing negative emotions [24]. Similar abilities are measured by interpersonal mindfulness through its awareness of self and others, non-judgemental acceptance and nonreactivity facets.

Findings are also in par with a study showing a negative relationship between lack of ER and SC measures in patients with traumatic brain injury [40]. Research on ER has shown that it has a positive effect on other social outcomes, such as relationship quality [56] and relationship satisfaction [57]. Interpersonal mindfulness may possibly enhance ER, which may have an effect on SC. However, more research would be needed to support this hypothesis.

Limitations and Future Directions

As little research has yet been conducted on interpersonal mindfulness, this study is more exploratory in nature. In consideration, some limitations are bear mentioning. Foremost, due to the cross-sectional nature of the study, it is not possible to infer causation from these results. Longitudinal data would be necessary to explain the effect interpersonal mindfulness would have on SC. Moreover, the study sample was restricted to voluntary participants from the Montreal region and was predominantly female. It also holds WEIRD characteristics, which in the literature has been labelled as problematic as it limits generalizability to the general population [44]. In addition, no known exclusion criteria were applied considering data was originally collected for a scale validation study, where exclusion criteria are not mandatory. Future studies examining gender-balanced samples with appropriate exclusion criteria (such as exclusion of individuals with mental disorders to not confound clinical and non-clinical populations) are warranted.

References

- [1] American Mindfulness Research Association (2019). *AMRA Resources and Services*. <https://goamra.org/resources/>
- [2] Carletto, S., Cavalera, C., Sadowski, I., Rovaris, M., Borghi, M., Khoury, B., Ostacoli, L., & Pagnini, F. (2020). Mindfulness-based interventions for the improvement of well-being in people with multiple sclerosis: A systematic review and meta-analysis. *Psychosomatic Medicine*, 82(6), 600–613. <https://doi.org/10.1097/PSY.0000000000000819>
- [3] Khoury, B., Lecomte, T., Fortin, G., Masse, M., Therien, P., Bouchard, V., Chapleau, M.-A., Paquin, K., & Hofmann, S. G. (2013). Mindfulness-based therapy: A comprehensive meta-analysis. *Clinical Psychology Review*, 33(6), 763–771. <https://doi.org/10.1016/j.cpr.2013.05.005>
- [4] Khoury, B., Sharma, M., Rush, S. E., & Fournier, C. (2015). Mindfulness-based stress reduction for healthy individuals: A meta-analysis. *Journal of Psychosomatic Research*, 78(6), 519–528. <https://doi.org/10.1016/j.jpsychores.2015.03.009>
- [5] Kabat-Zinn, J. (1994). *Wherever you go, there you are: Mindfulness meditation in everyday life*. Hyperion.
- [6] Mindful Nation UK. (2015) *Mindful Nation UK report*. The mindfulness initiative. <https://www.themindfulnessinitiative.org/mindful-nation-report>
- [7] Davidson, R. J., Kabat-Zinn, J., Schumacher, J., Rosenkranz, M., Muller, D., Santorelli, S. F., Urbanowski, F., Harrington, A., Bonus, K., & Sheridan, J. F. (2003). Alterations in brain and immune function produced by mindfulness meditation. *Psychosomatic Medicine*, 65(4), 564–570.

<https://doi.org/10.1097/01.PSY.0000077505.67574.E3>

- [8] Keng, S.-L., Smoski, M. J., & Robins, C. J. (2011). Effects of mindfulness on psychological health: A review of empirical studies. *Clinical Psychology Review*, 31(6), 1041–1056. <https://doi.org/10.1016/j.cpr.2011.04.006>
- [9] Goldberg, S. B., Tucker, R. P., Greene, P. A., Davidson, R. J., Wampold, B. E., Kearney, D.J., & Simpson, T. L. (2018). Mindfulness-based interventions for psychiatric disorders: a systematic review and meta-analysis. *Clinical Psychology Review*, 59, 52–60. <https://doi.org/10.1016/j.cpr.2017.10.011>
- [10] Dawson, A. F., Brown, W. W., Anderson, J., Datta, B., Donald, J. N., Hong, K., ... Galante, J. (2020). Mindfulness-based interventions for university students: a systematic review and meta-analysis of randomised controlled trials. *Applied Psychology: Health and Well-Being*, 12(2), 384–410. <https://doi.org/10.1111/aphw.12188>
- [11] Querstret, D., Morison, L., Dickinson, S., Cropley, M., & John, M. (2020). Mindfulness-based stress reduction and mindfulness-based cognitive therapy for psychological health and well-being in nonclinical samples: A systematic review and meta-analysis. *International Journal of Stress Management*, 27(4), 394-411. <http://doi.org/10.1037/str0000165>
- [12] Khoury, B., Grégoire, S., & Dionne, F. (2020). La dimension interpersonnelle de la pleine conscience. *Annales Médico-Psychologiques*, 178(3), 239–244. <https://doi.org/10.1016/j.amp.2018.10.018>
- [13] Pratscher, S. D., Wood, P. K., King, L. A., & Bettencourt, B. A. (2019). Interpersonal mindfulness: scale development and initial construct validation. *Mindfulness*, 10(6), 1044–1061. <https://doi.org/10.1007/s12671-018-1057-2>
- [14] McGill, J., Adler-Baeder, F., & Rodriguez, P. (2016). Mindfully in love: a meta-analysis of the association between mindfulness and relationship satisfaction. *Journal of Human Sciences and Extension*, 4(1). <https://www.jhseonline.com/article/view/623>
- [15] Fuller, J. L., & Fitter, E. A. (2020). Mindful parenting: A behavioral tool for parent well-being. *Behavior Analysis in Practice*, 13, 767–771 <https://doi.org/10.1007/s40617-020-00447-6>
- [16] Brown, K. W., & Ryan, R. M. (2003). The benefits of being present: Mindfulness and its role in psychological well-being. *Journal of Personality and Social Psychology*, 84(4), 822–848. <https://doi.org/10.1037/0022-3514.84.4.822>
- [17] Walach, H., Buchheld, N., Buttenmüller, V., Kleinknecht, N., & Schmidt, S. (2006). Measuring mindfulness—The Freiburg Mindfulness Inventory (FMI). *Personality and Individual Differences*, 40(8), 1543–1555. <https://doi.org/10.1016/j.paid.2005.11.025>
- [18] Baer, R. A., Smith, G. T., Hopkins, J., Krietemeyer, J., & Toney, L. (2006). *Using Self-Report Assessment Methods to Explore Facets of Mindfulness*. *Assessment*, 13(1), 27 –45. <https://doi.org/10.1177/1073191105283504>
- [19] Baer, R. A., Smith, G. T., & Allen, K. B. (2004). Assessment of Mindfulness by Self-Report: The Kentucky Inventory of Mindfulness Skills. *Assessment*, 11(3), 191–206. <https://doi.org/10.1177/1073191104267001>

- [20] Hari, R., & Kujala, M. V. (2009). Brain basis of human social interaction: from concepts to brain imaging. *Physiological reviews*, 89(2), 453–479. <https://doi.org/10.1152/physrev.00041.2007>
- [21] Stephens, G. J., Silbert, L. J., & Hasson, U. (2010). Speaker-listener neural coupling underlies successful communication. *PNAS Proceedings of the National Academy of Sciences of the United States of America*, 107(32), 14425–14430. <https://doi.org/10.1073/pnas.1008662107>
- [22] Chartrand, T. L., & Bargh, J. A. (1999). The chameleon effect: The perception–behavior link and social interaction. *Journal of Personality and Social Psychology*, 76(6), 893–910. <https://doi.org/10.1037/0022-3514.76.6.893>
- [23] Medvedev, O. N., Pratscher, S. D., & Bettencourt, A. (2020). Psychometric evaluation of the interpersonal mindfulness scale using rasch analysis. *Mindfulness*, 11(8), 2007–2015. <https://doi.org/10.1007/s12671-020-01415-5>
- [24] Kohut, H., Goldberg, A., & Stepansky, P. E. (1984). *How does analysis cure?* University of Chicago Press.
- [25] Lee, R. M., & Robbins, S. B. (1995). Measuring belongingness: The Social Connectedness and the Social Assurance scales. *Journal of Counseling Psychology*, 42(2), 232–241. <https://doi.org/10.1037/0022-0167.42.2.232>
- [26] Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin*, 117(3), 497–529. <https://doi.org/10.1037/0033-2909.117.3.497>
- [27] Hawkley, L. C., Browne, M. W., & Cacioppo, J. T. (2005). How Can I Connect With Thee? Let Me Count the Ways. *Psychological Science*, 16(10), 798–803. <https://doi.org/10.1111/j.1467-9280.2005.01617.x>
- [28] Williams, K. L., & Galliher, R. V. (2006). Predicting depression and self-esteem from social connectedness, support, and competence. *Journal of Social and Clinical Psychology*, 25(8), 855–874. <https://doi.org/10.1521/jscp.2006.25.8.855>
- [29] Lee, R. M., & Robbins, S. B. (1998). The relationship between social connectedness and anxiety, self-esteem, and social identity. *Journal of Counseling Psychology*, 45(3), 338–345. <https://doi.org/10.1037/0022-0167.45.3.338>
- [30] Barnes, S., Brown, K. W., Krusemark, E., Campbell, W. K., & Rogge, R. D. (2007). The role of mindfulness in romantic relationship satisfaction and responses to relationship stress. *Journal of Marital and Family Therapy*, 33(4), 482–500. <https://doi.org/10.1111/j.1752-0606.2007.00033.x>
- [31] Laurent, H., Laurent, S., Hertz, R., Egan-Wright, D., & Granger, D. A. (2013). Sex-specific effects of mindfulness on romantic partners' cortisol responses to conflict and relations with psychological adjustment. *Psychoneuroendocrinology*, 38(12), 2905–2913. <https://doi.org/10.1016/j.psyneuen.2013.07.018>
- [32] Gratz, K.L., Roemer, L. (2004). Multidimensional assessment of emotion regulation and

dysregulation: development, factor structure, and initial validation of the difficulties in emotion regulation scale. *Journal of Psychopathology and Behavioral Assessment* 26(1), 41–54. <https://doi.org/10.1023/B:JOBA.0000007455.08539.94>

[33] Gillespie, S. M., & Beech, A. (2018). *Treating emotion dysregulation in antisocial behavior: A neuroscientific perspective*. In A. Beech, A. J. Carter, R. E. Mann, & P. Rotshtein (Eds.), *The Wiley Blackwell Handbook of Forensic Neuroscience*. (pp. 677-701). Wiley-Blackwell.

[34] Tang, Y. Y., Hölzel, B. K., & Posner, M. I. (2015). The neuroscience of mindfulness meditation. *Nature Reviews Neuroscience*, 16(4), 213–225. <https://doi.org/10.1038/nrn3916>

[35] Dixon, M. L., Thiruchselvam, R., Todd, R., & Christoff, K. (2017). Emotion and the prefrontal cortex: An integrative review. *Psychological Bulletin*, 143(10), 1033–1081. <https://doi.org/10.1037/bul0000096>

[36] Roemer, L., Lee, J. K., Salters-Pedneault, K., Erisman, S. M., Orsillo, S. M., & Mennin, D. S. (2009). Mindfulness and emotion regulation difficulties in generalized anxiety disorder: preliminary evidence for independent and overlapping contributions. *Behavior therapy*, 40(2), 142–154. <https://doi.org/10.1016/j.beth.2008.04.001>

[37] Bell, K. L., & Calkins, S. D. (2000). Relationships as inputs and outputs of emotion regulation. *Psychological Inquiry*, 11(3), 160–163. https://doi.org/10.1207/S15327965PLI1103_04

[38] Buduris, A. K. (2021). Attachment style and emotion dysregulation as serial mediators of betrayal trauma experiences and level of satisfaction in romantic relationships. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 82(3-B).

[39] Israelashvili, J., Oosterwijk, S., Sauter, D., & Fischer, A. (2019). Knowing me, knowing you: emotion differentiation in oneself is associated with recognition of others' emotions. *Cognition & Emotion*, 33(7), 1461–1471. <https://doi.org/10.1080/02699931.2019.1577221>

[40] Winter, L., Moriarty, H. J., & Short, T. H. (2018). Beyond anger: Emotion regulation and social connectedness in veterans with traumatic brain injury. *Brain Injury*, 32(5), 593–599. <https://doi.org/10.1080/02699052.2018.1432895>

[41] Henrich, J., Heine, S. J., & Norenzayan, A. (2010). Beyond weird: towards a broad-based behavioral science. *Behavioral and Brain Sciences*, 33(2-3), 111–135. <https://doi.org/10.1017/S0140525X10000725>

[42] Shallcross, A., Lu, N. Y., & Hays, R. D. (2020). Evaluation of the psychometric properties of the five facet of mindfulness questionnaire. *Journal of Psychopathology and Behavioral Assessment*, 42(2), 271–280. <https://doi.org/10.1007/s10862-019-09776-5>

[43] RStudio Team. (2015). RStudio: Integrated Development Environment for R. Boston, MA. Retrieved from <http://www.rstudio.com/>

[44] Selya, A. S., Rose, J. S., Dierker, L. C., Hedeker, D., & Mermelstein, R. J. (2012). A practical guide to calculating Cohen's f^2 , a measure of local effect size, from PROC MIXED. *Frontiers in Psychology*, 3(111). <https://doi.org/10.3389/fpsyg.2012.00111>

[45] Arnold, A. J., Winkielman, P., & Dobkins, K. (2019). Interoception and social connection.

Frontiers in Psychology, 10(2589). <https://doi.org/10.3389/fpsyg.2019.02589>

- [46] Aspy, D. J., & Proeve, M. (2017). Mindfulness and loving-kindness meditation. *Psychological Reports*, 120(1), 102–117. <https://doi.org/10.1177/0033294116685867>
- [47] Trautwein, F.-M., Naranjo, J. R., & Schmidt, S. (2014). *Meditation effects in the social domain: Self-other connectedness as a general mechanism?* In S. Schmidt & H. Walach (Eds.), *Studies in neuroscience, consciousness and spirituality: Vol. 2. Meditation — Neuroscientific approaches and philosophical implications* (p. 175– 198). Springer International Publishing. https://doi.org/10.1007/978-3-319-01634-4_10
- [48] Walker, B. K. (2008). Bridging the distance: How social interaction, presence, social presence, and sense of community influence student learning experiences in an online virtual environment. *Dissertation Abstracts International Section A: Humanities and Social Sciences*, 68(12-A), 4969.
- [49] Parker, S. C., Nelson, B. W., Epel, E. S., & Siegel, D. J. (2015). *The science of presence: A central mediator of the interpersonal benefits of mindfulness.* In K. W. Brown, J. D. Creswell, & R. M. Ryan (Eds.), *Handbook of mindfulness: Theory, research, and practice* (p. 225–244). The Guilford Press.
- [50] Pratscher, S. D., Rose, A. J., Markovitz, L., & Bettencourt, A. (2018). Interpersonal mindfulness: Investigating mindfulness in interpersonal interactions, co-rumination, and friendship quality. *Mindfulness*, 9(4), 1206–1215. <https://doi.org/10.1007/s12671-017-0859-y>
- [51] Karos, L. K., Howe, N., & Aquan-Assee, J. (2007). Reciprocal and complementary sibling interactions, relationship quality and socio-emotional problem solving. *Infant and Child Development*, 16(6), 577–596. <https://doi.org/10.1002/icd.492>
- [52] Kurdek, L. A. (1990). Spouse attributes and spousal interactions as dimensions of relationship quality in first-married and remarried newlywed men and women. *Journal of Family Issues*, 11(1), 91–100. <https://doi.org/10.1177/019251390011001006>
- [53] Sprecher, S., & Duck, S. (1994). Sweet talk: The importance of perceived communication for romantic and friendship attraction experienced during a get-acquainted date. *Personality and Social Psychology Bulletin*, 20(4), 391–400. <https://doi.org/10.1177/0146167294204006>
- [54] Cant, R. P., & Aroni, R. A. (2008). Exploring dietitians' verbal and nonverbal communication skills for effective dietitian-patient communication. *Journal of Human Nutrition and Dietetics*, 21(5), 502–511. <https://doi.org/10.1111/j.1365-277X.2008.00883.x>
- [55] Laplante, J. P. (2020). The couple that sits together stays together: The effects of meditation on romantic relationships. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 81(3-B).
- [56] Williams, W. C., Morelli, S. A., Ong, D. C., & Zaki, J. (2018). Interpersonal emotion regulation: Implications for affiliation, perceived support, relationships, and well-being. *Journal of Personality and Social Psychology*, 115(2), 224–254. <https://doi.org/10.1037/pspi0000132>
- [57] Wiggins, K. T. (2013). Mindfulness and emotion in relationships: Emotion regulation, empathy, and affect as mediators of the association between mindfulness and relationship satisfaction. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 74(2-B(E))

The Effect of Chronic Corticosterone Administration During Abstinence on Heroin Seeking in Male Rats *

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Drug addiction remains a prevalent issue. Of the cycle of drug addiction, the most problematic phase seems to be the high rates of relapse after abstinence or treatment. Stress is one trigger of relapse. Recent findings suggest chronic food restriction, a mild stressor known to increase corticosterone, induces an increase in heroin seeking. Chronic administration of corticosterone during the abstinence period was used to analyze heroin seeking behavior. Male rats were trained to self-administer heroin for 10 days then separated into three groups (water, vehicle, corticosterone) for the duration of the 15-day abstinence period. Following abstinence, a heroin-seeking test was conducted under extinction conditions. We found no evidence to support the hypothesis that corticosterone affects heroin seeking, suggesting that the augmentation in heroin seeking induced by chronic food restriction is not mediated by corticosterone.

Drug addiction has multiple harmful effects. Substance disorder not only deteriorates the users' physical and mental well-being, but also impacts their families, friends, and the community [1]. In addition, there are large-scale societal costs, such as over-packed health institutes and public hazards. Drug addiction is a chronic disorder that is often described as a vicious cycle characterized by compulsive drug seeking, interwoven with recurrent periods of drug abstinence, and relapse [2,3]. Substance addiction produces long-lasting neurophysiological changes that persist even when there is prolonged abstinence. For instance, disrupted corticotrophin-releasing factor (CRF) release in the extended amygdala, an area key in drug reinforcement [4]. Changes induced in key regions, accompanied by environmental and psychosocial factors, then contribute to relapse [2]. Heroin is a highly addictive drug. The death rate among heroin users is 50 to 100 times greater than the rate of the general population, according to Smyth [5]. This illicit substance carries with it a very high rate of relapse, reaching up to 91% [5]. Zickler and colleagues reported that one-quarter of the people recovering from addiction have relapsed after 15 years of abstinence [6]. To date, the neurobiological mechanisms that mediate relapse to addictive drugs remain elusive, and this research project aims to bridge this gap.

Three key factors trigger relapse to drug use. The first is exposure to the drug itself after a period of abstinence. For example, abstinent drug users' risk of relapse significantly increases if they come in contact with the illicit drug [7–9]. Secondly, cues previously associated with drug use are potent triggers for reuse [6]. Cues like the original drug-taking context, the individuals that surrounded the drug user during consumption, as well as the materials or tools used to once administer a drug, are strong associations that can lead to relapse. Lastly, stress is commonly associated with drug relapse. Recovering drug users are vulnerable to a plethora of acute or chronic stressors, such as high periods of stress at work or traumatic life events [7–10]. Studies

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used to understand the effect of stress on drug relapse commonly use food restriction as either an acute or chronic stressor. In humans, chronic food restriction is known to increase drug taking and craving [11] whereas acute food deprivation has not been shown to affect the number of cigarettes smoked [12]. Food restriction provides an interesting avenue for studying stress-induced relapse. A potential mechanism explaining the association between stress and relapse is the activation of the hypothalamic-pituitary-adrenal (HPA) axis [9,13]. The HPA axis begins with the stimulation of the hypothalamus which then results in a cascade of events leading to the release of the non-human primate glucocorticoid [22].

A potential trigger of the HPA axis is dietary manipulations. Interestingly, the effects of dietary manipulations on drug-taking and seeking in rats are similar to those observed in human addicts (i.e. readily self-administering a drug). Research suggests that an increase in drug-taking is related to a decrease in body weight, and a lower bodyweight led to more drug intake [19]. More specifically experiments conducted by D'Cunha and colleagues demonstrated that chronic food restriction increased heroin seeking by 250% compared to rats that were sated [10]. Taken together, these results support the idea that dietary manipulation increases drug-taking and drug seeking in rodents.

Previous research has established the relation between chronic food restriction and increased drug intake in both human and rodent studies. However, the underlying neural mechanisms involving food restriction-induced augmentation of heroin seeking behaviour remain elusive. As previously mentioned, research has indicated that chronic food restriction during abstinence increases subsequent heroin seeking [10]. Of particular interest, is research by Carr and colleagues that found that animals that were food-restricted for 14 days compared to a food-sated control had elevated corticosterone concentration levels [20]. This experiment aims to further explore the role of chronic corticosterone by mimicking the elevated corticosterone effect observed following chronic food restriction. This goal will be achieved by chronically administering exogenous corticosterone during abstinence following heroin self-administration training. We hypothesize that this chronic administration of corticosterone will increase heroin seeking following abstinence.

Method

Animals

Twenty-three male Long Evans rats served as subjects for this experiment (Charles River, Raleigh, New Jersey, U.S.A.). Upon arrival, all rats weighed between 250-275g. Rats were paired housed in plastic shoebox cages during the acclimatization period for a week in the Animal Care Facility (ACF) on a reverse light/dark cycle (9:30 am lights off – 9:30 pm lights on). For the entire duration of the experiment, all rats had *ad libitum* access to food (Agribran Purina Canada Inc., Woodstock, Ontario) and water. Following acclimatization, rats underwent surgery followed by a two-day recovery, at which time they were housed in cages separately. After two days of post-operative recovery, they were transferred to the operant self-administration chambers for training.

Intravenous catheterization

Rats were implanted with an intravenous (i.v.) Silastic catheter (Dow Corning, Midland, MI, USA) 3 cm into the right jugular vein, which was held in place with silk sutures, as previously described in [10].

Apparatus

Training involved 10 operant conditioning chambers (Med Associates Inc., St. Albans, Vermont, USA; 32.0 cm X 24.0 cm x 25.0 cm) enclosed in sound-attenuating boxes equipped with a fan. Each chamber contained a red house light , a food hopper, and a water bottle. The chamber included two levers. The active lever is located 5 cm above the floor and is installed on the wall opposite to the houselight. The inactive lever is located 5 cm above the floor on the same wall as the active lever. An infusion pump (Razel Scientific Instruments, Stamford, CT) was installed inside the sound-attenuating cabinet. A cue light and a tone generator (2.9 kHz; 10 dB) are located directly above the active lever. The active lever was paired with a drug infusion. Pressing on the inactive lever did not result in a drug infusion, although responses were still recorded. This record served as a control for baseline, non-reinforced operant responding. Tygon tubing (Saint-Gobain, Courbevoie, France) was attached to a swivel (Lomir Biomedical Inc., Notre-Dame-de-l'Île-Perrot, QC, Canada) that was connected to a 20.0 ml syringe mounted on the infusion pump, through which the drug was delivered.

Drugs

Heroin (diacetylmorphine HCl; provided by the National Institute for Drug Abuse, Research Triangle Park, NC, USA) was prepared by dissolving it in 0.9% sterile saline (5.0 mg/ml). Based on the body weight of each rat, this solution was further diluted with 0.9% saline in order to yield 0.1 mg/kg/infusion.

Corticosterone (Sigma, Oakville, ON., Ca) was dissolved in 99.99% ethanol with the use of a sonicator. This solution was further diluted with water, yielding a 2% concentration of Ethanol and a concentration of 300 µg/ml corticosterone.

Procedure

Self-administration training. First, rats were placed in the operant chamber for a 24 h habituation period. Following the 24 h habituation period, rats underwent 10 days of heroin self-administration training. Rats were exposed to three 3 hr training sessions per day. An interval period of 3 hr separated each training session. Each training session started with the illumination of the house light, followed by the extension of the active lever and the activation of the cue light and the cue tone. This sequence lasted 30 s, or until a response on the active lever was made. Training involved a fixed-interval 20 (FI - 20) schedule of reinforcement with a 20-s timeout period. That is, pressing on the active lever resulted in a 0.1 mg/kg infusion of heroin over 12 s, as well as the activation of the cue light and tone and turning off of the house light. Furthermore, pressing on the active lever resulted in a 20 s timeout period. This timeout ensured that the rat would not overdose. Any lever presses made during the 20 s interval following the initial response did not result in an additional infusion, but the responses were recorded. The inactive lever is adjacent to the active lever and there are no programmed consequences. All responses on the inactive lever were also recorded.

Drug Withdrawal. Upon completion of 10 days of self-administration training, rats were removed from the operant conditioning chamber and moved to the ACF. Rats were individually housed for 24 h and underwent a drug washout period. During this washout period, rats had *ad libitum* access to food and water. Next, rats were separated into three treatment groups [water, vehicle (2% ethanol in water), corticosterone] and went through forced abstinence over 15 days.

Heroin Seeking Test. On the test day (15th day of the forced abstinence period), rats were brought back to the operant chamber for a 3-hr heroin-seeking test. The heroin-seeking test took place under the same conditions as the self-administration training, except pressing on the active lever did not result in a heroin infusion, i.e., rats were tested under extinction conditions.

Locomotor activity test. Three days following the blood collection rats completed a locomotor activity test. Rats were placed into a locomotor activity chamber ($39.0 \times 42.0 \times 50.0$ cm) for a one-hour test session. The chamber is composed of a 16×16 infrared photocells matrix (Coulbourn Instruments). The chamber recorded the total distance traveled by rats. This test was to ensure that, if an effect of corticosterone is determined, the observed effect was not due to differences in general locomotor activity.

Tissue Collection. Immediately following the locomotor activity test, the animals were humanely euthanized. This was done with an overdose injection of Euthanyl (sodium pentobarbital). Rats were then intracardially perfused with phosphate-buffer-saline (1X PBS), paraformaldehyde (4%). Brains were extracted and postfixed for 24 hours, dehydrated with 30% sucrose at 4°C for 48 hours then stored in -80°C .

Statistical Analyses.

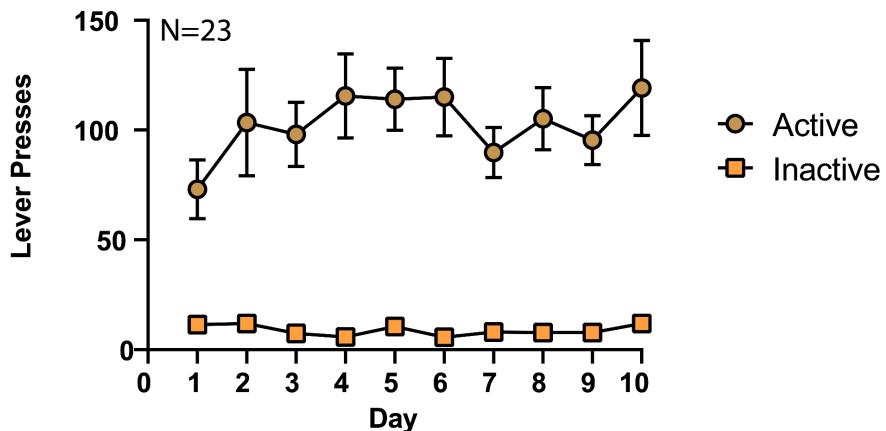
The critical threshold for statistically significant results was set at $p < .05$. The number of active and inactive lever presses made during the heroin-seeking test was analyzed separately using a one-way ANOVA to compare the mean lever presses between the 3 treatment groups. Total distance traveled during the locomotor test was analyzed using a two-way repeated-measures ANOVA with the between-subjects' factor of treatment group and the within-subjects' factor of time (6 x 10-minute intervals).

Data Integrity.

Six rats were removed due to failure to train, health issues, or catheter leakage. One rat was considered an outlier due to an extreme number of active lever responses made during the heroin self-administration training period (>2.5 SD above group average). Ten rats were removed from the statistical analysis due to technical issues (i.e., extreme noise and vibrations) where we believe that noise from the construction of a new building affected the behavior of the rats during the test. Therefore, the final analysis included 23 rats across the three experimental conditions: water ($n = 8$), vehicle ($n = 8$), CORT ($n = 7$).

Assumptions for a one-way ANOVA include normality, independence, and homogeneity of variance. None of these assumptions were violated following the inspection of the data. Assumptions for a two-way repeated-measures ANOVA include normality, independence, and sphericity. These assumptions were also not violated following the inspection of the data.

All rats learned to dissociate between the active and inactive lever and demonstrated reliable heroin self-administration. This learning is evident by the robust increase in active lever presses (see Figure 1) and a consistently low amount of inactive lever presses (see Figure 1) throughout the heroin self-administration training. Group Means and SEM for the number of active lever responses, inactive lever responses, infusions, and bodyweight during the last five days of heroin self-administration training, for each treatment group is presented in Table 1. There were no statistically significant differences between the different treatment groups on any of these variables.

Figure 1*Active and inactive lever pressing during self-administration training*

Note. The line graph shows a robust increase in active lever responses throughout the 10 days of heroin self-administration. Error bars represent standard error of the mean.

Table 1*Heroin Self-Administration Training*

	Infusions	Active Lever	Inactive Lever	Bodyweight
Water	36.10 ± 5.40	89.60 ± 17.47	10.18 ± 1.81	351.95 ± 6.45
Vehicle	38.60 ± 4.32	108.43 ± 14.65	8.40 ± 1.53	346.48 ± 4.46
CORT	36.00 ± 6.76	118.43 ± 29.34	7.00 ± 1.61	341.86 ± 8.15

Results

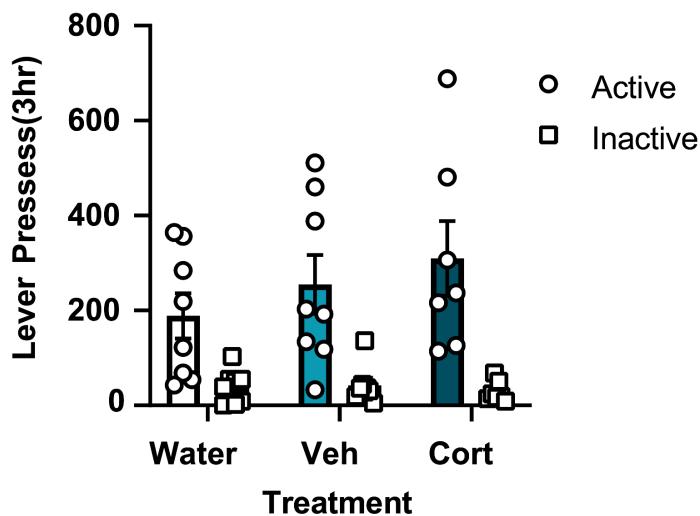
Heroin Seeking Test

A one-way ANOVA was conducted to determine if chronic corticosterone administration during the abstinence period (i.e., 15 days) had an impact on active and inactive lever responding during the heroin seeking test when compared to the control groups. The one-way ANOVA revealed no statistically significant differences between treatment groups for both active lever responses ($F(2, 20) = .92, p = .42, \eta^2 = 0.03$; see Figure 2) and inactive lever responses ($F(2, 20) = 0.31, p = .73, \eta^2 = .01$; see Figure 2). The model estimates for both one-way ANOVA's are presented in Appendix A. This suggests that all rats pressed the active and inactive lever at a similar rate, regardless of

the treatment that was administered. These results indicate that the hypothesis was not supported indicating that chronic corticosterone administration does not appear to increase heroin seeking following a period of prolonged abstinence.

Figure 2

Heroin seeking test after forced abstinence and corticosterone treatment



Note. The bar graph shows that the number of active lever responses during the heroin seeking slightly increased with corticosterone treatment, however this increase failed to reach statistical significance ($F_{(2, 20)} = 0.92, p = .42, \eta^2 = 0.03$). Responses on the inactive lever, as expected, did not differ across treatment conditions ($F_{(2,20)} = 0.31, p = .73, \eta^2 = 0.01$). Error bars represent standard error of the mean.

Although no statistically significant effects were detected, visual inspection of the data suggests a pattern of increased responses. To further analyze this relation, Cohen's d was calculated to assess the magnitude of the differences in active lever responses between the corticosterone treatment condition and the control treatment conditions: CORT vs. water, $d = 0.75$, CORT vs. vehicle $d = 0.30$, and vehicle vs. water $d = 0.45$. These effect sizes are relatively low compared to the effect sizes that are usually observed for the food restriction impact, revealing that although CORT increased active lever responses, this increase is largely attributed to the fact that the vehicle does have an effect on heroin seeking.

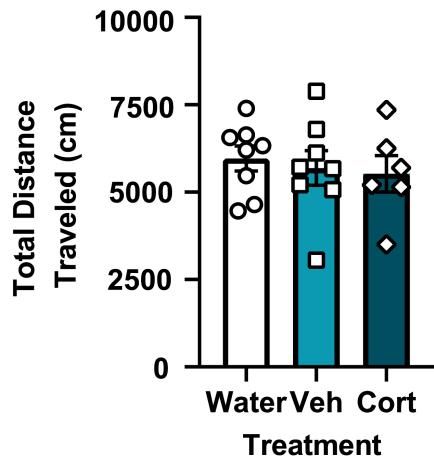
Locomotor Activity Test

Datum for the locomotor activity test were lost for one rat. Therefore, the final analysis included 22 rats across the three treatment conditions: water ($n = 8$), vehicle ($n = 8$), CORT ($n = 6$). The repeated-measures ANOVA revealed no statistically significant difference in total distance traveled between the treatment conditions ($F(2,19) = 0.42, p = .67, \eta^2 = 0.002$; see Figure 3), suggesting that chronic CORT administration did not have an impact on locomotor activity. There was a statistically significant main effect of time ($F(5,95) = 108.2, p < .001, \eta^2 = 0.85$), but no statistically significant time \times treatment interaction ($F(10,95) = 0.78, p < .64, \eta^2 = 0.07$). These results suggest that as the locomotor activity test progressed, all rats, regardless of their treatment

condition, gradually decreased their locomotor activity.

Figure 3

Total locomotor activity following corticosterone treatment



Note. The bar graph shows the total distance traveled. As expected, locomotor activity did not differ across the treatment conditions ($F_{(2,19)} = 0.42, p = .67, \eta^2 = 0.002$). Error bars represent standard error of the mean.

Discussion

The overall goal of this experiment was to assess the effect of chronic exogenous administration of corticosterone during abstinence on heroin seeking. We predicted that chronic corticosterone administration would increase heroin seeking compared to control groups. The results suggest that chronic exogenous corticosterone administration did not increase heroin-seeking following 15 days of abstinence, compared to vehicle-treated rats. Therefore, the main hypothesis that chronic corticosterone manipulation would augment heroin seeking was not supported. This was substantiated by the non-significant differences in lever responding between the treatment groups during the heroin seeking test. Corticosterone was administered orally, wherein the organic mechanism of the HPA axis was interfered with. When researching stress, we target this axis because its activation is linked with the occurrence of a stressful event [20]. The HPA axis organically releases corticosterone, once a stimulus triggers the hypothalamus, leading to the release of corticosterone [22].

Due to the exogenous administration of corticosterone at higher concentrations than peak plasma concentrations, there may have been an overwhelming effect to the system. This could have caused an attenuation of corticosterone release as well as other stress-related systems, which may have led to an inhibitory effect on the stress experienced by rats. Therefore, the rats in the corticosterone treatment group were possibly experiencing similar amounts of stress in relation to the other treatment conditions. Due to the possibility that all treatment groups were experiencing similar stress levels, this may have led them to have a similar amount of lever presses during the heroin seeking test. It is believed that heroin seeking increases following exposure to chronic food restriction due to the stress experienced by the rats [10]. Therefore, if the three treatment conditions experienced similar amounts of stress, there would be no reason to believe that there would

be differences in heroin seeking behaviours. When the HPA axis is stimulated the first neurohormone to be released is CRF, this cascade ends with the release of corticosterone from the adrenal glands [19]. A study by Shalev and colleagues demonstrated that CRF and not corticosterone was critically involved in the augmentation of heroin seeking following acute food deprivation [23]. Specifically, in this study when a CRF antagonist was administered to rats this attenuated subsequent heroin seeking. This effect was dose-dependent and resulted in a significant decrease in active lever responses compared to control. However, when rats had their adrenal glands surgically removed, a similar attenuation of heroin seeking following CRF administration was not observed. Therefore, due to the removal of adrenal glands and consequently no corticosterone release, this supports the role of CRF and not corticosterone when examining heroin seeking following acute food deprivation. We suspect that a similar effect would occur if replicated with animals that were chronically food-restricted, as opposed to food-deprived.

Another possibility for the lack of effect of corticosterone on heroin seeking is the difference in route of administration. More specifically, we administered corticosterone systemically, but local administration is an alternative. Research by Graf and colleagues demonstrated that animals injected with corticosterone directly into the nucleus accumbens, followed by cocaine administration showed a significant increase in cocaine seeking behaviour [24]. Therefore, this raises the possibility that local and not systemic administration results in an effect in drug seeking behavior. However, this research was conducted with cocaine, which has very different molecular effects compared to heroin. Therefore, local administration would need to be verified with heroin to validate the effect of corticosterone local administration. Thereby providing greater insight into the role that corticosterone may play in the alteration of heroin seeking following a period of prolonged abstinence.

This study is not without limitations. Firstly, because corticosterone was administered via the drinking water, we were unable to control the amount of volume consumed by each experimental animal. Therefore, the level of corticosterone varied among experimental animals. Secondly, as previously mentioned corticosterone is a stress-related hormone, secreted from the adrenal glands upon activation of the HPA axis. However, we exogenously administered corticosterone thereby interfering with the HPA mechanism. Rats were not exposed to a stressor. Lastly, ethanol was present in solution at a 2% concentration for both our vehicle and corticosterone treatment conditions. The consumption of ethanol may have influenced the heroin seeking behaviours of rats which may have had an impact on the results obtained in this study.

Despite these limitations, the results from this study provide us with a greater understanding into the role that corticosterone plays in the augmentation of heroin seeking following a prolonged period of abstinence. Chronic food restriction during abstinence in heroin trained rats induces increased heroin seeking, this effect was hypothesized to be mediated by elevated levels of corticosterone. However, we have indication that chronic exogenous corticosterone administration during an abstinence phase does not have a significant effect on rat's subsequent heroin seeking behaviours.

References

- [1] McLellan, A. T. (2017). Substance Misuse and Substance use Disorders: Why do they Matter in Healthcare? *Transactions of the American Clinical and Climatological Association*, 128, 112–130.
- [2] O'Brien, C. P., & McLellan, A. T. (1996). Myths about the treatment of addiction. *The Lancet*, 347, 207–274.
- [3] O'Brien, C. P., & Gardner, E. L. (2005). Critical assessment of how to study addiction and its treatment: human and non-human animal models. *Pharmacology & Therapeutics*, 108, 18–58., <https://doi.org/10.1016/j.pharmthera.2005.06.018>
- [4] Koob, G. F., & Volkow, N. D. (2016). Neurobiology of addiction: a neurocircuitry analysis. *The Lancet Psychiatry*, 3(8), 760–773. [https://doi.org/10.1016/S2215-0366\(16\)00104-8](https://doi.org/10.1016/S2215-0366(16)00104-8)
- [5] Smyth, B. P., Barry, J., Keenan, E., & Ducray, K. (2010). Lapse and relapse following inpatient treatment of opiate dependence. *Irish Medical Journal*, 103, 176–179.
- [6] Zickler, P. (2001). 33-year study finds lifelong, lethal consequences of heroin addiction. *NIDA Notes*, 16(4). https://archives.drugabuse.gov/NIDA_Notes/NNVol16N4/33year.html
- [7] de Wit, H. (1996). Priming effects with drugs and other reinforcers. *Experimental and Clinical Psychopharmacology*, 4(1), 5–10. <https://doi.org/10.1037/1064-1297.4.1.5>
- [8] Childress, A. R., Hole, A. V., Ehrman, R. N., Robbins, S. J., McLellan, A. T., & O'Brien, C. P. (1993). Cue reactivity and cue reactivity interventions in drug dependence. *NIDA Research Monograph*, 137, 73–95.
- [9] Sinha, R. (2001). How does stress increase risk of drug abuse and relapse. *Psychopharmacology*, 158(4), 343–359. <https://doi.org/10.1007/s002130100917>
- [10] D'Cunha, T. M., Sedki, F., Macri, J., Cosola, C., & Shalev, U. (2013). The effects of chronic food restriction on cue-induced heroin seeking in abstinent male rats. *Psychopharmacology*, 225, 241–250. <https://doi.org/10.1007/s00213-012-2810-1>
- [11] Franklin, J. C., Schiele, B. C., Brozek, J., & Keys, A. (1948). Observations on human behavior in experimental semistarvation and rehabilitation. *Journal of Clinical Psychology*, 4, 28–45. [https://doi.org/10.1002/1097-4679\(194801\)4:1](https://doi.org/10.1002/1097-4679(194801)4:1)
- [12] Cheskin, L. J., Hess, J. M., Henningfield, J., & A, G. D. (2005). Calorie restriction increases cigarette use in adult smokers. *Psychopharmacology*, 179, 430–436. <https://doi.org/10.1007/s00213-004-2037-x>
- [13] Goeders, N. (2003). The impact of stress on addiction. *European Neuropsychopharmacology*, 13(6), 435–441. <https://doi.org/10.1016/j.euroneuro.2003.08.004>
- [14] Shalev, U., Grimm, J. W., & Shaham, Y. (2002). Neurobiology of relapse to heroin and cocaine seeking: A review. *Pharmacological Review*, 54, 1–42.
- [15] Peck, J. A., & Ranaldi, R. (2014). Drug abstinence: Exploring animal models and behavioral

treatment strategies. *Psychopharmacology*, 231, 2045–2058. <https://doi.org/10.1007/s00213-014-3517-2>

[16] Shaham, Y., Shalev, U., Lu, L., De Wit, H., & Stewart, J. (2003). The reinstatement model of drug relapse: history, methodology and major findings. *Psychopharmacology*, 168(1–2), 3–20. <https://doi.org/10.1007/s00213-002-1224-x>

[17] Epstein, D. H., Preston, K. L., Stewart, J., & Shaham, Y. (2006). Toward a model of drug relapse: An assessment of the validity of the reinstatement procedure. *Psychopharmacology*, 189, 1–16. <https://doi.org/10.1007/s00213-006-0529-6>

[18] Reichel, C., & Bevins, R. (2009). Forced Abstinence Model of Relapse to Study Pharmacological Treatments of Substance Use Disorder. *Current Drug Abuse Reviewse*, 2(2), 184–194. <https://doi.org/10.2174/1874473710902020184>

[19] Meisch, R. A., & Carroll, M. E. (1987). *Oral Drug Self-Administration: Drugs as Reinforcers*. In Methods of Assessing the Reinforcing Properties of Abused Drugs (pp. 143–160). Springer New York. https://doi.org/10.1007/978-1-4612-4812-5_7

[20] Carr, K. D. (1996). Feeding, drug abuse, and the sensitization of reward by metabolic need. *Neurochemical Research*, 21(11), 1455–1467. <https://doi.org/10.1007/BF02532386>

[21] Silverman, M. N., Pearce, B. D., Biron, C. A., & Miller, A. H. (2005). Immune modulation of the hypothalamic-pituitary-adrenal (HPA) axis during viral infection. *Viral Immunology*, 18(1), 41–78. <https://doi.org/10.1089/vim.2005.18.41>

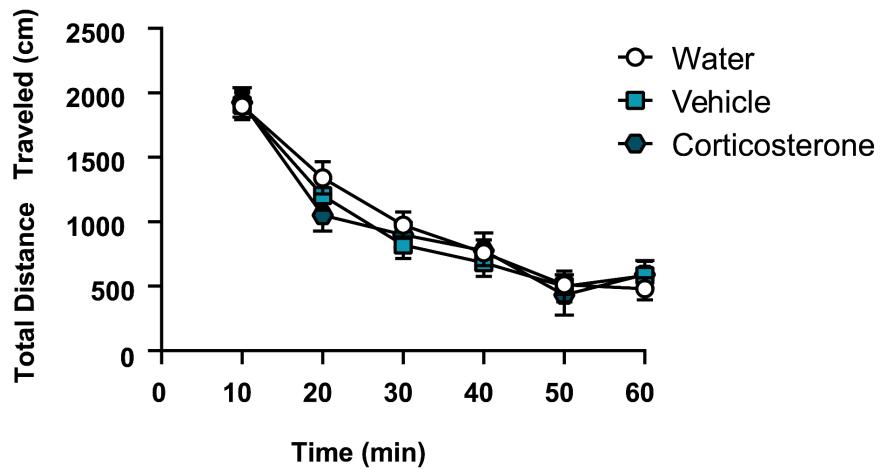
[22] Stephens, M. A., & Wand, G. (2012). Stress and the HPA axis: role of glucocorticoids in alcohol dependence. *Alcohol Research: Current Reviews*, 34(4), 468–483.

[23] Shaham, Y., Funk, D., & Erb, S. (1997). Corticotropin-releasing factor, but not corticosterone, is involved in stress-induced relapse to heroin-seeking in rats. *The Journal of Neuroscience*, 17(7), 2605–2614 <https://doi.org/10.1523/JNEUROSCI.17-07-02605.1997>.

[24] Graf, E. N., Wheeler, R. A., Baker, D. A., Ebbin, A. L., Hill, J. E., McReynolds, J. R., Robble, M. A., Vranjkovic, O., Wheeler, D. S., Mantsch, J. R., & Gasser, P. J. (2013). Corticosterone Acts in the Nucleus Accumbens to Enhance Dopamine Signaling and Potentiate Reinstatement of Cocaine Seeking. *Journal of Neuroscience*, 33(29), 11800–11810. <https://doi.org/10.1523/JNEUROSCI.1969-13.2013>

Appendix A

Locomotor activity across time following corticosterone treatment



Note. As expected, the locomotor activity across time decreased ($F(5, 95) = 108.2$, $p < .001$, $\eta^2 = 0.85$). There was not a significant difference across the treatment groups across time ($F(10, 95) = 0.78$, $p < .64$, $\eta^2 = 0.07$).

Neural Correlates of Appetitive Pavlovian Extinction *

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The infralimbic cortex (IL), prelimbic cortex (PL), nucleus accumbens core (NAcCore), and nucleus accumbens shell (NAcSh) have been previously implicated in operant conditioning and extinction, but less is known about their role in appetitive Pavlovian extinction. The objective of this study was to investigate c-fos immunoreactivity, an indirect marker of neuronal activity, in the IL, PL, NAcCore, medial NAcSh, and lateral NAcSh to assess their roles in early vs. late appetitive Pavlovian extinction. After appetitive Pavlovian conditioning, rats in the Paired, Inter-trial interval (ITI) Unpaired, and Home-cage (H-C) Unpaired training conditions underwent either one extinction or six extinction sessions. We observed similar c-fos immunoreactivity in the IL of Paired and ITI Unpaired conditions following one and six extinction sessions, whereas greater c-fos was found in the PL of Paired and ITI Unpaired rats following six extinction sessions. Together, these findings suggest that the IL, but not the PL, plays a role in the inhibition of conditioned responding through its continual activation in appetitive Pavlovian extinction.

Substance use disorders (SUDs) involve maladaptive behaviours which occur because of learned associations between the effects of the abused drug and discrete cues in the environment. Therefore, treatment for SUDs involves inhibiting the expression of these associations through a process known as extinction, a fundamental form of inhibitory learning. More precisely, extinction refers to a decrease in conditioned responding that is produced when the outcome an animal expects is withheld. Evidence suggests that this decrement in responding is not due to the original conditioned association being erased, but rather because animals learn a new and competing inhibitory association [1]. Accordingly, it is essential to gain a more extensive understanding of the neural mechanisms of appetitive extinction to develop better interventions for such maladaptive behaviours.

The Infralimbic Cortex to Nucleus Accumbens Shell Pathway in Appetitive Operant Extinction

In appetitive operant conditioning, the animal learns to perform an operant response (e.g., lever press) to receive a rewarding outcome (e.g., drug infusion) and then learn to inhibit responding during extinction. Previous work illustrated the important role of the infralimbic cortex (IL), but not the prelimbic cortex (PL) of the medial prefrontal cortex (mPFC), in facilitating appetitive operant extinction learning [2-4]. More specifically, the differential involvements of the mPFC subregions in extinction may be due to their specific projections to the nucleus accumbens (NAc), which is implicated in reward-motivated behaviours [5]. Findings indicate that the IL to nucleus accumbens shell (NAcSh) projection is involved in the extinction of operant conditioned responding, whereas the PL to nucleus accumbens core (NAcCore) projection is thought to be involved in promoting operant drug-seeking [6-8].

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The Infralimbic Cortex to Nucleus Accumbens Shell Pathway in Appetitive Pavlovian Extinction

Despite the evidence implicating the IL in appetitive operant extinction, less is known about its role in appetitive Pavlovian extinction. Because these are two different learning processes, the brain regions involved in operant extinction might differ from those in Pavlovian extinction [9]. In Pavlovian conditioning, the animal learns to predict the delivery of a positive unconditioned stimulus (US; e.g., sucrose) following the presentation of the conditioned stimulus (CS; e.g., auditory cue). In extinction, the CS is presented, but the US is no longer delivered, leading to a reduction in conditioned responding as measured by the number of port entries.

The IL and PL subregions of the mPFC seem to play different roles in appetitive Pavlovian extinction. Previous work has shown that activating the IL after extinction attenuates reinstatement, spontaneous recovery, and renewal [10] whereas lesioning of the IL before extinction training increases spontaneous recovery and reinstatement, suggesting that activity in the IL is sufficient to suppress appetitive Pavlovian conditioned responding after extinction [11]. Contrary to previous findings, inactivation of the IL immediately before the first extinction session seems to facilitate the acquisition of within-session extinction [9], and disrupted retrieval of the extinction memory, whereas PL inactivation had no effect [9] [12]. Together, these results suggest that the IL, but not the PL, plays an important role in the extinction of appetitive Pavlovian conditioning.

The opposite roles of the IL and PL in the extinction of Pavlovian conditioned responding may be related to their individual projections to the NAc. Ziminski and colleagues (2017) [13] found that neurons in the NAcSh are activated by the original CS-US association memory, and the number of the CS-activated neurons in the ensemble diminished after Pavlovian extinction as shown by lower c-fos expression. The diminished size of the original neuronal ensemble involved in conditioning could be explained by a new ensemble of neurons being recruited by extinction. This idea is consistent with the theory that extinction memories compete against the original CS-US association for expression. However, these limited findings underscore the necessity to further investigate the neural correlates of appetitive Pavlovian extinction learning.

The present study investigated the neural correlates of early vs. late appetitive Pavlovian extinction by comparing c-fos immunoreactivity [14] [15] in the IL, PL, NAcSh, and NAcCore. In the training phase, rats in the paired groups learned to form an association between a conditioned stimulus and sucrose solution delivery whereas the two control groups (ITI Unpaired and H-C Unpaired) received unpaired presentations of the conditioned and unconditioned stimuli. This acquisition phase was then followed by one or six extinction sessions, where the sucrose solution was no longer delivered.

We predicted greater c-fos immunoreactivity in the IL and the medial NAcSh since these regions have previously been implicated in the facilitation of appetitive extinction, as opposed to the PL and NAcCore. However, we predicted no effects of extinction on neural activity in the PL, lateral NAcSh and the NAcCore. We also expected greater c-fos expression in the IL and NAcSh of rats that underwent one extinction session, compared to those that underwent six extinction sessions. This prediction is supported by the Rescorla-Wagner model positing that maximal learning occurs the first time an event is surprising, in this case that is the first session where the CS is presented without the US [16]. Therefore, comparing the neuronal marker of c-fos expression following one and six extinction sessions allowed us to elucidate which brain regions are most

activated when a CS-US association is extinguished.

Method

Subjects

Thirty-eight Long-Evans male rats (220-240 grams; Charles River, Canada) were housed individually in polycarbonate cages containing a nylabone (Nylabones; Bio-Serv, Flemington, NJ), a polycarbonate tunnel (Rat Retreats, Bio-Serv), beta-chip bedding (Aspen Sani chips; Envigo, Indianapolis, IN) and shredded paper used for enrichment. The cages were maintained on a 12-hour light/ dark cycle with the lights on at 7:00 in a colony room. All rats had unlimited access to standard chow and water. All procedures performed in this experiment were approved by the Concordia University Animal Care Committee and respected the guidelines of the Canadian Council on Animal Care.

Apparatus

Behavioural testing occurred in similar conditioning chambers during each replicate of the experiment (ENV-009A, Med Associates Inc., St-Albans, VT, USA), located in a different room than the colony room. In each behavioral chamber, the floors were composed of stainless-steel rods (ENV-009 A-GF), there was a dual cup fluid port (ENV-200R3AM), a white-noise speaker (ENV-225SM), and a house light (75W, 100 mA, EV-215M) that signaled the beginning and end of each session. A sucrose solution was delivered into the fluid port via a syringe attached to a pump (PHM-100). The frequency of port entries into the fluid port was monitored by an infrared detector unit (ENV-254CB). All experiments were controlled by a computer with Med Associates interface and software (Med-PC for Windows, version IV; Med Associates Inc.).

Behavioral Procedures

Home-cage sucrose pre-exposure. 10% (w/v) sucrose and water was made available to rats in their home cage for 48 hours. Both bottles were weighed after 24 and 48 hours to measure the rats' preference of sucrose over water. The groups were counterbalanced based on body weight, sucrose preference and sucrose consumption into six experimental groups [9]: 6 Extinction (Ext) Paired ($n = 7$), 1 Ext Paired ($n = 7$), 6 Ext ITI Unpaired ($n = 6$), 1 Ext ITI Unpaired ($n = 6$), 6 Ext H-C Unpaired ($n = 6$), and 1 Ext H-C Unpaired ($n = 6$).

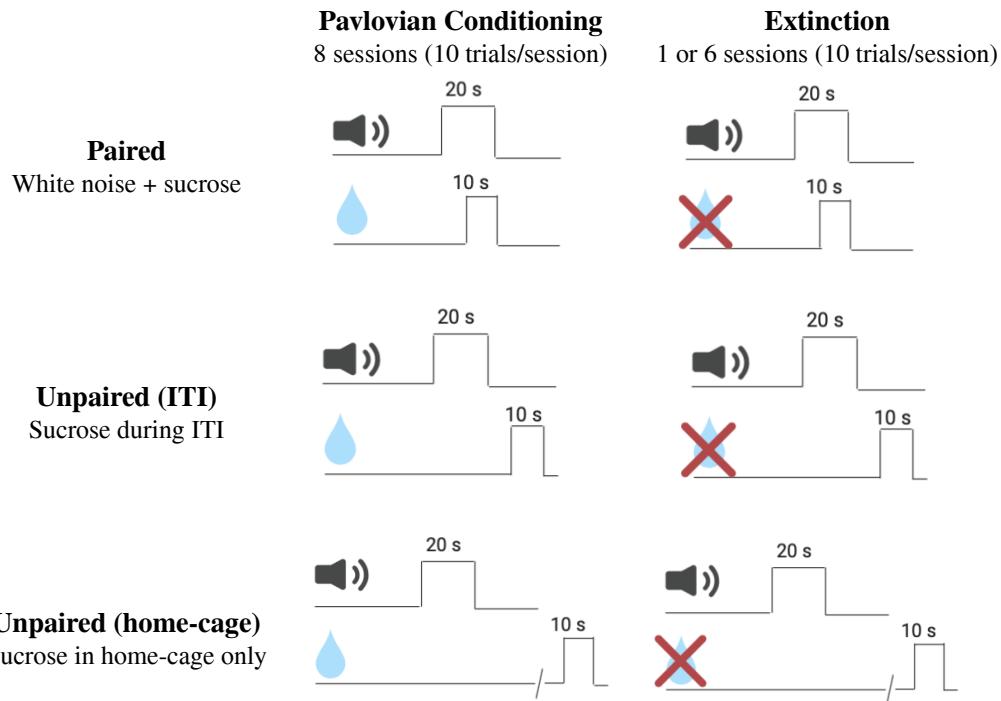
Habituation. After sucrose pre-exposure, the rats were habituated to the behavioural room and conditioning chamber for 20 minutes per day over 3 days.

Appetitive Pavlovian conditioning. The six experimental groups of rats received eight, 57-minutes Pavlovian conditioning sessions. After a 2-minute delay, the house light turned on, signaling the start of the session. During each session, a 20 second auditory stimulus (white noise) was presented ten times and was followed by a 120-, 240- or 360-second inter-trial interval (ITI). Rats in the Paired condition received a 0.3ml sucrose reward (10% w/v) after 10 seconds of stimulus presentation, while rats in the ITI Unpaired condition received the reward half-way through the variable ITI.

Rats in the H-C Unpaired condition received sucrose delivery in their home-cage at a random time from 1 hour to up to 4 hours after the session. The formation of the CS-US association was measured by the number of fluid port entries (see Figure 1).

Figure 1

Experimental design



Note. Rats first underwent a home-cage sucrose pre-exposure phase and then they were divided into six groups: 6 Ext Paired, 1 Ext Paired, 6 Ext ITI Unpaired, 1 Ext ITI Unpaired, 6 Ext H-C Unpaired and 1 Ext H-C Unpaired. Thirty-eight rats underwent eight daily, consecutive appetitive Pavlovian conditioning sessions. Animals in the Paired condition were exposed to 10 presentations of a white-noise cue (CS), which co-terminated with the delivery of sucrose (US) into a fluid port. Animals in the ITI Unpaired condition were exposed to 10 CS presentations followed by sucrose presentation during the ITI. Animals in the H-C Unpaired condition, were exposed to 10 CS presentations followed by sucrose delivery in their home-cage at a random time after the session. After Pavlovian conditioning, they underwent either 1 extinction session or six daily, consecutive extinction sessions during which sucrose was not delivered.

Extinction. During extinction, the white-noise was presented, but the sucrose was no longer delivered. Rats were either assigned to the 6 Ext group, receiving six extinction sessions during six consecutive days, or the 1 Ext group, receiving one extinction session.

Tissue Processing & C-Fos Immunohistochemistry

The rats were anesthetized with an injection of EuthanylTM 90 minutes after the start of the final extinction session followed by transcardiac perfusions using 4% paraformaldehyde and phosphate buffer saline. The brains were stored at -80°C until they were sectioned. Coronal sections of the brains were cut into six adjacent series at 40 µm using a cryostat (Microm HM 505E). The sections were then stored in cryoprotectant at -20°C until tissue processing using c-fos immunohistochemistry. One coronal series of each brain was processed for c-fos immunoreactivity, using

a standard immunohistochemical procedure.

Microscopy and Cell Counting

The anatomical regions of interest in this experiment for statistical comparisons were the mPFC and the ventral striatum. The images of c-fos-stained sections were digitally captured at 20X magnification using a Nikon Eclipse TiE inverted C2 confocal microscope operated using NIS-Elements software. The subregions of interest in the mPFC (IL and PL) and in the ventral striatum (medial NAcSh, lateral NAcSh, and NAcCore) were then identified using the George Paxinos and Charles Watson (2007) [17] rat brain atlas. The density of c-fos-labeled nuclei was analyzed as an estimate of the number of immunoreactive cells per subregion. The normalized density was then calculated by dividing the density by the averaged density of the H-C Unpaired of the same group (6 Ext vs. 1 Ext) and multiplying this value by 100. The two experimenters were blind to the experimental conditions of each rat during quantification.

Statistical Analyses

An $8 \times 2 \times 3 \times 2$ mixed ANOVA was used to assess the acquisition of Pavlovian appetitive conditioned responding. The within-subjects factors were Session (1 to 8), and Interval (pre-CS vs. CS); and the between-subjects factors were Training (Paired, ITI Unpaired, H-C Unpaired), and Group (6 Ext, 1 Ext). A $6 \times 2 \times 3$ mixed ANOVA across the six extinction sessions was also used to assess extinction of conditioned responding. The within-subjects factors were Session (1 to 6), and Interval (pre-CS vs. CS), and the between-subjects factor was Training (Paired, ITI Unpaired, H-C Unpaired). The dependent variable was the number of port entries for both acquisition and extinction analyses. To evaluate the final extinction session across all conditions and groups that correspond to when the brains were collected for c-fos immunoreactivity analysis, a two-way ANOVA was conducted. The dependent variable was the normalized number of port entries which represents the number of port entries made during the CS subtracted by the number of port entries made during the pre-CS, accounting for spontaneous, unconditioned port entries [10].

A two-way ANOVA was used to assess c-fos immunoreactivity for each subregion of interest separately. The between-subjects factors were Group (1 Ext, 6 Ext) and Training (Paired, ITI Unpaired, H-C Unpaired). The dependent variable was the normalized density of c-fos nuclei in each region of interest. Greenhouse-Geisser correction were reported following violations of sphericity. All data analyses were conducted using SPSS (IBM SPSS Statistics 23.0). Additionally, results were considered statistically significant at $p < .05$.

Results

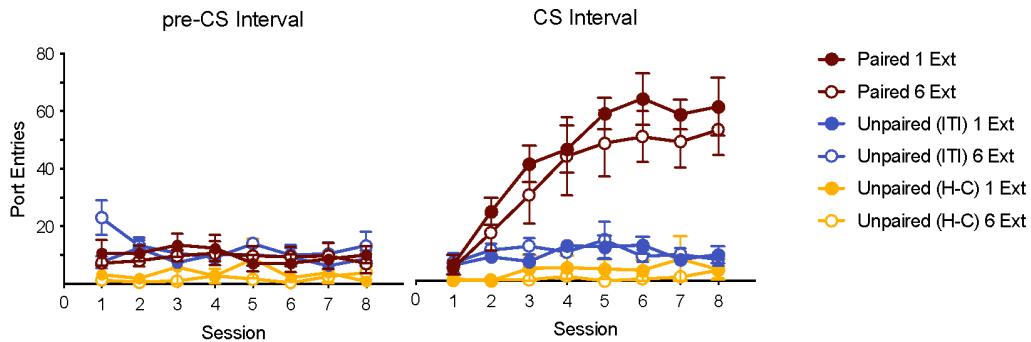
Summary Statistics

Acquisition. The mixed ANOVA revealed a main effect of Session [$F(4.42, 141.49) = 7.45, p < .005, \eta^2 = .03$; see Figure 2]. There was also a significant main effect of Training on the number of port entries [$F(2,32) = 49.92, p < .005, \eta^2 = .26$]. Bonferroni-corrected post-hoc comparisons revealed that the Paired group made significantly more port entries than both Unpaired groups ($p < .005$). This difference is because only the Paired training condition was exposed to a white-noise CS that co-terminated with sucrose. The ITI Unpaired training condition also displayed a significantly higher numbers of port entries than the H-C Unpaired training condition ($p = .009$). This result can be explained by the fact that the ITI Unpaired training condition received sucrose in the

conditioning chamber during the intertrial interval, whereas the H-C Unpaired condition received sucrose in the home-cage only. A main effect of Interval was also uncovered [$F(1, 32) = 48.04, p < .005, \eta^2 = .07$]. Bonferroni-corrected post-hoc comparisons showed that the number of port entries during the pre-CS was significantly different than during the CS ($p < .005$), with more port entries made during the CS interval. The analysis also detected an Interval x Session x Training interaction [$F(7.01, 112.16) = 9.38, p < .005, \eta^2 = .05$], indicating that the Interval x Session interaction described previously differed between the three training conditions. Bonferroni-corrected post-hoc comparisons discerned that port entries significantly increased during the CS for the Paired condition across conditioning sessions 1 to 8 ($p < .005$). However, no significant differences were found in the number of port entries of ITI Unpaired and H-C Unpaired during the CS across sessions 1 to 8 ($p = 1.000$).

Figure 2

Acquisition of appetitive Pavlovian conditioning.

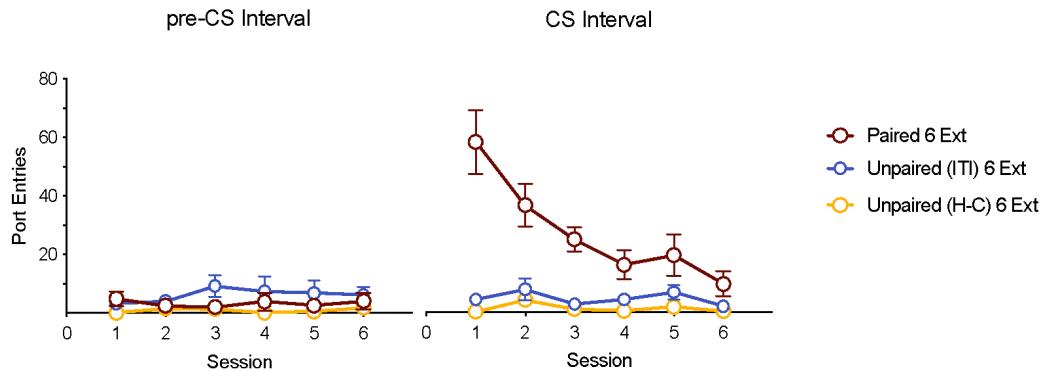


Note. This figure depicts the pre-CS and CS port entries made across conditioning sessions. An Interval x Session x Training interaction was detected, whereby responding to the discrete sucrose cue increased during the CS interval across conditioning sessions for the Paired conditions ($p < .005$), but not for either of the Unpaired conditions ($p = 1.000$). Port entries also did not differ during the pre-CS interval across sessions for the training conditions ($p = 1.000$). Error bars represent the SEM.

Extinction. The three-way mixed ANOVA revealed a main effect of Session [$F(2.91, 46.51) = 4.69, p = .007, \eta^2 = .03$; see Figure 3]. However, Bonferroni-corrected post-hoc comparisons showed that the number of port entries made in the 1st session was not significantly different compared to the 6th session ($p = .118$). A main effect of Training was also detected [$F(2,16) = 13.97, p < .005, \eta^2 = .20$]. Bonferroni-corrected post-hoc comparisons showed that the number of port entries was significantly different for the Paired compared to the ITI Unpaired ($p = .008$) and H-C Unpaired ($p < .005$) training conditions, with more port entries exhibited by the Paired condition. Additionally, the analysis revealed a main effect of Interval [$F(1,16) = 25.44, p < .005, \eta^2 = .07$]. Bonferroni-corrected post-hoc comparisons showed that the number of port entries made during the CS was significantly higher than those made during the pre-CS ($p < .005$).

Figure 3

Extinction of appetitive Pavlovian conditioned responding



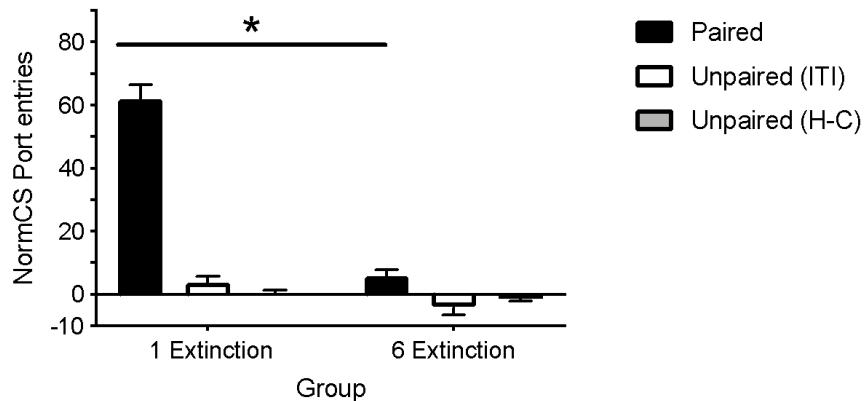
Note. This figure depicts the pre-CS and CS port entries made across extinction sessions. An Interval x Session x Training interaction was detected, whereby responding to the discrete sucrose cue decreased during the CS interval across extinction sessions for the Paired condition ($p < .005$), but not for either of the Unpaired conditions ($p = 1.000$). The number of port entries did not differ during the pre-CS interval across extinction sessions for the three training conditions ($p > .05$). Error bars represent the SEM.

The three-way mixed ANOVA also revealed an Interval x Session x Training interaction [$F(5.50, 43.99) = 5.73, p < .005, \eta^2 = .06$], indicating that the Interval x Session interaction described previously differed between the three training conditions. Bonferroni-corrected post-hoc comparisons showed that port entries significantly decreased during the CS for the Paired condition across extinction session 1 to 6 ($p < .05$). However, there was no significant difference across extinction sessions during the CS for both Unpaired groups ($p > .05$). Additionally, the number of port entries did not differ during the pre-CS across extinction sessions for the three training conditions ($p > .05$).

Final extinction. The two-way ANOVA revealed a main effect of Group [$F(1,32) = 102.91, p < .005, \eta^2 = .16$; see Figure 4]. Bonferroni-corrected post-hoc comparisons showed that the number of normalized port entries during the final extinction session was significantly higher for the subjects that had 1 extinction session compared to those that had 6 extinction sessions ($p < .005$). Additionally, the analysis detected a main effect of Training [$F(2,32) = 113.86, p < .005, \eta^2 = .36$], indicating that the training condition had a significant influence on the normalized number of port entries made during the final extinction session. Bonferroni-corrected post-hoc comparisons revealed that the Paired training condition made significantly more normalized port entries than the ITI Unpaired, and H-C Unpaired ($p < .005$) training conditions.

Figure 4

Number of normalized port entries made during the final extinction session following appetitive Pavlovian conditioning.



Note. This figure depicts the number of normalized port entries made during the final extinction session for each training condition. A Group x Training interaction was detected, whereby the number of normalized port entries was significantly higher for subjects in the Paired 1 Ext group compared to the Paired 6 Ext group ($p < .005$). No differences were found between the ITI Unpaired 1 Ext compared to ITI Unpaired 6 Ext group ($p = .051$) or for subjects in the H-C Unpaired 1 Ext compared to H-C Unpaired 6 Ext group ($p = .700$). Error bars represent the SEM. (*) Significant difference between Paired 1 Ext group and Paired 6 Ext group (Two-way ANOVA, $p < .05$).

Finally, the analysis discerned a Group x Training interaction [$F(2,32) = 68.02, p < .005, \eta^2 = .21$]. This result indicates that the number of normalized port entries made during the final extinction session across the different levels of group differed between the three training conditions. Bonferroni-corrected post-hoc comparisons showed that normalized port entries were significantly higher for the Paired training condition in the 1 Ext group compared to the 6 Ext group ($p < .005$). However, there were no significant differences for the ITI Unpaired 1 Ext group vs. 6 Ext group ($p = .051$), nor the H-C Unpaired 1 Ext vs. 6 Ext group ($p = .700$).

C-fos immunoreactivity. The c-fos immunoreactivity was analyzed in the mPFC (IL, PL) and the ventral striatum (NAcCore, medial NAcSh, lateral NAcSh).

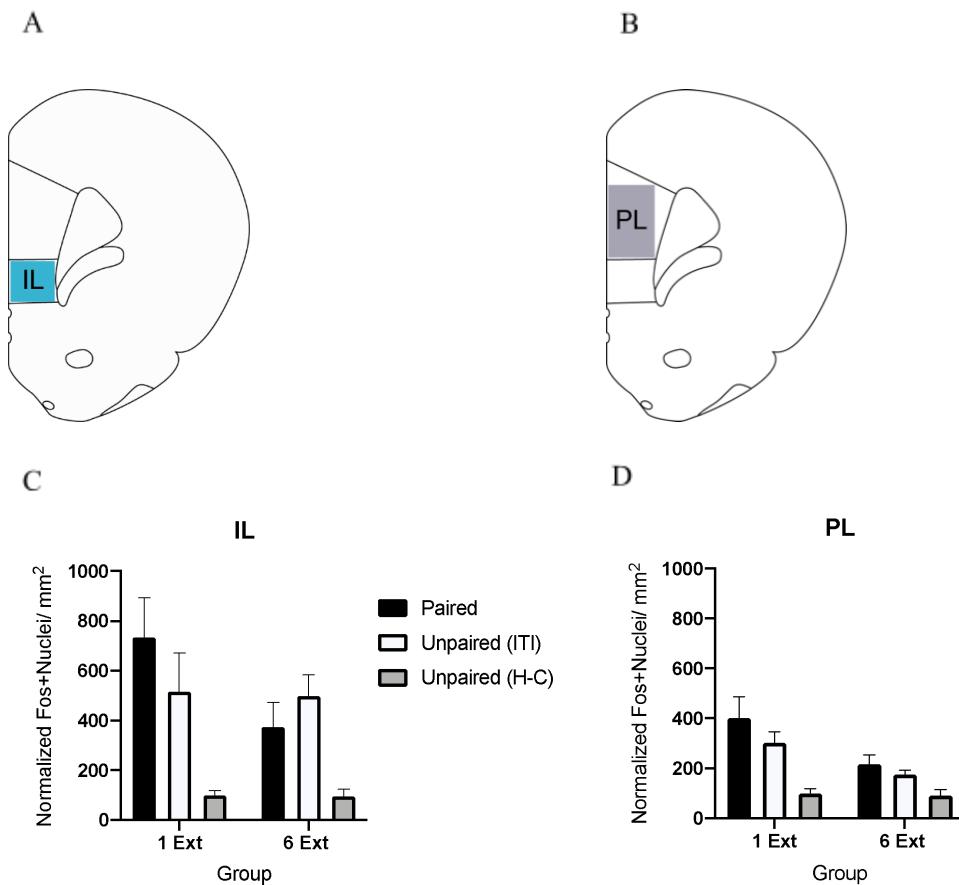
Medial prefrontal cortex.

The two-way ANOVAs revealed a main effect of Training in the IL [$F(2, 32) = 10.43, p < .005, \eta^2 = .36$; see Figure 5C]. Bonferroni-corrected post-hoc comparisons showed that the normalized density of c-fos nuclei in the IL was significantly higher for the Paired ($p = .001$) and ITI Unpaired ($p = .003$) conditions compared to the H-C Unpaired condition, while there was no significant difference in the normalized density of c-fos nuclei between the Paired and ITI Unpaired conditions ($p = 1.000$). There was no significant main effect of Group [$F(1, 32) = 2.03, p = .164, \eta^2 = .04$], nor a significant interaction between Group and Training [$F(2, 32) = 1.80, p = .182, \eta^2 = .06$] on the normalized density of c-fos nuclei in the IL. Further, a main effect of Training was also detected in the PL [$F(2, 32) = 10.11, p < .005, \eta^2 = .32$; see Figure 5D]. Bonferroni-corrected post-hoc comparisons showed that the normalized density of c-fos nuclei was significantly higher for the Paired ($p < .005$) and ITI Unpaired ($p = .022$) conditions compared to the H-C Unpaired condition. Ad-

ditionally, the analysis detected a Group main effect in the PL [$F(1, 32) = 7.23, p = .011, \eta^2 = .12$]. Bonferroni-corrected post-hoc comparisons showed that the normalized density of c-fos nuclei was significantly higher for the 1 Ext group compared to the 6 Ext group ($p = .011$). There was no significant Group x Training interaction [$F(2, 32) = 1.74, p = .192, \eta^2 = .06$] in the PL, indicating that the normalized density of c-fos nuclei across the different levels of group did not differ between the three training conditions.

Figure 5

c-fos immunoreactivity in the medial prefrontal cortex



Note. **A.** Normalized number of c-fos-immunoreactive nuclei per square millimeter in the infralimbic cortex (IL). **B.** Normalized number of c-fos-immunoreactive nuclei per square millimeter in the prelimbic cortex (PL). **C.** Mean \pm SEM normalized number of c-fos immunoreactive nuclei per square millimeter in the IL. **D.** Mean \pm SEM normalized number of c-fos immunoreactive nuclei per square millimeter in the PL. A Group main effect in the prelimbic cortex was detected, whereby the normalized density of c-fos nuclei was significantly higher for the 1 Ext group compared to the 6 Ext group ($p = .011$).

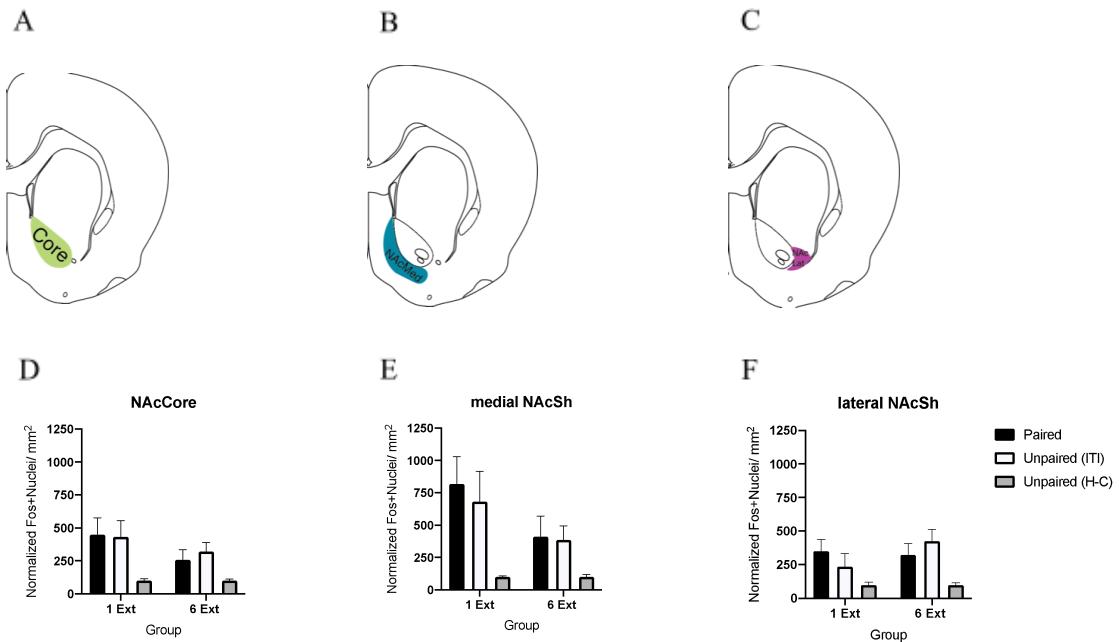
Ventral striatum.

The two-way ANOVAs revealed a main effect of Training in the NAcCore [$F(2,32) = 6.08, p = .006, \eta^2 = .26$; see Figure 6D], the medial NAcSh [$F(2,32) = 6.18, p = .005, \eta^2 = .25$; see Figure 6E], and in the lateral NAcSh [$F(2,32) = 6.51, p = .004, \eta^2 = .27$; see Figure 6F]. Bonferroni-corrected

post-hoc comparisons showed that the normalized density of c-fos nuclei in the NAcCore was significantly higher for the Paired ($p = .017$) and ITI Unpaired ($p = .012$) conditions compared to the H-C Unpaired training condition. Additionally, Bonferroni-corrected post-hoc comparisons showed that the normalized density of c-fos nuclei in the medial NAcSh was significantly higher for the Paired ($p = .007$) and ITI Unpaired ($p = .033$) compared to the H-C Unpaired training condition. The Paired and ITI Unpaired conditions did not significantly differ in their normalized density of c-fos nuclei ($p = 1.000$). Moreover, Bonferroni-corrected post-hoc comparisons showed that the normalized density of c-fos nuclei in the lateral NAcSh was significantly higher for the Paired ($p = .008$) and ITI Unpaired ($p = .014$) conditions compared to the H-C Unpaired training condition. There was also no significant difference in the normalized density of c-fos nuclei between the Paired and ITI Unpaired conditions ($p = 1.000$). Overall, these results indicate that the training condition had a similar effect on the normalized density of c-fos nuclei in each of these regions of interest. Furthermore, a trend towards a Group main effect in the medial NAcSh was also detected [$F(1, 32) = 3.33, p = .077, \eta^2 = .07$]. The analyses did not, however, detect a main effect of Group for the NAcCore [$F(1, 32) = 2.02, p = .165, \eta^2 = .04$], or the lateral NAcSh [$F(1, 32) = .85, p = .364, \eta^2 = .02$].

Figure 6

c-fos immunoreactivity in the ventral striatum



Note. **A.** Normalized number of c-fos-immunoreactive nuclei per square millimeter in the nucleus accumbens core (NAcCore). **B.** Normalized number of c-fos-immunoreactive nuclei per square millimeter in the medial nucleus accumbens shell (medial NAcSh). **C.** Normalized number of c-fos-immunoreactive nuclei per square millimeter in the lateral nucleus accumbens shell (lateral NAcSh). **D.** Mean \pm SEM normalized number of c-fos-immunoreactive nuclei per square millimeter in the NAcCore. **E.** Mean \pm SEM normalized number of c-fos-immunoreactive nuclei per square millimeter in the medial NAcSh. The two-way ANOVA revealed a trend main effect of Group on the dependent variable in the medial NAcSh ($p = .077$). **F.** Mean \pm SEM normalized number of c-fos-immunoreactive nuclei per square millimeter in the lateral NAcSh.

Discussion

In the present study, we examined neural activity in regions of the mPFC and ventral striatum following early and late extinction of appetitive Pavlovian conditioned responding using c-fos immunohistochemistry. More specifically, we sought to identify differences in neural activity in the IL, PL, and subregions of the NAc, following one or six extinction sessions.

The normalized density of c-fos nuclei was significantly higher for the 1 Ext group compared to the 6 Ext group, suggesting that the PL was more active in early extinction than late extinction. Thus, our results support the hypothesis of the opposing roles of the IL and PL subregions of the mPFC as demonstrated in both aversive extinction [18] and appetitive extinction paradigms [12]. This is consistent with our results, suggesting that as inhibitory learning occurs in extinction training, the PL is no longer recruited to facilitate the expression of conditioned responding.

Additionally, the normalized density of c-fos nuclei was similar between the 1 Ext and 6 Ext groups for the IL, suggesting that the number of extinction sessions did not influence c-fos immunoreactivity. We predicted greater c-fos expression in the IL and NAcSh of rats who underwent early extinction compared to those who underwent late extinction. Our data do not suggest a differential involvement of the IL during early compared to late extinction training as was found in an appetitive operant extinction [4]. In our experiment, the IL subregion of the medial PFC was continually active to inhibit conditioned responding during appetitive Pavlovian extinction. One possibility for this difference is that the IL mediates extinction differently in appetitive operant compared to appetitive Pavlovian extinction. Thus, these results provide novel information on the role of the IL in appetitive Pavlovian extinction and highlights potential differences in the neural correlates of the two types of extinction learning.

Previous research has also shown that the subregions of the NAc play differential roles in extinction learning explained by the projections they received from different regions of the medial PFC. Particularly, the NAcSh mediates appetitive extinction via its input from the IL, whereas the NAcCore promotes appetitive conditioned responding through its input from the PL [8, 6, 2]. Therefore, we expected higher c-fos expression in the NAcSh compared to the NAcCore. Contrary to our expectation, no differences of c-fos expression were found following early vs. late extinction for any regions of the NAc for the Paired training condition. Although, not statistically significant, upon visual inspection of the data, we did observe greater c-fos expression in the IL and medial NAcSh, compared to the other regions. Thus, this result seems to align with previous research supporting the role of the IL to NacSh projection in extinction, but further research should verify this result. Nevertheless, it is important to keep in mind that c-fos immunoreactivity alone cannot determine neural pathways, but region activation studies could. Similar to the c-fos immunoreactivity results observed for the IL, we did not find any c-fos expression differences following early vs. late extinction training for the medial NAcSh, suggesting that the continual activation of this subregion of the NAc is necessary for the inhibition of conditioned responding.

Interestingly, greater c-fos expression was found in the regions of the mPFC, and ventral striatum of Paired and ITI Unpaired conditions, compared to the H-C Unpaired condition. We had hypothesized that both Unpaired control conditions would show low levels of c-fos immunoreactivity compared to the Paired conditions following extinction training. This was because the ITI Unpaired and H-C Unpaired conditions did not acquire the CS-US association during conditioning, and therefore did not extinguish this association when sucrose was omitted. However, the

rats in the ITI Unpaired condition showed similar patterns of neural activation to the Paired condition. This suggests that rats in the ITI Unpaired condition may have acquired and extinguished a learned association leading to c-fos expression. Rather than learning a CS-US association, the ITI Unpaired rats may have learned a context-US association, an association between the context of the conditioning chamber and the sucrose delivery during the intertrial interval. For this reason, it was crucial to include the H-C Unpaired control group, which never learned a context-US association, to compare baseline c-fos immunoreactivity in these regions to conditions that underwent extinction of either a CS-US association, or a context-US association.

Limitations

One limitation of the present study is the use of c-fos immunoreactivity as an indirect marker of neuronal activity and the assumption that little or no c-fos protein is expressed under baseline conditions, which allows visualization of active neurons. Further, c-fos expression alone does not provide information about brain connectivity. Therefore, the brain pathways of learning and memory can be investigated only by combining c-fos immunoreactivity with other methods such as retrograde tracers [19]. Additionally, stimulus activation does not always lead to the activation of immediate early genes that lead to the transcription of the c-fos protein in neurons. Therefore, a lack of c-fos expression does not necessarily translate into lack of neuronal activity.

Future Directions

Future research should further investigate appetitive Pavlovian extinction using optogenetics which allows for direct manipulation of the neural activity of regions of interest with high temporal resolution. Importantly, this method would enable us to investigate not only regions, but pathways in the brain, such as the IL-NAcSh pathway in appetitive Pavlovian extinction.

References

- [1] Brooks, D., & Bouton, M. (1993). A retrieval cue for extinction attenuates spontaneous recovery. *Journal of Experimental Psychology: Animal Behavior Processes*, 19(1), 77-89. doi:10.1037/0097-7403.19.1.77
- [2] Peters, J., LaLumiere, R.T., & Kalivas, P.W. (2008). Infralimbic prefrontal cortex is Responsible for inhibiting cocaine seeking in extinguished rats. *Journal of Neuroscience*, 28(23), 6046-6053. doi: 10.1523/jneurosci.1045-08.2008
- [3] Chen, W., Wang, Y., Sun, A., Zhou, L., Xu, W., Zhu, H., & Liu, H. (2016). Activation of AMPA receptor in the infralimbic cortex facilitates extinction and attenuates the heroin-seeking behavior in rats. *Neuroscience Letters*, 612, 126–131. doi: 10.1016/j.neulet.2015.11.024
- [4] Warren, B., Mendoza, M., Cruz, F., Leao, R., Caprioli, D., Rubio, F,... & Hope, B. (2016). Distinct Fos expressing neuronal ensembles in the ventromedial prefrontal cortex mediate food reward and extinction memories. *Journal of Neuroscience*, 36(25), 6691-6703. doi: 10.1523/jneurosci.0140-16.2016
- [5] Kalivas, P., & Volkow, N. (2005). The neural basis of addiction: A pathology of motivation and

choice. *The American Journal of Psychiatry*, 162, 1403–1413. doi: 10.1176/appi.ajp.162.8.1403

[6] Cruz, F., Babin K., Leao R., Goldart E., Bossert J., Shaham Y., & Hope B. (2014). Role of nucleus accumbens shell neuronal ensembles in context-induced reinstatement of cocaine-seeking. *Journal of Neuroscience*, 34(22), 7437–7446. doi: 10.1523/jneurosci.0238-14.2014

[7] Peters, J., Kalivas, P., & Quirk, G. (2009). Extinction circuits for fear and addiction overlap in prefrontal cortex. *Learning and Memory*, 16(787), 279-288. doi: 10.1101/lm.1041309.16

[8] Augur, I., Wyckoff, A., Aston-Jones, G., Kalivas, P., & Peters, J. (2016). Chemogenetic activation of an extinction neural circuit reduces cue-induced reinstatement of cocaine seeking. *Journal of Neuroscience*, 36(39), 10174-10180. doi: 10.1523/jneurosci.0773-16.2016

[9] Mendoza, J., Sanio, C., & Chaudhri, N. (2015). Inactivating the infralimbic but not Prelimbic medial prefrontal cortex facilitates the extinction of appetitive Pavlovian conditioning in Long-Evans rats. *Neurobiology of Learning and Memory*, 118, 198-208. doi: 10.1016/j.nlm.2014.12.006

[10] Villaruel, F., Lacroix, F., Sanio, C., Sparks, D., Chapman, C., & Chaudhri, N. (2018). Optogenetic activation of the infralimbic cortex suppresses the return of appetitive Pavlovian conditioned responding following extinction. *Cerebral Cortex*, 28(12), 4210-4221. doi: 10.1093/cercor/bhx275

[11] Rohdes, S., & Killcross, S. (2004). Lesions of rat infralimbic cortex enhance recovery and reinstatement of an appetitive Pavlovian response. *Learning and Memory*, 11(5), 611–616. doi: 10.1101/lm.79704

[12] Lay, B., Nicolosi, M., Usypchuk, A., Esber, G., & Iordanova, M. (2019). Dissociation of appetitive overexpectation and extinction in the infralimbic cortex. *Cerebral Cortex*, 29(4), 3687- 3701. doi:10.1093/cercor/bhy248.

[13] Zimniski, J., Hessler, S., Margetts-Smith, G., Sieburg, M., Crombag, H., & Koya, E. (2017). Changes in appetitive associative strength modulates nucleus accumbens, but not orbitofrontal cortex neuronal ensemble excitability. *Journal of Neuroscience*, 37(12), 3160-3170. doi: 10.1523/jneurosci.3766-16.2017

[14] Brenhouse, H., & Stellar, J., (2006). c-Fos and deltaFosB expression are differentially altered in distinct subregions of the nucleus accumbens shell in cocaine-sensitized rats. *Neuroscience*, 137(3), 773-80. doi: 10.1016/j.neuroscience.2005.09.039

[15] Hope, B., Hyman, S., & Nestler, J. (1992). Regulation of immediate early gene expression and AP-1 binding in the rat nucleus accumbens by chronic cocaine. *Proceedings of the National Academy of Sciences*, 89(13), 5764-5768. doi:10.1073/pnas.89.13.5764

[16] Rescorla, R., & Wagner, A. (1972). A theory of Pavlovian conditioning: Variations in the effectiveness of reinforcement and nonreinforcement. In AH. Black & W.F. Prokasy (eds.), *Classical Conditioning II: current research and theory* (pp. 64–99). New York, NY: Appleton-Century-Crofts

[17] Paxinos, G., & Watson, C. (2007). The Rat Brain in Stereotaxic Coordinates. Academic: New York

[18] Sierra-Mercado, D., Padilla-Coreano, N., & Quirk, G.J. (2011). Dissociable roles of prelimbic

and infralimbic cortices, ventral hippocampus, and basolateral amygdala in the expression and extinction of conditioned fear. *Neuropsychopharmacology*. 36(2), 529-38. doi: 10.1038/npp.2010.184.

[19] Keefer S., & Petrovich G. (2017) Distinct recruitment of basolateral amygdala-medial pre-frontal cortex pathways across Pavlovian appetitive conditioning. *Neurobiology of Learning and Memory*, 141, 27-32. doi: 10.1016/j.nlm.2017.03.006

Can We Manipulate Sleep Spindles? *

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Faulty memory consolidation during sleep might be a contributing factor to age-related memory decline. The timing between two electrical brain activities, slow oscillations (SO), and sleep spindles is thought to be important in this process. Studies have shown that precisely timed sound stimulation presented during SO up-states can enhance SO amplitude, which improves participants' memory consolidation. However, it is not known if sound can influence the amplitude of sleep spindles synchronized to SO and if the effect differs when the sound is presented at different spindle phases. In this within-subjects design, 4 young adults received sound stimulation during sleep timed to hit peaks of SO. A trend emerged from the preliminary results of a possible influence of sound on sleep spindles' amplitude, with an effect that differed by the sleep spindles' phase divisions. If sleep spindles can be manipulated and possibly enhanced, like SO, their timing can possibly be influenced, providing a new method to improve deteriorating memory processes.

Memory deteriorates with normal and pathological aging, which is evident in those with mild cognitive impairment and Alzheimer's Disease [1,2]. Stimulation techniques might be used to influence memory processes that are decaying during normal aging such as encoding, consolidation, and retrieval [3]. During the encoding of a memory, neurons of brain areas that are relevant for a task are active. Pavlides and Winson have found a similar re-activation of brain areas during sleep to the ones involved in the encoding of the memory [4]. The process of re-activation is thought to be critical in consolidating newly encoded experiences from short- term to long-term storage [5]. Particularly, sleep is important in the consolidation of memories and patterns of neuronal activation are thought to contribute to age-related memory decline [1]. Thus, stimulation of these neuronal activities might be part of the solution to lessen the effects of memory deterioration.

Sleep is divided into two categories: non-rapid eye movement sleep (NREM) and rapid eye-movement sleep (REM). The NREM sleep is predominant in the beginning of the night and can be further subdivided into three stages, while REM represents a single stage, predominate in the latter half of the night [3]. In sleep research an electroencephalogram (EEG) measures electrical brain activity in frequencies, an electromyogram (EMG) measures muscular activity, and an electrooculogram (EOG) measures eye movement throughout the night [6]. Each sleep stage is defined by the outputs of these three devices and is characterized by different electrical rhythms, representing communications between populations of neurons. The first stage of NREM sleep can be described as light sleep. The second and third stages of the NREM sleep are of particular interest to the memory consolidation process [7]. During these stages, electrical activity sleep bursts of 11-16Hz, named sleep spindles, occur. Additionally, slower waves with a frequency of 0.5-1.5 Hz, named slow oscillations (SO) are also present [3]. NREM 2 is characterized by a higher proportion of sleep spindles, whereas during NREM 3 there is a higher SO occurrence [3, 5]. SO and sleep spindles have separate, but related roles in the sleep-dependent memory consolidation. SO originate in the neocortex through the hyperpolarization and depolarization of populations of neurons and they

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are thought to synchronize neuronal activity such as sleep spindles [8]. The process of sleep spindle generation in the reticular nucleus of the thalamus seems to be driven by depolarized neurons being close to their firing threshold and the synchronicity of their firing. Researchers propose that once generated, spindles re-instate newly formed memories, by the modulation of neocortical activity through cortico-thalamic loops [9, 10]. Specifically, these oscillations would modulate the re-activation of neurons initially active during the encoding of the memory. In addition to the individual roles of SO and sleep spindles in memory consolidation, it appears that their interaction is essential as well [11].

Sleep spindles and SO interact by being precisely timed, meaning that they can work in synchrony and the timing between them influences sleep dependent memory consolidation [1, 11, 12]. Mikutta and colleagues found that participants with a higher number of sleep spindles coupled to the SO up-states during sleep (i.e., rising phase of the wave) performed better on the morning recall of a learning task completed the night prior, compared to participants with fewer sleep spindles coupled to the up-states of SO [11]. Therefore, the optimal timing between the two waves for memory consolidation seems to be the coupling of a sleep spindle to the up-state of a SO [10, 13-15]. The optimal coupling phase is also apparent from studies with older participants who displayed a delayed coupling related to decreased retention scores [1]. A possible solution to improve memory consolidation processes in older adults is to influence sleep spindles and SO, and to repair the delayed coupling between them.

An intervention that influences SO and possibly sleep spindles is sound stimulation [13]. Sound stimulation is a common technique used in sleep research as it is a non-invasive tool and auditory information continues to be processed through cortical pathways during sleep [16-18]. Different techniques such as open-loop (OLAS) and closed-loop auditory stimulation (CLAS) can be used to send an auditory stimulus during the occurrence of specific brain waves. The former delivers a sound at a time that is pre-programmed and independent of brain activity whereas the latter sends the sound when a specific brain activity is detected. CLAS targeting SO independent from spindles has been successfully done. Ngo and colleagues [13] found that CLAS sent during the up-state of SO enhanced the SO rhythm, the coupling proportion between spindles and SO, and memory recall, in comparison to a random stimulation condition. Therefore, it appears the up-state phase of SO is a successful target for sound stimulation of memory consolidation. In contrast, sending sounds during sleep spindles occurring in the up-states of SO, using a mix of CLAS and OLAS was unsuccessful in improving memory recall [18]. One explanation is that the sound stimulus was not timed to the endogenous activity of the spindle. The phase of spindles might be of importance to potentially influence their amplitude, such that during SO up-states the neuronal membranes are more depolarized and have a higher probability of firing. A similar property of sleep spindles' neuronal membranes may be expected, as well as a different effect of sound stimulation at various sleep spindle phases.

The aim of this study was to further the understanding of how spindles may be manipulated. Thus, auditory stimulation was presented during sleep spindles, through a mix of OLAS and CLAS. First, the general change in amplitude following spindle stimulation was investigated. A novel investigation in the field was pursued through our second goal, which was to determine whether the effect of sound differed based on the phase of the spindle to which the auditory stimulus was presented. It was hypothesized that the sound stimulus presented to a spindle in a SO up-state would influence the sleep spindles' amplitude. Additionally, if an effect of sound on sleep spindles was present it would differ based on the phase of the spindles during which the

sound was presented.

Method

Participants

Participants were recruited through word-of-mouth. The participants were not eligible if they had: cognitive and hearing impairments; sleep disorders; were pregnant or breastfeeding; had a history of cardiac, neurological, and psychiatric conditions in the past 12 months; or were using any sleep or wakefulness altering drugs, which affect spindle density [19]. Participants received a monetary compensation for their participation of \$150. The research study received approval from Concordia's Ethical Research Board and all participants provided informed consent to take part in the experiment.

Measures

The participants were asked to fill in a sleep diary (adapted from Himmer and colleagues [20]), which measured total time in bed and roughly total time asleep (e.g., When did you go to bed?). Before the experiment, participants were asked to complete the Munich Chrono Type Questionnaire (MCTQ) which confirmed the participants' normal sleep patterns (a full night of sleep with no awakenings) and thus eligibility to the study [21]. An example of a question from the MCTQ is "On workdays I have to get up at [blank] o'clock", the participants were then asked to fill in the blank using short answers. The MCTQ's reliability cannot be determined as it is a scale, however, Di Milia and colleagues [22] have characterized the MCTQ's level of agreement with the Morningness-Eveningness Questionnaire (a questionnaire that assesses chronotype with a high level of reliability of 0.80) as satisfactory.

Apparatus

The Endpoint Connected Hilbert Transformation (ECHT) Box, which takes EEG measurements and outputs sounds, was used [23]. The EEG data was gathered using one recording electrode (forehead placement) and a reference electrode (mastoid bone placement). The sound stimulus (40ms pink noise) was presented binaurally at 55dB SPL through Etymotic ER-3C earphones with insert foam tips [24]. The sound level was decided based on previous similar literature [13,25]. The ECHT box was set to employ a CLAS of SO. The system was programmed to output two types of auditory stimulation: sound for 5 minutes and sham the next 5 minutes. The sound stimulation consisted of detection of sleep spindles and sound output. The sham condition comprised only the detection of sleep spindles for further analysis with no output of sound. The sleep spindles that received sound were compared to those that received sham, with a further analysis of the sleep spindles' phases to which the two conditions were presented.

Procedure

Following recruitment, the participants were briefed about the study. The equipment was later delivered to the participants' home address. Participants gave their written consent and were asked to fill out the sleep diary two days prior to the first experimental night and throughout the nights slept with the equipment. Furthermore, instructions and support for the setup of the apparatus and data collection were provided.

Participants were asked to use the equipment during a total of 5 nights. The duration of the

experiment was determined based on previous research [26]. During one night of sleep, an approximate 20% of sleep spindles were found to be coupled to the SO up-states [26]. Thus, in this study, to maximize the amount of sleep spindles that were stimulated, a 5 nights design was used. In total, 10 days were allocated for the completion of the experiment.

Before going to sleep the participants started a program on the laptop, which was set to present the sound stimulus 30 minutes after start, to allow the participants to fall asleep and enter the NREM 2 [23]. The recording was stopped by the participant in the morning after waking up and the sleep diary was then completed.

Data Collection

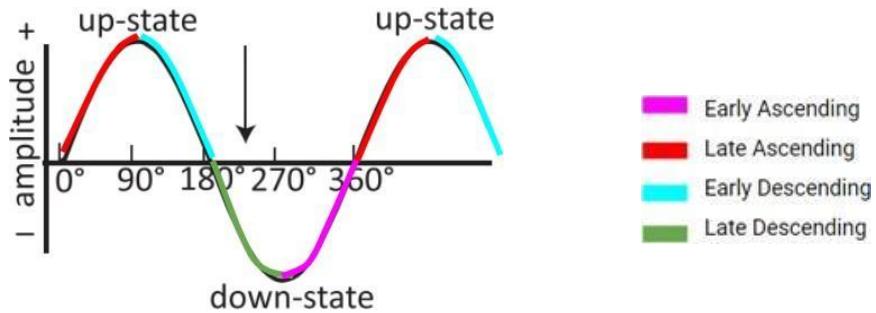
To target sleep spindles nesting in the SO up-states, a mix of OLAS and CLAS was run using a sound (55dB) and a sham (0dB) stimulus. The data acquisition was performed using a sampling rate of 500Hz. An intervention using both OLAS and CLAS was necessary to detect shorter durations than SO (i.e., sleep spindles) as CLAS does not operate fast enough to detect a spindle and send a sound stimulation during it (typically 0.5-3 seconds). The current study design consisted of first targeting the SO-upstates using CLAS by outputting an auditory stimulus fast enough to fall on the up-state of SO. Secondly, based on previous research it was expected that SO would coincide with sleep spindles [26,27]. As a result, only spindles indirectly targeted through stimulation of SO were considered in this study using OLAS.

Data Processing

Once the EEG data was collected, it was analyzed with Python Version 3.8.10 [28] using a custom script that outputted: the time points of auditory and sham stimulation, and raw EEG data. The data were then analyzed using a custom MATLAB script [29]. A filter of 0.1-40Hz was applied to the raw EEG data to filter out low and high frequencies and a customized algorithm of sleep spindle detection was applied [30]. Once the sleep spindles were detected, the oscillations forming the sleep spindle were considered as separate waves and subdivided into four different phase groups based on their electrical activity (named bins): early ascending (bin 1), late ascending (bin 2), early descending (bin 3), and late descending (bin 4). This division of sleep spindles by phases was based on a similar investigation done with SO by Batterink and colleagues [31]. The division of SO is shown in Figure 1 and similar phase categories of sleep spindles are shown in Figure 2 [31]. The EEG data were then fragmented into time windows, (i.e., epochs of 1000ms before the presentation of the sound stimulus and 1000ms after). As a result, each participant had epochs containing sleep spindles that have received sound or sham. A distinction between spindles' phases that have received sound and those that have received sham were made. Spindle amplitude post stimulation was used as a quantifying measure of the effect of sound on sleep spindles.

Figure 1

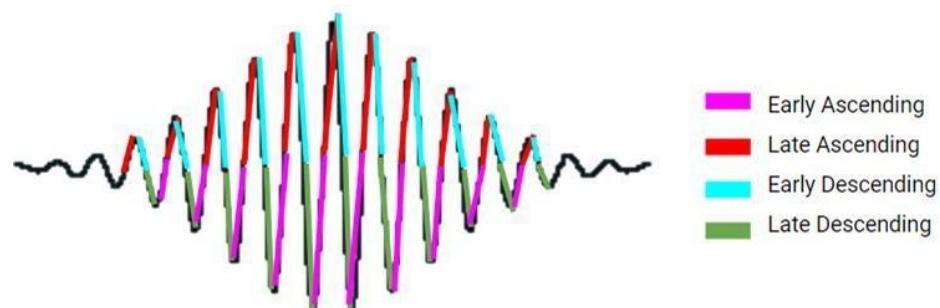
Division of a slow wave by its phases



Note. The figure depicts a SO separated in four parts based on its phases, shown in colour. The legend is provided for comparison with this project's division of sleep spindles. The arrow depicts the optimal phase to which the sound should be presented to participants. Y-axis: Amplitude of the wave; X-axis: Phase of the wave. Figure adapted from Batterink and colleagues (2016; see reference for any other information).

Figure 2

Division of a sleep spindle by its phases



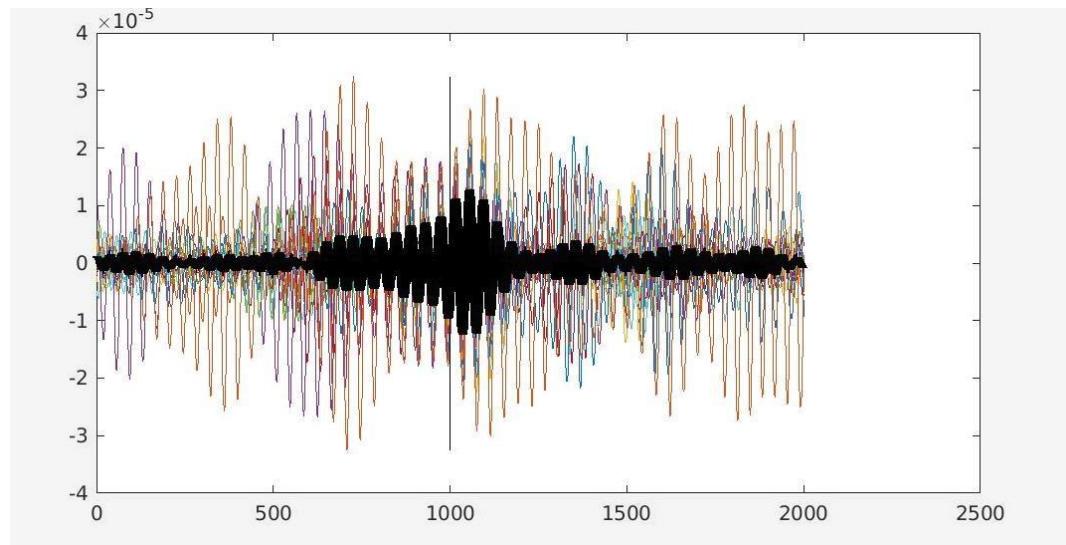
Note. The figure shows the proposed division of a sleep spindle into four phase parts, i.e. bins, which was done during the analysis. The time series for the research and control group, i.e. the phases of the sleep spindles when stimulation occurred and did not occur, will be averaged over time. Each bin is color-coded and assigned a descriptive name.

Data Analysis

The sleep spindles were visualized to determine the appropriate time windows for amplitude comparisons using R2018a MATLAB version [29]. As shown in Figure 3, the average activity of sleep spindles decreases within 1 sec after the presentation of the auditory stimulus. This time interval is adequate as sleep spindles are on average 1000-2000ms and there is approximately a maximum of half of the spindle left following stimulation [32]. The epochs considered in the analysis contain sleep spindles following the time of sound entry.

Figure 3

Average Sleep Spindle Signal in Bin 1 for 1 Night



Note. Visualization of sleep spindles pre and post stimulation from a sample participant and spindle bin. In color: sleep spindles in bin 1 during 1 night of participant 3. In black: average signal of the sleep spindles. X axis: datapoints; Y axis: Voltage (microVolts). Line at 1000 datapoints represents the sound stimulation. For reference 500 datapoints is 1 sec, thus it can be seen that the sleep spindle power decreases around 1 sec after sound stimulation.

Root Mean Square (RMS) values were used to quantify the sleep spindles' amplitude. The power of sleep spindles was considered through RMS values from the moment that the auditory stimulation was presented up to 1 second afterward. RMS values were compared between sound and sham conditions within this time interval. Global RMS averages (of all spindles) were computed in the two conditions to explore a possible effect of sound stimulation on sleep spindles. RMS averages for the four bins, in the sound and sham conditions, were used to look at how the effect might differ based on the phase of the sleep spindle to which the auditory stimulus was presented.

In the present study median values of the sleep spindles in both stimulation conditions were compared within each individual participant. The Mann-Whitney U test was employed and bootstrapped 95% Monte Carlo confidence intervals (CI) based on 10,000 samples were computed. The alpha level was specified at 0.05 and the hypotheses tested in both research questions were two-tailed. The effect size reported was Pearson's r and it was calculated by hand [33]. The effect sizes were interpreted as small at .1, medium at .3, and large at .5 and they were indicative of the proportion of variability between the two conditions accounted for by the sound stimulus [33, 34]. The negative values of the effect size are explained by the sound condition being subtracted from the sham condition in the statistics. Thus, a negative value indicates a higher median in the sound condition. It is important to note that statistical significance and effect sizes must be interpreted with caution considering the small sample size of the study.

Results

Demographics

The original sample of the current study was comprised of 5 individuals, however, following one drop out due to the participant's reported low quality of sleep, the final sample consisted of 4 individuals. The majority of the sample were males (75%) with an average age of 25 ($SD = 1.70$, range = 23-26 years).

Through communications during the experiment and the sleep diary, participants have reported having similar sleep quality and duration during the experiment compared to their usual sleep, with no awakenings caused by the sound stimulation. All the participants reported a normal sleep pattern throughout the experiment and no equipment malfunctions. In this study, participants slept an average of 6.75 hours and displayed a mean of 359.80 sleep spindles coupled to the SO upstates ($SD = 167.80$) for the total duration of the experiment were detected for each participant, which is consistent with a healthy sleep architecture observed in previous studies [26,27]. The results for each participant are presented separately, for both research questions.

Participant 1

The Effect of Sound on Sleep Spindles' Amplitude

A statistically significant difference was found between the sleep spindle amplitude ($Mdn = 4.32e-6$) in the sound stimulation condition and that of the sham stimulation condition ($Mdn = 3.46e-6$; $U = 31029.00$, $p < .001$, $r = -0.15$, 95% CI [0.00; 4.77e-4]). This implies a higher median of sleep spindle amplitude in the sound condition than in the sham one (see Table 1). The negative small effect size is indicative of a small proportion of variability between the two conditions accounted for by the stimuli introduced in the sound condition.

Table 1

Results of Mann Whitney U Test for Stimulation Condition on Sleep Spindle Amplitude

RMS Global	<i>U</i>	<i>p</i>	Pearson's <i>r</i>	95% CI	
				Lower	Upper
Participant 1	31029.00	<.001**	-0.15	0	4.77e-4
Participant 2	15610.00	.27	-0.06	.26	.28
Participant 3	17043.00	.43	-0.04	.42	.44
Participant 4	2364.00	.55	-0.05	.54	.56

Note. RMS Global = all of the RMS values in both stimulation conditions (sound and sham); CI = confidence interval.

* $p < .05$, ** $p < .001$.

The Effect of Sound on Sleep Spindles' Amplitude Based on Phase Divisions

To further explore if the phase of the sleep spindles which the sound was presented exhibited

a different effect on the sleep spindles' amplitude, the phase categories "bins" were compared in the sound and sham condition (see Table 2). The comparison between sound ($Mdn = 4.37e-6$) and sham ($Mdn = 3.14e-6$) stimulations presented to bin 3 resulted in a statistically significant difference between the medians of the 2 groups ($U = 3662.50, p < .001, r = -0.25, 95\% CI [1.82e-4; 0.001]$). It can be concluded that the sleep spindle amplitude in the sound condition was higher than that of the sham condition. The effect size indicates a medium proportion of this difference to be explained by the introduced sound. No significant effects were found of sound on the RMS values of sleep spindles in the other bins. However, small effect sizes were apparent.

Table 2

Mann Whitney U Test for Stimulation Condition on Sleep Spindle Amplitude by Phase Divisions

RMS Divided	Bins	<i>U</i>	<i>p</i>	Pearson's <i>r</i>	95% CI	
				Lower	Upper	
Participant 1	Bin 1	646.00	.42	-0.09	.41	.43
	Bin 2	4458.00	.28	-0.08	.27	.29
	Bin 3	3662.50	<.001**	-0.26	1.82e-4	.001
	Bin 4	543.50	.54	-0.07	.544	.564
Participant 2	Bin 1	172.00	.03*	-0.32	.025	.032
	Bin 2	1556.00	.27	-0.10	.26	.27
	Bin 3	2849.00	.10	-0.13	.09	.11
	Bin 4	96.00	.16	-0.24	.15	.17
Participant 3	Bin 1	498.50	.14	-0.18	.135	.149
	Bin 2	2024.00	.53	-0.06	.515	.534
	Bin 3	2121.50	.38	-0.08	.37	.39
	Bin 4	114.00	.04*	-0.33	.038	.046
Participant 4	Bin 1	34.00	.72	-0.10	.71	.73
	Bin 2	231.00	.36	-0.14	.35	0.37
	Bin 3	236.50	.09	-0.23	.088	.099
	Bin 4	68.50	.73	-0.07	.72	.74

Note. RMS Divided= all of the RMS values in both stimulation conditions (sound and sham) divided by bins; CI= confidence interval.

* $p < .05$, ** $p < .001$.

Participant 2

The Effect of Sound on Sleep Spindles' Amplitude

Overall, no statistically significant difference was found when comparing the amplitude of sleep spindles in the sound condition to the sham one ($U = 15610.00, p < .27, r = -0.06, 95\% \text{ CI } [0.26; 0.28]$). However, a small effect size was present.

The Effect of Sound on Sleep Spindles' Amplitude Based on Phase Divisions

A significant difference and a medium effect size were found in the first bin's sleep spindle amplitude in the sound condition ($Mdn = 1.82e-6$) compared to the sham one ($Mdn = 1.43e-6; U = 172.00, p = .03, r = -0.32, 95\% \text{ CI } [0.025; 0.032]$). No statistically significant difference of medians was found in bin 2, bin 3 or bin 4 (see Table 2). However, small effect sizes were observed.

Participant 3

The Effect of Sound on Sleep Spindles' Amplitude

The difference in the amplitude of sleep spindles between the sound and sham condition was not statistically significant ($U = 17043.00, p < .43, r = -0.04, 95\% \text{ CI } [0.42; 0.44]$). A small effect size was present, suggesting that there is virtually no variability between conditions in this analysis accounted by the stimuli introduced in the sound condition.

The Effect of Sound on Sleep Spindles' Amplitude Based on Phase Divisions

In contrast, the sound stimulation condition ($Mdn = 1.46e-6$) exhibited a statistically significant effect in bin 4 ($U = 114.00, p = .04, r = -0.33, 95\% \text{ CI } [0.038; 0.046]$) compared to the sham condition ($Mdn = 1.16e-6$). As well, a medium effect size is suggestive of a medium proportion of the variance between the sleep spindles amplitude in conditions being accounted for by the introduced stimuli. No statistically significant difference was found between the two conditions for bin 1, bin 2, and bin 3 (see Table 2). This was further supported by small effect sizes.

Participant 4

The Effect of Sound on Sleep Spindles' Amplitude

No statistically significant difference and a small effect size were found between the sleep spindle amplitude in the sound and sham conditions ($U = 2364.00, p < .55, r = -0.05, 95\% \text{ CI } [0.54; 0.56]$).

The Effect of Sound on Sleep Spindles' Amplitude Based on Phase Divisions

A similar pattern of results emerged in the investigation of differences for the bin divisions. None of the bins had a statistically significant difference between the stimulation conditions (see Table 2). Bin 1 ($r = -0.10$), bin 2 ($r = -0.14$), bin 3 ($r = -0.23$) and bin 4 ($r = -0.07$) have exhibited small effect sizes. Mixed results can be concluded from the data of participant 4.

Discussion

The goal of this study was to investigate the possibility of stimulating sleep spindles' amplitude with sound, specifically, if spindles differ in their sensitivity to sound by phase. The preliminary results suggest a trend of sound increasing sleep spindles' amplitude following stimulation, however this was not consistent across participants. Mixed results were found regarding specific

phases being more sensitive to sound stimulation than others with bin 1, bin 3 and bin 4 of interest. These findings agree with previous research, which has shown an influence of sound on sleep spindles [16-18]. The hypothesis that sound will affect the amplitude of sleep spindles coupled to the SO up-states was supported only by data collected from participant 1. That is, the sound stimuli increased sleep spindles' amplitude immediately after stimulation. A successful stimulation of sleep spindles may render influencing the timing between SO and spindles possible. If the timing between the two waves can be influenced, the memory processes that are related to the coupling might be affected as well, which is of relevance in older adults. However, a similar study to the present one did not find an effect on sleep spindles via sound [18]. One limitation that is shared by Ngo and colleagues' and the current study was considering the whole spindles and not taking the sleep spindles' phases into account (relating to the endogenous activity of the waves). Thus, it is possible that the effect of sound on individual bins could not be resolved when the data was considered as a whole, because the RMS values might have averaged out in the analysis. This limitation is addressed in the second analysis of this study by tackling the effect of sound on sleep spindles by phase divisions, which is a novel investigation in the field.

The increase of spindle amplitude following stimulation was found to be dependent upon the phase of the spindle to which sound was presented to. The female participant (i.e., participant #2) showed a significant stimulation of the ascending phase (bin 1) of the sleep spindle sensitive to sound, compared to the male counterparts that showed sensitivity in the descending phases of the spindles (bin 3 and bin 4). Overall, hormonal differences between sexes affect sleep, such as total amount of sleep, specific stage duration, and oscillatory activity in frequency bands, more specifically females have shown higher spindle density, duration and amplitude compared to males [35-37]. Thus, sex differences might affect the activity or properties of sleep spindles, which in turn could explain a different phase sensitivity to sound. Nevertheless, more studies on the phase-dependent intrinsic properties of sleep spindles need to be conducted to tie the above results to the differences in sleep spindle activity between sexes. Overall, spindles seem to be influenced by sound stimuli.

Furthermore, during sleep, spindles are thought to protect the individual from stimuli, including sounds that may disrupt their sleep [38]. This property might explain the increase of amplitude of the sleep spindles following presentation of the sound stimulus. Thus, the results of this study are mostly consistent with previous work that has found an increase in the number of spindles during sleep and spindle density, defined as number of spindles per minute, following sound stimulation [16,17]. Particularly, the sensitivity of the spindle bins to sound found in this study followed the SO stimulation literature. The up-states of SO are an effective target for sound stimulation; thus, the same train of thought might be assumed for sleep spindles, due to the close interaction between the two waves [13-15].

This study was affected by limitations stemming from the spindle detection algorithm, sample size and tools used. First, faulty detections of sleep spindles could have been included by the automatic sleep spindle detection algorithm used, however, this limitation can only be addressed by improving the algorithms currently available to researchers [30]. Second, the small sample size of the study calls for caution in the interpretation of the findings. This study provides preliminary results as methodological refinement, not generalizability, was the primary goal of this study.

It is important to note that this data set was collected from a young adult sample, thus no generalization claims toward older individuals can be made. As well, measures of memory con-

solidation were not used. That is, the goal accomplished by this experiment was to determine if an effect on sleep spindles using sound stimulation is possible. Future studies can use these findings as basis to investigate the potential influences of sound on memory consolidation achieved through stimulation of sleep spindles and SO in younger and older samples. More specifically, future studies could focus on other variables of sleep spindles that might be affected by sound rather than amplitude, such as spindle density (defined as the number of spindles per minute). Further, for the purposes of this project the EEG signal used was only filtered to the spindle frequency from the signal collected. An effect of sound might not be limited to solely the spindle activity band post stimulation, but other bands as well. Similarly, when sound stimulation was presented during SO, the effects of the stimulation manifested not only in the SO activity band, but the faster spindle band as well [13-15]. Lastly, it's important to note that this study only used one frontally-placed electrode to determine SO and sleep spindles. EEG signals may appear differently based on electrode placements and should be considered when comparing results.

In summary, the results suggest a potential influence of sound stimulation on sleep spindles. This effect seems to be directional toward an increase of sleep spindles' amplitude following sound stimulation. Additionally, targeting specific phases of sleep spindles seems to be of importance, because some participants showed phase-dependent specificity to sound. If spindles are indeed sensitive to sound stimulation by phase, this information can be used to successfully target their oscillations and possibly affect memory consolidation processes. This study supplements the current understanding of how sleep spindles might be manipulated using sound stimulation and brings a novel interest of investigation by phase of spindles [16-18]. Subsequent research should investigate whether presenting sounds can stimulate sleep spindles separately from SO. These findings would suggest that these electrical signals could be rhythmically influenced and potentially be used as a treatment intervention for cognitive impairments in older adults [1, 12]. Once more information on the manipulation of sleep spindles is available, this may lead to the opportunity to explore the re-alignment of SO and sleep spindles that is lost with age and is crucial to the memory consolidation process.

References

- [1] Helfrich, R. F., Mander, B. A., Jagust, W. J., Knight, R. T., & Walker, M. P. (2018). Old Brains come uncoupled in sleep: Slow wave-spindle synchrony, brain atrophy, and forgetting. *Neuron*, 97, 221–230. <https://doi.org/10.1016/j.neuron.2017.11.020>
- [2] Liu, S., Pan, J., Tang, K., Lei, Q., He, L., Meng, Y., Cai, X., & Li, Z. (2019). Sleep spindles, K-complexes, limb movements and sleep stage proportions may be biomarkers for amnestic mild cognitive impairment and Alzheimer's disease. *Sleep and Breathing*, 24, 637–651. <https://doi.org/10.1007/s11325-019-01970-9>
- [3] Diekelmann, S., & Born, J. (2010). The memory function of sleep. *Nature Reviews Neuroscience*, 11, 114–126. <https://doi.org/10.1038/nrn2762>
- [4] Pavlides, C., & Winson, J. (1989). Influences of hippocampal place cell firing in the awake state on the activity of these cells during subsequent sleep episodes. *The Journal of Neuroscience*, 9, 2907–2918. <https://doi.org/10.1523/JNEUROSCI.09-08-02907.1989>

- [5] Stickgold, R. (2005). Sleep-dependent memory consolidation. *Nature*, 437, 1272–1278. <https://doi.org/10.1038/nature04286>
- [6] Reilly, R. B., & Lee, T. C. (2010). Electrograms (ECG, EEG, EMG, EOG). In T.C. Lee & P.F. Niederer (Eds.), *Basic Engineering for Medics and Biologists: An ESEM Primer* (pp.90-108). IOS Press. <https://doi.org/10.3233/978-1-60750-527-3-90>
- [7] Rasch, B., & Born, J. (2013). About sleep's role in memory. *Physiological reviews*, 93, 681-766. <https://doi.org/10.1152/physrev.00032.2012>
- [8] Mölle, M., Marshall, L., Gais, S., & Born, J. (2002). Grouping of spindle activity during slow oscillations in human non-rapid eye movement sleep. *Journal of Neuroscience*, 22, 10941–10947. <https://doi.org/10.1523/jneurosci.22-24-10941.2002>
- [9] Antony, J. W., Schönauer, M., Staresina, B. P., & Cairney, S. A. (2019). Sleep Spindles and Memory Reprocessing. *Trends in Neurosciences*, 42, 1–3. <https://doi.org/10.1016/j.tins.2018.09.012>
- [10] Staresina, B. P., Bergmann, T. O., Bonnefond, M., Van Der Meij, R., Jensen, O., Deuker, L., Elger, C. E., Axmacher, N., & Fell, J. (2015). Hierarchical nesting of slow oscillations, spindles and ripples in the human hippocampus during sleep. *Nature Neuroscience*, 18, 1679–1686. <https://doi.org/10.1038/nn.4119>
- [11] Mikutta, C., Feige, B., Maier, J. G., Hertenstein, E., Holz, J., Riemann, D., & Nissen, C. (2019). Phase-amplitude coupling of sleep slow oscillatory and spindle activity correlates with overnight memory consolidation. *Journal of Sleep Research*, 28, 1–6. <https://doi.org/10.1111/jsr.12835>
- [12] Muehlroth, B. E., Sander, M. C., Fandakova, Y., Grandy, T. H., Rasch, B., Shing, Y. L., & Werkle-Bergner, M. (2019). Precise Slow Oscillation–Spindle Coupling Promotes Memory Consolidation in Younger and Older Adults. *Scientific Reports*, 9, 1–15. <https://doi.org/10.1038/s41598-018-36557-z>
- [13] Ngo, H. V. V., Martinetz, T., Born, J., & Mölle, M. (2013). Auditory closed-loop stimulation of the sleep slow oscillation enhances memory. *Neuron*, 78, 545–553. <https://doi.org/10.1016/j.neuron.2013.03.006>
- [14] Ong, J. L., Lo, J. C., Chee, N. I., Santostasi, G., Paller, K. A., Zee, P. C., & Chee, M. W. (2016). Effects of phase-locked acoustic stimulation during a nap on EEG spectra and declarative memory consolidation. *Sleep Medicine*, 20, 88–97. <https://doi.org/10.1016/j.sleep.2015.10.016>
- [15] Papalambros, N. A., Santostasi, G., Malkani, R. G., Braun, R., Weintraub, S., Paller, K. A., & Zee, P. C. (2017). Acoustic enhancement of sleep slow oscillations and concomitant memory improvement in older adults. *Frontiers in Human Neuroscience*, 11, 1–14. <https://doi.org/10.3389/fnhum.2017.00109>
- [16] Antony, J. W., & Paller, K. A. (2016). Using oscillating sounds to manipulate sleep spindles. *Sleep*, 40, 1–8. <https://doi.org/10.1093/sleep/zsw068>
- [17] Lustenberger, C., Patel, Y. A., Alagapan, S., Page, J. M., Price, B., Boyle, M. R., & Fröhlich, F. (2018). High-density EEG characterization of brain responses to auditory rhythmic stimuli during wakefulness and NREM sleep. *Neuroimage*, 169, 57–68. <https://doi.org/10.1016/j.neuroimage>.

2017.12.007

- [18] Ngo, H. V. V., Seibold, M., Boche, D. C., Mölle, M., & Born, J. (2019). Insights on auditory closed-loop stimulation targeting sleep spindles in slow oscillation up-states. *Journal of Neuroscience Methods*, 316, 117–124. <https://doi.org/10.1016/j.jneumeth.2018.09.006>
- [19] Plante, D. T., Goldstein, M. R., Cook, J. D., Smith, R., Riedner, B. A., Rumble, M. E., Jelenchick, L., Roth, A., Tononi, G., Benca, R. M., & Peterson, M. J. (2015). Effects of oral temazepam on sleep spindles during non-rapid eye movement sleep: A high-density EEG investigation. *European Neuropsychopharmacology*, 25, 1600–1610. <https://doi.org/10.1016/j.euroneuro.2015.06.005>
- [20] Himmer, L., Muller, E., Gais, S., & Schonauer, M. (2017). Sleep-mediated memory consolidation depends on the level of integration at encoding. *Neurobiology of Learning and Memory*, 137, 101-106. <https://doi.org/10.1016/j.nlm.2016.11.019>
- [21] Roenneberg, T., Wirz-Justice, A., & Merrow, M. (2003). Life between clocks: daily temporal patterns of human chronotypes. *Journal of biological rhythms*, 18, 80-90. <https://doi.org/10.1177/0748730402239679>
- [22] Di Milia, L., Adan, A., Natale, V., & Randler, C. (2013). Reviewing the psychometric properties of contemporary circadian typology measures. *Chronobiology International*, 30, 1261-1271. <https://doi.org/10.3109/07420528.2013.817415>
- [23] Elemind Technologies, Inc. (2018). Endpoint Connected Hilbert Transformation Box; adapted from the Princeton Computational Memory Lab.
- [24] Etymotic Research (2016). ER3C Insert Earphones.
- [25] Ngo, H. V. V., Claussen, J. C., Born, J., & Mölle, M. (2013). Induction of slow oscillations by rhythmic acoustic stimulation. *Journal of Sleep Research*, 22, 22–31. <https://doi.org/10.1111/j.1365-2869.2012.01039.x>
- [26] Staresina, B. P., Bergmann, T. O., Bonnefond, M., Van Der Meij, R., Jensen, O., Deuker, L., Elger, C. E., Axmacher, N., & Fell, J. (2015). Hierarchical nesting of slow oscillations, spindles and ripples in the human hippocampus during sleep. *Nature Neuroscience*, 18, 1679–1686. <https://doi.org/10.1038/nn.4119>
- [27] Marzano, C., Moroni, F., Gorgoni, M., Nobili, L., Ferrara, M., & De Gennaro, L. (2013). How we fall asleep: regional and temporal differences in electroencephalographic synchronization at sleep onset. *Sleep medicine*, 14, 1112-1122. <http://dx.doi.org/10.1016/j.sleep.2013.05.021>
- [28] Van Rossum, G., & Drake, F. L. (2009). Python 3 Reference Manual. Scotts Valley, CA: CreateSpace. Version 3.8.10.
- [29] MATLAB. (2018). 9.7.0.1190202 (R2018a). Natick, Massachusetts: The MathWorks Inc.
- [30] Lacourse, K., Delfrate, J., Beaudry, J., Peppard, P., & C. Warby, S. (2019). A sleep spindle detection algorithm that emulates human expert spindle scoring. *Journal of Neuroscience Methods*, 316, 3–11. <https://doi.org/10.1016/j.jneumeth.2018.08.014.A>

- [31] Batterink, L. J., Creery, J. D., & Paller, K. A. (2016). Phase of spontaneous slow oscillations during sleep influences memory-related processing of auditory cues. *Journal of Neuroscience*, 36, 1401–1409. <https://doi.org/10.1523/JNEUROSCI.3175-15.2016>
- [32] De Gennaro, L., & Ferrara, M. (2003). Sleep spindles: an overview. *Sleep medicine reviews*, 7, 423-440. [https://doi.org/10.1016/S1087-0792\(02\)00116-8](https://doi.org/10.1016/S1087-0792(02)00116-8)
- [33] Fritz, C. O., Morris, P. E., & Richler, J. J. (2012). Effect size estimates: Current use, calculations, and interpretation. *Journal of Experimental Psychology: General*, 141, 2–18. <https://doi.org/10.1037/a0024338>
- [34] Cohen J. (1988). Statistical Power Analysis for the Behavioral Sciences. Lawrence Erlbaum Associates, Publishers.
- [35] Bixler, E. O., Papaliaga, M. N., Vgontzas, A. N., Lin, H. M., Pejovic, S., Karataraki, M., Vela-Bueno, A. & Chrousos, G. P. (2009). Women sleep objectively better than men and the sleep of young women is more resilient to external stressors: effects of age and menopause. *Journal of sleep research*, 18, 221-228. <https://doi.org/10.1111/j.1365-2869.2008.00713.x>
- [36] Mong, J. A., & Cusmano, D. M. (2016). Sex differences in sleep: impact of biological sex and sex steroids. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 371, 20150110. <https://doi.org/10.1098/rstb.2015.0110>
- [37] Fernandez, L. M. J., & Luthi, A. (2020). Sleep spindles: mechanisms and functions. *Physiological Reviews*, 100, 805-868. <https://doi.org/10.1152/physrev.00042.2018>
- [38] Dang-Vu, T. T., McKinney, S. M., Buxton, O. M., Solet, J. M., & Ellenbogen, J. M. (2010) Spontaneous brain rhythms predict sleep stability in the face of noise. *Current Biology*, 20, R626–R627. <https://doi.org/10.1016/j.cub.2010.06.032>

“What if” asked in the shadow of trauma: An analysis of counterfactual thinking in the development and maintenance of Post-traumatic Stress Disorder *

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The present review examines the psychological phenomenon of counterfactual thought (CFT) as a potential mitigating factor in the development and maintenance of Post-Traumatic Stress Disorder (PTSD). CFT is the human proclivity to look back on past events and imagine how things could have been different. The adaptive nature of CFT is dependent on the controllability of the event and the modality of CFT used to reimagine alternatives. The preparative and affective functions that upward and downward CFT provide were not found to retain their adaptive value following a traumatic event. Rather, due to variables beyond an event's controllability, such as counterfactual vividness and frequency, CFT was found to be more harmful than memories of the trauma themselves. However, CFT is not invariably a detriment following trauma. Benefit finding and prescriptions of fate were found to be causal links between CFT and finding meaning in life. Discerning meaning from life is an adaptive state necessary for sustaining well being. As it pertains to trauma, Frankl hypothesized meaning as essential for coping with tragedy. Based on the findings of the review, it is proposed that CFT should be targeted directly during clinical interventions in order to retain the healthy qualities of the cognition. Future research should seek to identify more causal links between CFT and personal meaning following adverse events, in order to tailor treatment to individual differences in CFT and the idiographic manner in which trauma occurs, is experienced, and is subsequently coped with.

Counterfactual thinking (CFT) is a cognitively based psychological phenomenon defined as a reimagining of alternatives to past outcomes. Parsed into two modalities, upward CFT can be understood as the reconsideration of circumstances of a past event which may have led to a better outcome [1]. When a marriage fails, for example, an individual may look back on what they could have done differently to avoid the divorce. Downward CFT is an imaginative act on how an outcome could have been worse. A college student maimed by a drunk driver may engage in downward CFT by thinking to themselves “I could be in a wheel chair.” Upward CFT can calibrate future behavior when reassessing an event under personal control. The individual experiencing marital difficulties, by considering what they could have done differently, may make personal changes in order to salvage the relationship. The preparative function of upwards CFT corresponds with Folkman’s [2] problem-focused coping strategy. A problem-focused coping strategy seeks to reduce stress by actively dealing with the situation causing it. However, if the event is outside of personal control, such as being struck by a drunk driver, upward CFT can be maladaptive.

Extensive emotional harm can be done through the upward re-examination of an event for which one’s behavior had no influence over the outcome or the imagined alternatives [1]. Con-

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versely, downward CFT is adaptive in such a situation. Through imagining a worse outcome, such as a graver injury or death, the student hit by the drunk driver may experience emotional relief in spite of what happened. Engagement in downward CFT serves as an affective function, aligning with Folkman's [2] emotion-focused coping strategy, which deals with stress through the management of adverse emotions caused by the situation. If the circumstances of controllability were inverted, however, downward CFT may deter the adoption of a problem-focused coping strategy [2]. CFT and the adaptive implication of each direction are dependant on the nature of the event being reimagined. This begs the question, what are the psychological implications of CFT when the event is both outside of personal control and a more adverse outcome is unimaginable?

Post-Traumatic Stress Disorder (PTSD) is a psychiatric disorder that develops within those who have experienced a traumatic event which threatened their notion of physical safety. Combat and sexual violence are events which commonly lead to a diagnosis of PTSD. The symptomology of PTSD is marked by unwanted dreams, intrusive memories, and phobic avoidance related to the traumatic event well after the event has ended. The etiological phases of risk for PTSD development are organized temporally around the event in pre-, during-, and post-trauma phases [3]. The pre-trauma phase concerns itself with how individual differences such as trait neuroticism or a familial history of mental illness can act as potential risk factors [3]. The during-trauma phase focuses on a traumatic event and the way it is cognitively processed [3]. What follows is the post-trauma phase, which examines how poor coping strategies and lack of social support following an event can influence the development of PTSD [3]. Recently, research has shifted from the during-trauma phase to phases which precede and follow the event [3]. The shift is representative of a desire for a better understanding as to how individual differences in psychological functioning may interact before and after the traumatic event in the development and maintenance of PTSD. Due to the retrospective nature of counterfactual thought, the present review will focus on the post-trauma phase. The review will examine CFT under the conditions of a traumatic event, and how the adaptive pathways of upward and downward CFT may mitigate or aggravate the traumatic event in the minds of those effected.

In three experiments conducted by Roese [1], both the preparative and affective function of CFT were empirically validated. The research provides a structural understanding of both modalities of CFT and how they function as coping strategies. However, outside of its controllability, little attention is paid to the nature of the event itself or other characteristics which may influence CFT and the adaptive functions of each its directions. With regards to events that are unrectifiable, Burgess and colleagues, [4] and Wood and colleagues [5] conducted studies in which downward CFT served as affective protection for victims of sexual violence and cancer patients respectively. Contrary to these findings however, in a sample of terrorist attack survivors, the affective and preparative functions of downward and upward CFT failed to mitigate the development of PTSD [6]. Rather, emotion-focused coping strategies associated with downward CFT correlated with the development of PTSD [6]. The failure of Roese's [1] findings to replicate in the sample of terrorist attack victims suggests that other variables may hold influence over the adaptive outcomes of counterfactual thought. Counterfactual vividness, which is how clearly the event and its alternatives are mentally generated, was found to be maladaptive in both directions of CFT [6]. Proximity to an alternative outcome, such as downward CFT to death or upward CFT to how an injury may have been preventable, during an already traumatic event, was also found to negate the adaptive qualities of CFT [6]. Additionally, higher frequency of CFT, as well as fluidity, the capability to switch back and forth from each modality, were also found to be maladaptive [6]. These characteristics, when added to an already uncontrollable and traumatic event, not only compound to

dismantle the adaptive value of both directions of CFT, but also serve to make CFT more difficult to cope with than the intrusive memories of the event itself [6]. However, just as how the adaptive coping mechanism of CFT is not assured, engagement in CFT post trauma does not guarantee the development of PTSD. Rather, many subjects (45%) who self-reported engaging in CFT following a terrorist attack, an event that is uncontrollable in which downward counterfactuals are difficult to generate, developed the disorder [6]. The portion of those that did not develop PTSD suggest that CFT is not invariably maladaptive post trauma and that individual differences in CFT may mitigate how effectively an individual copes with a traumatic event.

Interestingly, Kray and colleagues [7] conducted a study which found that CFT was correlated to finding meaning in life. Meaning in life can be understood as the human need to create a personal narrative in order to cope with the inherent absurdity of day-to-day existence [8]. Kray and colleagues [7] hypothesized that considering upward and downward alternatives to crucial moments in life generated greater meaning from them than directly examining the events. What causally links CFT to meaning in life are what Kray and colleagues [7] identify as prescriptions of fate and recognition of positive consequences. Fate prescriptions are defined as appraising events as pre-ordained, which in itself is a form of CFT [7]. Following an adverse event, a person may say “it was meant to be” which aids in the construction of a personal narrative [7]. Benefit finding, similar to Folkman’s [2] positive reappraisal, can be understood as ascribing positive aspects to events despite the adverse repercussions. Being that finding meaning is central across a life span, and is causally linked to counterfactual thought, how may it aid in a life shattered by tragedy? [8] Victor Frankl, an Austrian psychiatrist who survived the Auschwitz concentration camp, argued that discerning meaning from tragedies is a necessity in order to psychologically survive them [9]. In Frankl’s [9] view, those who succumbed to the trauma of Auschwitz, were unable to create meaning for their lives in spite of their suffering.

The findings on CFT offer a better understanding of how PTSD develops and is maintained. The affective and preparative functions of upward and downward CFT are not always adaptive when an event is perceived as both uncontrollable and traumatic. The inability of Roese’s [1] findings to replicate show that CFT is one of the many cognitions post-traumatic stress can distort. Vividness, frequency, and proximity to downward and upward alternatives can amalgamate to not only deconstruct the adaptive qualities of the cognition, but make the alternatives more harmful than the memories of the event itself. The clinical implications of these findings suggest that CFT should be targeted directly during therapeutic interventions, in order for the cognition to retain its adaptive function [7]. Kray and colleagues [7] findings provide a foundation for how clinicians can approach CFT distorted by trauma. A clinician can provide guidance when prescribing fate and finding benefit, when discerning meaning from trauma and life following a PTSD-inducing event proves to be too difficult of task to be undertaken alone.

The limitations of both Kray and colleagues [7] and Roese’s [1] studies are that they were conducted on a sample of undergraduates in which the event was simulated in an experiment. It would be ethically unsound to replicate an adverse event which could be construed as traumatic. Due to these limitations, the boundaries of the adaptive functions of CFT were overlooked in Roese’s [1] research, and possibly the causal links between CFT and the adaptive state of having meaning in life in Kray and colleagues [7] study. Fate prescriptions, for example, would be difficult to employ in a secular population. Moreover, prescribing the cause of a traumatic event to fate could cause a deeply religious person to displace blame to God causing further degeneration of adaptive coping and an already threatened notion of physical safety. However, the weaknesses

of these studies pose a possible direction for future research.

The present review has demonstrated that there is more nuance to how CFT interacts with traumatic events beyond its controllability via the upward and downward modalities of CFT. Researchers should seek to find more causal links between CFT and the adaptive state of meaning post trauma in order to simultaneously expand and enhance models of interventions targeting maladaptive patterns of CFT. Though the Covid-19 virus has and continues to pose a great challenge to society, the pandemic will also offer a unique opportunity for future research into anxiety disorders. This potentially life-threatening pathogen could potentially act as a traumatic event which precipitates the development of PTSD. Studies can be designed around samples of participants with a diagnosis of PTSD stemming from the Covid-19 pandemic. With this form of traumatic event, greater focus can be paid to the post trauma phases in which counterfactual thinking occurs. However, the pandemic has yet to enter a post phase, both stalling, and making, future research into CFT and PTSD ever more vital upon the pandemic's end. A more comprehensive understanding is needed of how CFT, as an individual difference, interacts with the various dimensions of traumatic events, and at what point exactly do the cognition's adaptive functions distort in the minds of those who have suffered trauma. With a comprehensive understanding of the interaction of CFT and trauma, researchers and clinicians can help alleviate the pain of constantly reimagining maladaptive alternatives to devastation, and re-instill healthy coping so those afflicted with trauma can go on to lead full and healthy lives.

References

- [1] Roesel, N. J. (1994). The functional basis of counterfactual thinking. *Journal of Personality and Social Psychology, 66*, 805-818.
- [2] Folkman, S. (1984). Personal control and stress and coping processes: A theoretical analysis. *Journal of Personality and Social Psychology, 46*, 839-852.
- [3] North, C. S., Suris, A. M., Davis, M., & Smith, R. P. (2009). Toward validation of the diagnosis of posttraumatic stress disorder. *American Journal of Psychiatry, 166*(1), 34-41.
- [4] Burgess, A. W., & Holmstrom, L. (1979). *Rape: Crisis and recovery*. Bowie, MD: Brady.
- [5] Wood, J. V., Taylor, S. E., & Lichtman, R. R. (1985). Social comparison in adjustment to breast cancer. *Journal of Personality and Social Psychology, 49*, 1169-1183.
- [6] Gilbar, O., Plivazky, N., & Gil, S. (2010). Counterfactual thinking, coping strategies, and coping resources as predictors of PTSD diagnosed in physically injured victims of terror attacks. *Journal of Loss and Trauma, 15*(4), 304-324.
- [7] Kray, L. J., George, L. G., Liljenquist, K. A., Galinsky, A. D., Tetlock, P. E., & Roesel, N. J. (2010). From what might have been to what must have been: Counterfactual thinking creates meaning. *Journal of personality and social psychology, 98*(1), 106.
- [8] Becker, E. (1973). *The Denial of Death*. New York etc. The Free Press
- [9] Frankl, V. E. (1985). *Man's search for meaning*. Simon and Schuster.

Parenting Behaviours in MDD and BD and Offspring Psychopathology *

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The offspring of parents with major depressive disorder (OMDD) and bipolar disorder (OBD) are at high risk for developing mental disorders. In addition to genetic factors, environmental risk, such as deficits in parenting, is purported to be associated with these negative outcomes. Despite this, a comparison between major depressive disorder (MDD) and bipolar disorder (BD) in terms of parenting behaviours and offspring psychopathology has not yet been conducted. Therefore, the aim of this review article is to inform further research by exploring parenting behaviours of parents with MDD and BD and offspring psychopathology. Parents with MDD and BD share several negative parenting behaviours including low levels of support or care, negative affect, low levels of structure and control. Mania, a symptom of BD, is accompanied by self-centeredness, poor judgment, high levels of impulsivity, and overinvolvement. Although we see some differences in the parenting behaviours between parents with MDD and BD, no differences have been found in family functioning and environment between the two disorders. Further, multiple specific parenting behaviours have been shown to mediate the relationship between parental MDD and BD and offspring psychopathology. Several areas of further research are discussed including the development of common conceptual framework and direct comparisons between MDD and BD.

Major depressive disorder (MDD) is one of the most common psychiatric illnesses causing great personal and economic burden for individuals, families, and society [1,2]. It is estimated that 10% to 15% of the general population will experience clinical depression during their lifetime [3]. Major depressive disorder (MDD) is a debilitating disease that is characterized by depressed mood, diminished interests, impaired cognitive function, and vegetative symptoms, such as disturbed sleep or appetite. MDD is more common in women than in men [4]. For instance, the lifetime prevalence of MDD among women is almost twice that of men [5]. This is an important consideration in the effects of MDD disorders on mothers' ability to parent as they are often the only or the primary parental figure. Further MDD is particularly more prevalent among both men and women who were divorced, separated, or widowed, as compared to those who were single [6] further complicating parental roles.

Bipolar disorder (BD), while much less common than MDD, affects about 45 million people worldwide [7]. BD is a chronic and debilitating mental illness characterized by extreme fluctuations in mood, from manic highs to depressive lows [8]. BD is accompanied by maladaptive personality traits, most predominantly neuroticism. BD is also accompanied by impaired psychosocial functioning such as adopting ineffective coping styles, specifically emotion-focused coping, a coping strategy in which individuals focus on reducing negative emotions as opposed to resolving the actual problem or stressor at hand [9,10,11]. Adults with BD also experience high levels of negative life events [12, 13] and tend to have ineffective coping strategies in dealing with stressful situations [10, 11]. These deficits in personality and psychosocial functioning related to BD can negatively affect parenting skills and abilities [9].

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Genetic factors play important roles in the development of MDD and BD, as indicated by family, twin, and adoption studies. Therefore, the offspring of parents with BD and MDD are at a higher risk for developing mental health disorders. Twin studies suggest a heritability of 40% to 50% for MDD, and family studies indicate a twofold to threefold increase in lifetime risk of developing MDD among first-degree relatives [14]. The offspring of depressed parents are three to four times more likely to develop a depressive disorder than children of non-depressed parents [15]. Estimated heritability rates of BD are even greater than for MDD at 79 – 93% [16]. Cross-sectional and longitudinal studies of the offspring of parents with bipolar disorder (OBD) report up to 70% greater risk for any psychiatric disorder compared to offspring of healthy parents. Specifically, these studies demonstrated an elevation of mood disorders, anxiety disorders, neurodevelopmental disorders, and substance use [17, 18, 19, 20].

In addition to genetic factors, environmental risk, such as deficits in parenting, have been described as a dominant influence on the development of psychopathology in the offspring of parents with MDD (OMDD) and the offspring of parents with BD (OBD) [21, 22, 23]. Parenting style and parent-child interactions are typically negatively affected by MDD and BD [24, 25]. Positive parenting practices consist of parental warmth, support, positive affect, sensitivity and is associated with greater levels of self-regulation in children [26]. Contrarily, negative parenting comprises of hostility, neglect, excessive intrusiveness, and over-control [27]. Negative parenting can create a dysfunctional caregiving environment, which is a well-established risk factor for a broad array of mental disorders for the OBD and the OMDD across the lifespan [28, 29, 25].

Despite the importance of both conditions in the population and the evident role that parenting deficits in both disorders play in the development of offspring psychopathology, a comparison between MDD and BD in terms of parenting behaviours and offspring psychopathology has not yet been conducted. Therefore, to inform further research, this review article has the following aims: First, to shed light on the parental behaviours shared by both BD and MDD and the parental behaviours unique to each. Second, an examination of the psychopathology of the offspring of parents with BD and MDD. Lastly, an exploration of specific parental behaviours that mediate the relationship between parental BD or MDD and offspring psychopathology.

Similarities in Parenting Behaviours

Parents with BD and parents with MDD share several negative parenting behaviours including low levels of support or care, negative affect, low levels of structure and control. Low levels of support or care have been found in both parents with MDD and BD. For instance, Iacono and colleagues (2018) found that parents with BD provided less support to their offspring [21]. Specifically, the parents showed low levels of parental warmth or affection, nurturance, and emotional expressiveness as measured by the Parenting Dimensions Inventory [30]. Similarly, BD parents reported significantly lower parental care characterized by warmth and affection scores compared to healthy controls [31]. In terms of MDD, less supportive parental behaviours compared to healthy control have also been well documented. Cummings and colleagues (2005) found that parents with MDD showed less support, specifically, lower levels of warmth, nurturance, and greater levels of disengagement or un-involvement [32]. In addition, in a study on the effects of depression on parenting conducted by Wilson and Durbin (2010) [33], a decrease in warmth, sensitivity, responsiveness, and an increase in disengagement was found among fathers and mothers with depression. While it seems that both parents with BD and MDD show lower levels of engagement compared to healthy controls, it has been found that bipolar mothers tend to be significantly less

engaged in interaction than mothers with depression and well mothers [34].

A cardinal symptom shared by both BD and MDD is negative affect such as psychological distress, anxiety, depressed mood, hostility, and other unpleasant emotions [35, 36]. Therefore, as expected, negative affect towards children is common among parents with MDD and BD [37, 33]. For instance, parents with BD show high levels of criticism and hostility towards their children [38]. Criticism has also been reported in depressed parents [37]. Depression is significantly associated with more hostile parenting [39]. Parents with MDD and BD both show more irritability than controls [32, 40]. While comparing maternal critical or irritable behaviours between the two disorders, no significant difference has been found between mothers with BD and depressed mothers [34]. While both parents with BD and with MDD have been found to show negative affect towards their children, Inoff-Germain and colleagues (1992) have found that mothers with BD show more negative judgments, more negative affect, and had more negative reactions toward their children than mothers with MDD and mothers with no mental disorder [41]. Given these findings, it is evident that both parents with MDD and BD show signs of negative affect which can be considered a negative parenting trait. However, the research results on whether parents with BD or parents with MDD demonstrate more evidence of negative affect is mixed.

Parental structure, defined as organization, consistency, stability, and predictability [30] has been explored in parents with BD and MDD. Parents with BD have been found to provide less structure to their offspring, specifically, they are more likely than healthy control parents to provide low levels of organization, consistency, and stability in the home during middle childhood [21]. High levels of the trait neuroticism, a tendency to react with elevated emotionality to stressors, in parents with BD have also been hypothesized to elicit unstable and disorganized caregiving environments in middle childhood [13, 42, 43]. Parents with MDD show more unpredictable parental behaviours, such as inconsistent parenting, compared to healthy parents [32]. Inconsistent parenting can lead to unpredictable situations and feelings of confusion and insecurity in the OBD and the OMDD, especially if offspring are mostly uninformed about their parent's mental health disorder [44]. While low levels of structure are seen in both parents with MDD and BD, it is more characteristic of parents with BD as the disorder causes extreme mood swings [45].

The findings on parental control, defined as frequency and type of disciplinary strategies, are mixed. The literature on control in parents with MDD is scarce. However, lax control (i.e., carelessness, or negligent control) has been found in parents with MDD in one study by Du Rocher Schudlich et al. (2008) [46]. Most of the literature states that parents with MDD are inconsistent and struggle with effective discipline [37, 47, 48]. Parents with higher levels of depressive symptoms reported lower levels of parental monitoring and more inconsistent discipline [49]. Several studies found BD parents scored lower on control compared to controls [50, 21], while others found that they scored higher on control compared to controls [51, 45, 52]. Yet, other studies have reported no significant differences in BD-parented families on control compared to healthy participants [52].

The similarities seen in both disorders are likely due to the occurrence of depressive episodes shared by the two disorders. The parenting behaviours of mothers and fathers who have MDD or BD may reflect correlates of their depressive symptoms, such as physical and emotional unavailability, unresponsiveness, confusion, self-absorption, negative or sad affect, hopelessness, and irritability [53]. Such characteristics particularly may impair parents' communication with their children as well as their success in setting limits and granting autonomy to their children. Research indicates more negative communication styles in bipolar parents compared to controls

[54] and parents with other, non-bipolar, psychopathology [41].

Differences in Parenting

We see few differences in parenting behaviours between parents with MDD and BD. The main differences can be explained by the manic episodes seen in BD which are absent in MDD. Major depressive disorder is unipolar, meaning that there are no manic episodes or symptoms while bipolar disorder includes symptoms of both depression and mania. While symptoms of depression lead to deficits in parenting abilities, mania presents its own challenges to parenting. In the manic phase, mothers report being very self-centred, exhibited poor judgment, and behaved in ways that made them poor role models during their manic phases [55]. Since manic episodes are characterized by high levels of activity and impulsivity, this can translate into unpredictability and inconsistency in parenting [55]. Joyce (1984) found that within the bipolar group, those who reported high overprotection had more hospitalizations for mania, suggesting that this parenting dimension may be associated with the severity of the manic symptoms [56].

Despite the evident negative parenting behaviours accompanying both MDD and BD, offspring of mothers with BD have been found to display more comfortable and happy interactions with their mothers than the offspring of depressed mothers [34]. Although we see some differences in the parenting behaviours between parents with MDD and BD, studies have found no differences in general on family functioning and environment between the two disorders [46, 57, 58].

OBD Psychopathology

The OBD are at significantly increased risk for developing a wide range of severe psychiatric disorders and accompanying dysfunction [59]. A study conducted by Vandeleur and colleagues (2012) exploring mental disorders in the OBD and the OMDD found that rates of mood and anxiety disorders were elevated among the OBD (34.5% any mood; 42.5% any anxiety) [60]. Specifically, mood disorders and especially BD were distinctly elevated among the OBD compared to the offspring of parents without BD [60]. Parental concordance for bipolar spectrum disorders was associated with a further elevation in the rates of mood disorders in offspring (64.3% both parents versus 27.2% one parent) [60].

In a systematic review by Stapp and colleagues (2020), the OBD did not differ significantly in the prevalence of psychiatric disorders from offspring of parents with unipolar depression [34, 58, 61]. Evidence of increased rates of disruptive behaviour disorders and attention deficit hyperactive disorder (ADHD) in the OBD has also been found [62]. However, the recent findings of Birmaher et al. (2009) showed that increased rates of disruptive behaviour disorders and ADHD in the OBD are attributable to general parental psychopathology or other related factors rather than to parental mood disorders [63].

Mediators Between Parental BD and Offspring Psychopathology

Several parental factors have been found to mediate the relationship between parental BD and offspring psychopathology. Parental control emerged as the strongest mediator of the relation between parents' BD and offspring psychopathology in a study done by Iacono and colleagues (2018) [21]. However, a previous study with this sample found that both low support and low structure, but not control, were associated with higher scores for internalizing and externalizing

problems [13]. Low levels of structure provided by parents in middle childhood mediated the relation between having a parent with BD and elevated rates of internalizing and externalizing difficulties in the OBD during middle childhood [21]. Low structure in parenting has also been seen to increase the risk for later high-risk sexual behaviours and poor interpersonal functioning in the OBD [13, 42, 43]. High levels of over-reactivity, activation, and chaos, which can be categorized as low structure, in the household are particularly associated with emotional difficulties in children [25]. Structure provided by parents in middle childhood has been shown to influence cortisol reactivity in adolescence among the OBD [13, 50]. In turn, persistent abnormalities in individuals' biological sensitivity to stress have been associated with an increased vulnerability for the development of an affective disorder [64, 65]. Relative to control offspring, the OBD tend to experience more frequent and severe stressful life events [43]. Among those who eventually develop a mental disorder, negative life events often precede onset [66, 67]. Radke-Yarrow and colleagues (1993) found that children of bipolar mothers developed more problems later in childhood than children of unipolar (depressed) mothers and suggested that children may be more vulnerable to psychosocial stresses created by bipolar illness as they grow older [68]. To conclude, low control, low structure, and low support exhibited by parents with BD, along with stressful life events often accompanied with having a parent with BD have been found to mediate the relationship between parental BD and offspring psychopathology.

OMDD Psychopathology

A study conducted by Vandeleur and colleagues (2012) exploring mental disorders in the OBD and the OMDD found that rates of mood and anxiety disorders were elevated among OMDD (25.5% any mood; 44.6% any anxiety) as compared to those of controls (12.6% any mood; 22.8% any anxiety) [60]. Moreover, the rates of depression did not differ between the OBD and the OMDD [60]. In addition, parental MDD independently predicted alcohol substance use disorder among older offspring in this research [60].

Pathways from Parental MDD to Offspring Psychopathology

The OMDD are at increased risk for psychopathology and internalizing and externalizing disorders more generally [69]. Several parental factors have been found to mediate the relationship between parental MDD and offspring psychopathology. In a study done by Lau and colleagues (2018), lower maternal and paternal warmth was independently associated with internalizing problems in the OMDD [31]. Lower maternal warmth alone predicted externalizing problems [31]. Specifically, Lau and colleagues (2018) found that in general, mother's internalizing psychopathology explained lower levels of affective practices which includes behaviours such as affection and warmth [31]. Those practices could lead, in turn, to an increase in children's maladaptive symptoms [70]. Du Rocher Schudlich and Cummings (2007) found that disrupted parenting, specifically parental rejection, lax control, and psychological control, by mothers and fathers partially mediated the relations between maternal and paternal dysphoric mood and children's internalizing and externalizing problems [71].

In a large, longitudinal, population-based study of Canadian youth ages 10 to 15, children's reports of both positive parenting behaviours (i.e., nurturance and monitoring) and negative parenting behaviours (i.e., rejection) mediated the relationship between parental depressive symptoms and children's internalizing (e.g., anxiety, depressive symptoms) and/or externalizing (e.g., aggression, noncompliance) problems [72].

Low warmth and high levels of parental criticism or intrusion have been found to directly influence depressive symptoms in the general population of youth [73, 74]. In addition, negative parenting style is mirrored in negative interactions in conflict situations with their children that, in turn, were found to be associated with higher self-reported symptoms of depression in youth [75].

Future Directions

The findings of the present review article have several potential implications. First, the high rates of psychiatric disorders in the OBD and the OMDD emphasize the need to pay particular attention to the offspring of parents with mood disorders. The results may have important implications for theories of abnormal development among the OBD and the OMDD. Lastly, the present findings not only emphasize the importance of parental practices as a means of mental illness prevention but also highlight the importance of addressing issues of control, support, structure, and negative affect in the home environment of the OBD and the OMDD.

This review article found that parenting behaviours demonstrated by both parents with MDD, and BD seemed to fall under three constructs: structure, support, control. However, the terminologies and measures varied. Therefore, there is a need for a common conceptual framework for parenting behaviours.

Few studies examined the impact of parenting behaviour of mothers compared to fathers with MDD or BD on offspring outcomes. Studies on resilience in the offspring of parents with mood disorders were few and far between. It would be beneficial to explore these areas of research further, especially as they can inform intervention and possibly reveal protective factors.

Families with one or more depressed parents often have additional factors that generally impose risk for children, such as substance use disorders, poverty, exposure to violence, minority status, cultural and linguistic isolation, and marital conflict, which interfere with good parenting qualities and healthy child-rearing environments [49]. Therefore, it would be beneficial to analyse the extent to which each of these additional factors interferes with parenting abilities if they work independently or as an additive or interaction with the effects of depression in parents.

Given the overwhelming evidence supporting parenting behaviours as a mediator in the relationship between parental BD or MDD and offspring psychopathology [21, 75], gaining more information on the mechanism behind how parenting behaviours explain this relationship would be valuable. Potential mediators between negative parental behaviours demonstrated by parents with MDD and BD and offspring psychopathology could be further explored such as stress, emotional dysregulation, and attachment. Negative parental behaviours have been found to negatively impact family functioning and the home environment in general which have been implicated in offspring psychopathology [45]. Therefore, it may be useful to focus on family functioning rather than parenting behaviours.

Conclusion

Parents with MDD and parents with BD share several negative parenting behaviours including low levels of support or care, negative affect, low levels of structure and control. While symptoms of depression lead to deficits in parenting abilities for both disorders, mania presents its own challenges to parenting as it is accompanied by self-centeredness, poor judgment, high levels of impulsivity, and overinvolvement. Although we see some differences in the parenting behaviours

between parents with MDD and BD, no differences have been found in family functioning and environment between the two disorders. Regardless, the OMDD and the OBD are at significantly increased risk for developing a wide range of severe psychiatric disorders and accompanying dysfunctions. Further, multiple specific parenting behaviours have been shown to mediate the relationship between parental MDD and BD and offspring psychopathology.

Further research based on a common conceptual framework for the study of parental behaviours and outcomes is needed. Research directly comparing MDD and BD as well as comparing mothers and fathers would be advantageous. Lastly, future research could examine factors related to resilience in offspring, specific mediators, and family functioning to better inform researchers, health care providers, and the general public on effective parenting skills that protect offspring from mental health problems.

References

- [1] Copeland, W. E, Wolke, D., Angold, A., & Costello, E. (2013). Adult psychiatric outcomes of bullying and being bullied by peers in childhood and adolescence. *JAMA Psychiatry* 70(4),419–426
- [2] Wittchen, H. U., Jacobi, F., Klose, M., & Ryl, L. (2010). Gesundheits- berichterstattung des Bundes Heft 51: *depressive Erkrankungen*. Robert Koch-Institut, Berlin
- [3] Tsuang, M. T., Taylor, L., & Faraone, S. V. (2004). An overview of the genetics of psychotic mood disorders. *J Psychiatr Res.*, 38, 3-15
- [4] Diflorio, A. & Jones, I. (2010). Is sex important? Gender differences in bipolar disorder. *Int Rev Psychiatry*, 22(5):437-52. DOI: 10.3109/09540261.2010.514601. PMID: 21047158.
- [5] Noble, R.E. (2005). Depression in women. *Journal of Metabolism* 54(5),49-52. PMID: 15877314.
- [6] Picco, L., Subramaniam, M., Abdin, E., Vaingankar, J. A., & Chong, S. A. (2017). Gender differences in major depressive disorder: findings from the Singapore Mental Health Study. *Singapore medical journal*, 58(11), 649–655. <https://doi.org/10.11622/smedj.2016144>
- [7] GBD Disease and Injury Incidence and Prevalence Collaborators. (2018). Global, regional, and national incidence, prevalence, and years lived with disability for 354 diseases and injuries for 195 countries and territories, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. *The Lancet*. DOI:[https://doi.org/10.1016/S0140-6736\(18\)32279-7](https://doi.org/10.1016/S0140-6736(18)32279-7)
- [8] American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). <https://doi.org/10.1176/appi.books.9780890425596>
- [9] Hodgins, S., Faucher, B., Zarac, A., & Ellenbogen, M. A. (2002). Children of parents with bipolar disorder: a population at high risk for major affective disorders. *Child Adolesc Psychiatr Clin N Am*, 11, 533–553. doi: 10.1016/S1056-4993(02)00002-0.
- [10] Fletcher, K., Parker, G. B., & Manicavasagar, V. (2013). Coping profiles in bipolar disorder. *Comprehensive psychiatry*, 54(8), 1177-1184.

- [11] Moon, E., Chang, J., Choi, S., Ha, T.H., Cha, B., Sang Cho, H., Park, J.M., Lee, B.D., Min Lee, Y., Choi, Y., Ha, K. (2014). Characteristics of stress-coping behaviours in patients with bipolar disorders. *Psychiatry Research* 218(1-2), 69-74. <https://doi.org/10.1016/j.psychres.2014.03.047>.
- [12] Bender, R. E., Alloy, L. B., Sylvia, L. G., Uroševic, S., & Abramson, L. Y. (2010). Generation of life events in bipolar spectrum disorders: A re-examination and extension of the stress generation theory. *Journal of clinical psychology*, 66(9), 907-926
- [13] Ellenbogen, M. & Hodgins, S. (2004). The impact of high neuroticism in parents on children's psychosocial functioning in a population at high risk for major affective disorder: A family-environmental pathway of intergenerational risk. *Development and Psychopathology*, 16, 113-36. [10.1017/S0954579404044438](https://doi.org/10.1017/S0954579404044438).
- [14] Lohoff F. W. (2010). Overview of the genetics of major depressive disorder. *Current psychiatry reports*, 12(6), 539–546. <https://doi.org/10.1007/s11920-010-0150-6>
- [15] Weissman, M. M., Wickramaratne, P., Nomura, Y., Warner, V., Pilowsky, D., & Verdelli, H. (2006). Offspring of depressed parents: 20 years later. *American Journal of Psychiatry*, 163(6):1001–1008
- [16] Barnett, J. H., & Smoller, J. W. (2009). The genetics of bipolar disorder. *Neuroscience*, 164(1), 331–343. <https://doi.org/10.1016/j.neuroscience.2009.03.080>
- [17] Hillegers, M. H., Reichart, C. G., Wals, M., Verhulst, F. C., Ormel, J., & Nolen, W. A. (2005). Five-year prospective outcome of psychopathology in the adolescent offspring of bipolar parents. *Bipolar disorders*, 7(4), 344-350.
- [18] Duffy, A., Alda, M., Crawford, L., Milin, R., & Grof, P. (2007). The early manifestations of bipolar disorder: a longitudinal prospective study of the offspring of bipolar parents. *Bipolar Disorders*, 9(8), 828-838.
- [19] Udal, A. H., Øygarden, B., Egeland, J., Malt, U. F., & Groholt, B. (2012). Memory in early onset bipolar disorder and attention-deficit/hyperactivity disorder: similarities and differences. *Journal of abnormal child psychology*, 40(7), 1179-1192.
- [20] Birmaher, B., Gill, M. K., Axelson, D. A., Goldstein, B. I., Goldstein, T. R., Yu, H., ... & Keller, M. B. (2014). Longitudinal trajectories and associated baseline predictors in youths with bipolar spectrum disorders. *American journal of psychiatry*, 171(9), 990-999.
- [21] Iacono, V., Beaulieu, L., Hodgins, S., & Ellenbogen, M. A. (2018). Parenting practices in middle childhood mediate the relation between growing up with a parent having bipolar disorder and offspring psychopathology from childhood into early adulthood. *Development and psychopathology*, 30(2), 635-649.
- [22] Alloy, L. B., Abramson, L. Y., Urosevic, S., Walshaw, P. D., Nusslock, R., & Neeren, A. M. (2005). The psychosocial context of bipolar disorder: environmental, cognitive, and developmental risk factors. *Clinical psychology review*, 25(8), 1043-1075.
- [23] Post, R. M., Leverich, G. S., Xing, G., & Weiss, S. R. (2001). Developmental vulnerabilities to the onset and course of bipolar disorder. *Development and psychopathology*, 13(3), 581-598.

- [24] Lovejoy, M. Christine, E O'Hare, & G Neuman. (2000). "Maternal depression and parenting behaviour: A meta-analytic review." *Clinical psychology review* 20(5), 561-592.
- [25] Calam, R., Jones, S., Sanders, M. R., Dempsey, R., & Sadhnani, V. (2012). Parenting and the emotional and behavioural adjustment of young children in families with a parent with bipolar disorder. *Behavioral and Cognitive Psychotherapy*, 40(4), 425-437.
- [26] Moilanen, K.L., Shaw, D.S., Dishion, T.J., Gardner, F., Wilson, M. (2009). Predictors of Longitudinal Growth in Inhibitory Control in Early Childhood. *Social Development*. 19(2):326-347.
- [27] Eisenberg, N., Taylor, Z.E., Widaman, K.F., Spinrad, T.L. (2015). Externalizing symptoms, effortful control, and intrusive parenting: A test of bidirectional longitudinal relations during early childhood. *Development and Psychopathology*, 27(4), 953–968
- [28] Yap, M. B. H., & Jorm, A. F. (2015). Parental factors associated with childhood anxiety, depression, and internalizing problems: A systematic review and meta-analysis. *Journal of affective disorders*, 175, 424-440.
- [29] Yap, M.B.H., Pilkington, P.D., Ryan, S.M., Jorm, A.F. (2014). Parental factors associated with depression and anxiety in young people: A systematic review and meta-analysis. *Journal of affective disorders*, 156, 8-23. <https://doi.org/10.1016/j.jad.2013.11.007>.
- [30] Slater, M. A., & Power, T. G. (1987). *Parenting Dimensions Inventory*. In Multidimensional assessment of parenting in single-parent families. In J. P. Vincent (Ed.), *Advances in family intervention, assessment and theory: An annual compilation of research* (Vol. 4, pp. 197-228). Greenwich, CT: JAI Press.
- [31] Lau, P., Hawes, D. J., Hunt, C., Frankland, A., Roberts, G., Wright, A., ... & Mitchell, P. B. (2018). Family environment and psychopathology in offspring of parents with bipolar disorder. *Journal of affective disorders*, 226, 12-20.
- [32] Cummings, M. E., Keller, P. S., & Davies, P. T. (2005). Towards a family process model of maternal and paternal depressive symptoms: Exploring multiple relations with child and family functioning. *Journal of Child Psychology and Psychiatry*, 46(5), 479-489.
- [33] Wilson, S. C. & Durbin, E. (2010). Effects of paternal depression on fathers' parenting behaviours: A meta-analytic review. *Clinical Psychology Review*, 30(2), 167-180.
- [34] Tarullo, L. B., DeMulder, E. K., Martinez, P. E., & Radke-Yarrow, M. (1994). Dialogues with preadolescents and adolescents: Mother-child interaction patterns in affectively ill and well dyads. *Journal of abnormal child psychology*, 22(1), 33-51.
- [35] Dunn, B. D., German, R. E., Khazanov, G., Xu, C., Hollon, S. D., & DeRubeis, R. J. (2020). Changes in Positive and Negative Affect During Pharmacological Treatment and Cognitive Therapy for Major Depressive Disorder: A Secondary Analysis of Two Randomized Controlled Trials. *TEACHING Exceptional Children*, 8(1), 64–68. <https://doi.org/10.1177/004005990703900310>
- [36] Rowland, J. E., Hamilton, M. K., Lino, B. J., Ly, P., Denny, K., Hwang, E. J., ... & Green, M. J. (2013). Cognitive regulation of negative affect in schizophrenia and bipolar disorder. *Psychiatry research*, 208(1), 21-28.

- [37] Cummings, E. M., & Davies, P. T. (1994). Maternal depression and child development. *Journal of child psychology and psychiatry*, 35(1), 73-122.
- [38] Miklowitz, D. J., Goldstein, M. J., Nuechterlein, K. H., Snyder, K. S., & Mintz, J. (1988). Family factors and the course of bipolar affective disorder. *Archives of general psychiatry*, 45(3), 225-231.
- [39] Celano, M., Bakeman, R., Gaytan, O., Smith, C.O., Kico, A., and Henderson, S. (2008). Caregiver depressive symptoms and observed family interaction in low-income children with persistent asthma. *Family Process*, 47, 7-20.
- [40] Suppes, T., Eberhard, J., Lemming, O., Young, A., & McIntyre, R. (2017). Anxiety, irritability, and agitation as indicators of bipolar mania with depressive symptoms: a post hoc analysis of two clinical trials. *International Journal of Bipolar Disorder* 5, 36. <https://doi.org/10.1186/s40345-017-0103-7>
- [41] Inoff-Germain, G., Nottelmann, E.D., Radke-Yarrow, M. (1992). Evaluative communications between affectively ill and well mothers and their children. *Journal of Abnormal Child Psychology*, 20, 189–212.
- [42] Nijjar, R., Ellenbogen, M. A., & Hodgins, S. (2016). Sexual risk behaviours in the adolescent offspring of parents with bipolar disorder: prospective associations with parents' personality and externalizing behaviour in childhood. *Journal of abnormal child psychology*, 44(7), 1347-1359.
- [43] Ostiguy, C. S., Ellenbogen, M. A., & Hodgins, S. (2012). Personality of parents with bipolar disorder and interpersonal functioning among their offspring: A prospective 10-year study. *Development and psychopathology*, 24(2), 573-587.
- [44] Lenz, A,. (2005). Vorstellungen der Kinder?? ber die psychische Erkrankung ihrer Eltern: eine explorative Studie. *Prax Kin- derpsychol Kinderpsychiatr*. 54(5):382–398
- [45] Chang, K. D., Blasey, C., Ketter, T. A., & Steiner, H. (2001). Family environment of children and adolescents with bipolar parents. *Bipolar disorders*, 3(2), 73-78
- [46] Du Rocher Schudlich, T., Youngstrom, E., Calabrese, J., & Findling, R. (2008). The Role of Family Functioning in Bipolar Disorder in Families. *Journal of abnormal child psychology*, 36, 849-63. 10.1007/s10802-008-9217-9
- [47] Downey, G., & Coyne, J. C. (1990). Children of depressed parents: an integrative review. *Psychological bulletin*, 108(1), 50.
- [48] Kaslow, N. J., Deering, C. G., & Racusin, G. R. (1994). Depressed children and their families. *Clinical Psychology Review*, 14(1), 39-59.
- [49] England, M. J. & Sim, L. J. (2009). Associations Between Depression in Parents and Parenting, Child Health, and Child Psychological Functioning. National Research Council (US) and Institute of Medicine (US) Committee on Depression, Parenting Practices, and the Healthy Development of Children, *Depression in parents, parenting, and children: Opportunities to improve identification, treatment, and prevention*. National Academies Press. <https://www.ncbi.nlm.nih.gov/books/NBK215128/>

- [50] Ellenbogen, M. A., & Hodgins, S. (2009). Structure provided by parents in middle childhood predicts cortisol reactivity in adolescence among the offspring of parents with bipolar disorder and controls. *Psychoneuroendocrinology*, 34(5), 773-785.
- [51] Ferreira, G. S., Moreira, C. R., Kleinman, A., Nader, E. C., Gomes, B. C., Teixeira, A. M. A., ... & Caetano, S. C. (2013). Dysfunctional family environment in affected versus unaffected offspring of parents with bipolar disorder. *Australian & New Zealand Journal of Psychiatry*, 47(11), 1051-1057
- [52] Romero, S., DelBello, M. P., Soutullo, C. A., Stanford, K., & Strakowski, S. M. (2005). Family environment in families with versus families without parental bipolar disorder: a preliminary comparison study. *Bipolar disorders*, 7(6), 617-622.
- [53] Radke-Yarrow, M., Cummings, E. M., Kuczynski, L., & Chapman, M. (1985). Patterns of attachment in two-and three-year-olds in normal families and families with parental depression. *Child development*, 884-893.
- [54] Vance, Y. H., Huntley Jones, S., Espie, J., Bentall, R., & Tai, S. (2008). Parental communication style and family relationships in children of bipolar parents. *The British journal of clinical psychology*, 47(3), 355-359 <https://doi.org/10.1348/014466508X282824>
- [55] Venkataraman, M., & Ackerson, B. J. (2008). Parenting among mothers with bipolar disorder: strengths, challenges, and service needs. *Journal of Family Social Work*, 11(4), 389-408.
- [56] Joyce, P. R. (1984). Parental bonding in bipolar affective disorder. *Journal of Affective Disorders*, 7(3-4), 319-324.
- [57] Weintraub, S. (1987). Risk Factors in Schizophrenia: The Stony Brook High-risk Project, *Schizophrenia Bulletin*, 13(3), 1987, 439-450. <https://doi.org/10.1093/schbul/13.3.439>
- [58] Stapp, E. K., Mendelson, T., Merikangas, K. R., & Wilcox, H. C. (2020). Parental bipolar disorder, family environment, and offspring psychiatric disorders: A systematic review. *Journal of affective disorders*, 268, 69-81
- [59] Henin, A., Biederman, J., Mick, E., Sachs, G. S., Hirshfeld-Becker, D. R., Siegel, R. S., McMurrich, S., Grandin, L., & Nierenberg, A. A. (2005). Psychopathology in the offspring of parents with bipolar disorder: a controlled study. *Biological psychiatry*, 58(7), 554-561. <https://doi.org/10.1016/j.biopsych.2005.06.010>
- [60] Vandeleur, C., Rothen, S., Gholam-Rezaee, M., Castelao, E., Vidal, S., Favre, S., Ferrero, F., Halfon, O., Fumeaux, P., Merikangas, K.R., Aubry, J.M., Burstein, M., & Preisig, M. (2012). Mental disorders in offspring of parents with bipolar and major depressive disorders. *Bipolar Disorder*, 14, 641-653.
- [61] Hammen, C., Burge, D., Burney, E., & Adrian, C. (1990). Longitudinal study of diagnoses in children of women with unipolar and bipolar affective disorder. *Archives of general psychiatry*, 47(12), 1112-1117.
- [62] Birmaher, B., Axelson, D., Goldstein, B. et al. (2010). Psychiatric disorders in preschool offspring of parents with bipolar disorder: the Pittsburgh Bipolar Offspring Study (BIOS). *American Journal of Psychiatry*, 167, 321-330

- [63] Birmaher, B., Axelson, D., Monk, K. et al. (2009). Lifetime psychiatric disorders in school-aged offspring of parents with bipolar disorder: the Pittsburgh Bipolar Offspring Study. *Archives of General Psychiatry*, 66, 287–296
- [64] Ellenbogen, M. A., Hodgins, S., Linnen, A. M., & Ostiguy, C. S. (2011). Elevated daytime cortisol levels: a biomarker of subsequent major affective disorder?. *Journal of affective disorders*, 132(1-2), 265-269.
- [65] Goodyer, I. M., Bacon, A., Ban, M., Croudace, T., & Herbert, J. (2009). Serotonin transporter genotype, morning cortisol and subsequent depression in adolescents. *The British Journal of Psychiatry*, 195(1), 39-45
- [66] Hillegers, M. H., Burger, H., Wals, M., Reichart, C. G., Verhulst, F. C., Nolen, W. A., & Ormel, J. (2004). Impact of stressful life events, familial loading and their interaction on the onset of mood disorders: study in a high-risk cohort of adolescent offspring of parents with bipolar disorder. *The British Journal of Psychiatry*, 185(2), 97-101
- [67] Wals, M., van Os, J., Reichart, C. G., Hillegers, M. H., Ormel, J., Verhulst, F. C., & Nolen, W. A. (2004). Multiple dimensions of familial psychopathology affect risk of mood disorder in children of bipolar parents. *American journal of medical genetics. Part B, Neuropsychiatric genetics: the official publication of the International Society of Psychiatric Genetics*, 127B(1), 35–41. <https://doi.org/10.1002/ajmg.b.20165>
- [68] Radke-Yarrow, M., Nottelmann, E., Belmont, B., & Welsh, J. D. (1993). Affective interactions of depressed and nondepressed mothers and their children. *Journal of Abnormal Child Psychology*, 21(6), 683-695
- [69] Miettunen, J., Murray, G. K., Jones, P. B., Mäki, P., Ebeling, H., Taanila, A., ... & Moilanen, I. (2014). Longitudinal associations between childhood and adulthood externalizing and internalizing psychopathology and adolescent substance use. *Psychological medicine*, 44(8), 1727-1738.
- [70] Bellina, M., Grazioli, S., Garzitto, M., Mauri, M., Rosi, E., Molteni, M., ... & Nobile, M. (2020). Relationship between parenting measures and parents and child psychopathological symptoms: a cross-sectional study. *BMC psychiatry*, 20(1), 1-11
- [71] Du Rocher Schudlich, T. D., & Cummings, E. M. (2007). Parental dysphoria and children's adjustment: marital conflict styles, children's emotional security, and parenting as mediators of risk. *Journal of abnormal child psychology*, 35(4), 627–639. <https://doi.org/10.1007/s10802-007-9118-3>
- [72] Elgar, F. J., Mills, R. S., McGrath, P. J., Waschbusch, D. A., & Brownridge, D. A. (2007). Maternal and paternal depressive symptoms and child maladjustment: the mediating role of parental behavior. *Journal of abnormal child psychology*, 35(6), 943–955. <https://doi.org/10.1007/s10802-007-9145-0>
- [73] Barber, B. K., Stoltz, H. E., & Olsen, J. A. (2005). Parental support, psychological control, and behavioural control: Assessing relevance across time, culture, and method: IV. Assessing relevance across time: US analyses and results. *Monographs of the Society for Research in Child Development*.
- [74] Gray, M. R., & Steinberg, L. (1999). Unpacking authoritative parenting: Reassessing a multi-

dimensional construct. *Journal of Marriage and the Family*, 574-587

- [75] Olino, T. M., McMakin, D. L., Nicely, T. A., Forbes, E. E., Dahl, R. E., & Silk, J. S. (2016). Maternal depression, parenting, and youth depressive symptoms: mediation and moderation in a short-term longitudinal study. *Journal of Clinical Child & Adolescent Psychology*, 45(3), 279-290.