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Automatic Imitation in Comorbid PTSD & Alcohol Use Disorder and Controls: an RCT of Intranasal Oxytocin

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ABSTRACT

Introduction: Mimicking movements of others makes both the imitating and imitated partners feel closer. Oxytocin may increase focus on others and has been shown to increase automatic imitation in healthy controls (HC). However, this has not been replicated, and oxytocin's effects on automatic imitation have not been demonstrated in clinical populations. This study attempts to replicate effects on HC and examine effects on people with comorbid posttraumatic stress disorder and alcohol use disorder (PTSD-AUD).
Methods: Fifty-four males with PTSD-AUD and 43 male HC received three intranasal treatment conditions (placebo, oxytocin 20 International Units (IU), and oxytocin 40 IU) in a randomized order, across three separate testing days, as part of a double-blind, crossover parent study. At 135 min post-administration, each performed the imitation-inhibition task, which quantifies automatic imitation as the congruency effect (CE). After exclusions, the final analyzed data set included 49 participants with PTSD-AUD and 38 HC.
Results: In HC, oxytocin 20 IU demonstrated a statistically significant increase in CE, and 40 IU showed a trend-level increase. In PTSD-AUD, oxytocin did not significantly increase CE. Post-hoc analysis showed the PTSD-AUD group had higher CE than HC on placebo visits.
Discussion: Our data suggest PTSD-AUD is associated with higher automatic imitation than HC in the absence of oxytocin administration. We successfully replicated findings that oxytocin increases automatic imitation in HC. This demonstrates an unconscious motor effect induced by oxytocin, likely relevant to more complex forms of imitative movements, which have the potential to improve social connection. We did not find a significant effect of oxytocin on automatic imitation in PTSD-AUD. Future research should examine imitation in both sexes, at peak oxytocin levels, and on increasingly complex forms of imitation.

1. Introduction

Humans automatically and unconsciously mimic the movements of others, such as facial expressions and posture (Chartrand and Lakin, 2013). This likely contributes to social connection as mimicry leads to both imitating and imitated partners feeling more liked by, and closer to, the other (Stel and Vonk, 2010). One aspect of the tendency to mimic can be quantified (Chartrand and Lakin, 2013; Heyes, 2011; Leighton et al., 2010) by the imitation-inhibition task, which focuses on automatic imitation of simple finger movements. More imitation on this task has been linked to prosocial states and traits, where focus is more on others than the self. For example, priming with prosocial statements

or cartoon movies of helping others increases automatic imitation (Wang and Hamilton, 2013), while priming with self-referential statements decreases automatic imitation (Spengler et al., 2010). Similarly, ratings of self-centered, narcissistic traits are inversely correlated with automatic imitation (Obhi et al., 2014). Despite its social relevance, little is known about how to pharmacologically manipulate automatic imitation, or whether it is abnormal in psychiatric disorders with social deficits.

The oxytocin system is critical to pair bonding and infant-parent attachment. Oxytocin administration in humans may also shift focus from self to other (Zhao et al., 2016), implying it could increase the tendency to imitate other people's movements. Indeed, intranasal

oxytocin increased synchrony, the intersection of imitation and timing, in body language (Ramseyer et al., 2019), in dance movements (Josef et al., 2019), and on a similar finger task (Gebauer et al., 2016). De Coster et al. (2014) showed that a single intranasal oxytocin administration also increases automatic imitation of finger movements in undergraduates, even when the element of timing is removed. However, this result has not been replicated, and oxytocin's effects on automatic imitation in clinical populations have not yet been investigated. Individuals with comorbid posttraumatic stress disorder and alcohol use disorder (PTSD-AUD) have poor social connection (Grant et al., 2015; Ouimette et al., 2006) and evidence of aberrant self-other processing (Cracco et al., 2020; Frewen et al., 2017; Hudson et al., 2020). Thus, they may have low automatic imitation, which could be increased by oxytocin. Determining if automatic imitation is abnormal in PTSD-AUD, and if a single administration of oxytocin increases imitation in this population, could reveal a promising intervention.

The dose-response curve for intranasal oxytocin's effects on automatic imitation is also unknown. Therefore, we examined the effects of two dosages of intranasal oxytocin on automatic finger imitation in individuals with PTSD-AUD and age-matched healthy controls (HC). We 1) assessed baseline differences between PTSD-AUD and HC groups, 2) attempted to replicate oxytocin's effects on automatic imitation in HC, and 3) examined oxytocin's effects on automatic imitation in individuals with PTSD-AUD, expecting oxytocin to increase automatic imitation in both groups.

2. Materials and methods

2.1. Participants

Fifty-four males with PTSD-AUD (mean age 50.48 ± 13.02) and 43 male HC (48.46 ± 13.31) recruited from the San Francisco Bay Area received the intervention. No participants had contraindications to receiving intranasal oxytocin (see supplement S2.1-2). PTSD-AUD participants met criteria for both PTSD and AUD on the Clinician Administered PTSD Scale and the Structured Clinical Interview for DSM-5. We excluded individuals in legally mandated alcohol treatment programs, or with acute alcohol withdrawal, suicidal ideation with plan within 30 days, or a suicide attempt within six months. HC had no history of PTSD or substance use disorders, though mild AUD was allowed. All gave written informed consent and were compensated for time and travel. For parent study details, see Stauffer et al. (2019).

2.2. Procedure

As the oxytocin literature suffers from mixed results with poor replication, in order to separate exploratory from confirmatory analyses, we pre-registered our methods, hypotheses, and analytic plan: https://aspredicted.org/vy72m.pdf. While this was after data collection, the first author and statistician remained blinded when the statistical analysis plan for this arm of the study was chosen and pre-registered. We used a placebo-controlled, double-blind, crossover design. Across three testing days ("visits"), separated by a washout period of at least one week, participants received each of three intranasal treatment conditions (placebo, oxytocin 20 International Units [IU], and oxytocin 40 IU) in a randomized order. We conducted unrelated tasks, such as a cue-induced-craving of alcohol paradigm and a fear-potentiated acoustic startle paradigm, published separately, and then, ~135 min after intranasal administration, performed the imitation-inhibition task (see Fig.S1; adapted from De Coster et al., 2014 and Brass et al., 2000) to quantify automatic imitation.

During the imitation-inhibition task, participants first depressed two keyboard keys with the index and middle fingers of their right hand, and were told to lift and replace their index finger as fast as possible whenever the screen displayed a '1', or their middle finger whenever the screen displayed a '2.' They were told to respond only to this

number, even though hand images would also appear. Then, during each trial, the screen displayed a '1' or '2' between the index and middle finger of hand images that, like participants, made index or middle finger lifting movements. The screen randomly displayed three trial types: a) neutral trials, where the hand was stationary, b) congruent trials, where the hand performed the same movement that the participant was instructed to perform, and c) incongruent trials, where the hand performed the finger movement different from what the participant was instructed to perform. Participants completed 12 practice trials and then 50 experimental trials, ~5 min in total (Cracco et al., 2018). Reaction times (RT) during incorrect responses were excluded (see S2.2).

The *congruency effect* (CE), a measure of automatic imitation, represents the RT difference between incongruent and congruent trials. Because slower incongruent RT can represent "trouble inhibiting imitation" of the other, and faster congruent RT can represent "easily imitating", higher CE encompasses two ways to express "more imitation" (Wang and Hamilton, 2013).

2.3. Statistical analysis plan

We used a multilevel model to evaluate the main effects of intranasal oxytocin dosage (placebo vs. oxytocin 20 IU, and placebo vs. oxytocin 40 IU) and group (PTSD-AUD vs. HC) on our primary outcome, CE. Given that we had strong group-specific *a priori* hypotheses, we used the same model to assess group differences in CE on the placebo day only, as well as to examine oxytocin effects on CE in the diagnostic groups separately. We expected: 1) the PTSD-AUD group's placebo-day CE would be lower compared to HC; and that oxytocin administration would increase CE in 2) HC, and 3) in PTSD-AUD.

We removed visits with CE greater than three SD above or below the mean for each dosage group (4 removed) and those with a non-response rate of over 50 % for all trials on a specific visit (1 removed). We log-transformed our non-normal outcome of CE (z-skew = 6.1 corrected to 2.69, and z-kurtosis = 4.1 corrected to 2.13; Kim, 2013). The final analyzed data set included 138 visits from 49 participants with PTSD-AUD and 107 visits from 38 healthy controls. We included a treatment order variable, and group-by-dosage and group-by-order interaction terms in the models. We included candidate confounders in final models only if these predicted the outcome in univariate analysis at the $p < 0.10$ level. Statistical analysis was done using R (version 1.1463; R Foundation for Statistical Computing, Vienna, Austria) and the "nlme: Linear and Nonlinear Mixed Effects Models.R" package (version 3.1–131).

3. Results

3.1. Pre-registered analysis

3.1.1. Congruency effect model, overall group

Age significantly increased CE in univariate regression and was included in the multilevel model. Race and positive urine toxicologies (THC, cocaine, benzodiazepines, amphetamines, and methamphetamines) were not significant univariate predictors. The multilevel model revealed that oxytocin increased logCE in the overall sample 5.2 % beyond placebo (converted from ΔlogCE = 0.02), equivalent to a 6 msec increase in our average subject (95 %CI, 0.11–11.76 msec; see S3.1 − 1). Both dosages had similar positive effect sizes on logCE, with p = 0.049 for 20 IU and p = 0.08 for 40 IU (Table 1a). There was no effect of treatment order, and no significant group-by-dosage interactions in our models, though one combination showed a trend.

3.1.2. Congruency effect model, HC group only

The same model, limited to HC, revealed that logCE was significantly increased by oxytocin 20 IU, and by 40 IU at a trend level (see Table 1c; Fig. 1b).

Table 1
Summary of Multilevel Model Regression Analyses of Congruency Effect

Model 1a. [Both groups] Predictors of Log_{10}(CE)				
Variable	Level	Effect(ΔlgCE)	SE	p
OXT Dosage*	(ref. = Placebo)	–	–	–
	20 IU*	**0.02**	**0.01**	**0.049**
	40 IU	0.02	0.01	0.08
Diagnosis Group	(ref. = Control)	–	–	–
	PTSD-AUD	0.02	0.03	0.47
Age*		**0.00**	**0.00**	**< 0.01**
PTSD-AUD x 20 IU Dosage		−0.01	0.01	0.56
PTSD-AUD x 40 IU Dosage		−0.03	0.02	0.08
Crossover Order				> 0.34
Diagnosis Group x Order				> 0.34

Model 1b.† [Placebo visit only] Predictors of Log_{10}(CE)				
Variable	Level	Effect(ΔlgCE)	SE	p
OXT Dosage	(Placebo only)	–	–	–
Diagnosis Group*	(ref. = Control)	–	–	–
	PTSD-AUD*	**0.04**	**0.01**	**0.01**
Age*		**<0.01**	**<0.01**	**< 0.01**

Model 1c. [HC only] Predictors of Log_{10}(CE)				
Variable	Level	Effect(ΔlgCE)	SE	p
Diagnosis Group	(HC only)	–	–	–
OXT Dosage*	(ref. = Placebo)	–	–	–
	20 IU*	**0.02**	**0.01**	**0.04**
	40 IU	0.02	0.01	0.06
Age*		**<0.01**	**<0.01**	**<0.01**
Crossover Order				> 0.31

Model 1d. [PTSD-AUD only] Predictors of Log_{10}(CE)				
Variable	Level	Effect(ΔlgCE)	SE	p
Diagnosis Group	(PTSD-AUD only)	–	–	–
OXT Dosage	(ref. = Placebo)	–	–	–
	20 IU	0.01	0.01	0.20
	40 IU	−0.01	0.01	0.51
Age*		**<0.01**	**<0.01**	**<0.01**
Crossover Order				> 0.15

Abbrev.: CE = congruency effect; Effect = per unit effect size (regression coefficient); Δlg = change in log units; SE = standard error; OXT = oxytocin; IU = international units; PTSD = posttraumatic stress disorder; AUD = alcohol use disorder. †Post-hoc analysis.

3.1.3. Congruency effect model, PTSD-AUD group only

The same model, limited to PTSD-AUD participants, revealed that neither dosage of oxytocin significantly increased logCE (Table 1d; Fig. 1c).

3.2. Post-hoc analysis

3.2.1. Placebo visits

The same model, limited to placebo visits only, revealed that the PTSD-AUD group, compared to HC, had significantly higher CE (see Table 1b; Fig. 1a) on the placebo visit.

3.2.2. Equivalence

To confirm oxytocin did not increase CE in PTSD-AUD, we used the "Two One-Sided Test" (TOST) to evaluate equivalence (Lakens, 2017). Aiming to detect equivalence with 80 % power in this dependent sample, and allowing for alpha of 0.1, we determined the tightest range within which we could determine equivalence was effect size d = -0.4 to 0.4, requiring n = 42 dependent pairs (present study: n = 46). Within this range, for the PTSD-AUD group, TOST showed equivalence between placebo and both dosages' effects on log CE [M($\pm$SD); placebo: 2.65 ($\pm$0.07) log-msec vs. oxytocin 20 IU: 2.66 ($\pm$0.07) log-
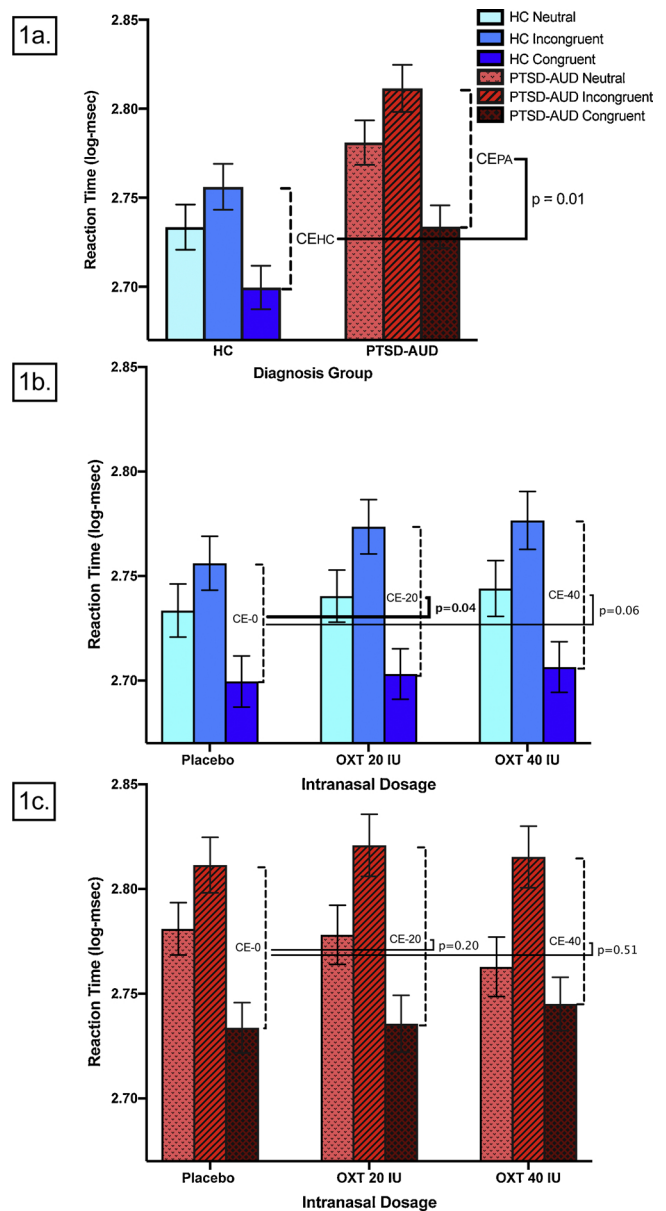

Fig. 1. (1a) Placebo-visit logCE was higher in PTSD-AUD compared to HC.†
(1b) Oxytocin increased logCE in the HC group. (1c) Oxytocin did not increase logCE in the PTSD-AUD group.
Abbrev.: HC = healthy controls; PTSD = post-traumatic stress disorder; AUD = Alcohol Use Disorder; CE = congruency effect; CEHC = CE in HC; CEPA = CE in PTSD-AUD; IU = international units; OXT = oxytocin. Neutral = average RT for neutral trials; Incongruent = average RT for incongruent trials; Congruent = average RT for congruent trials (see 2.2). Vertical bars indicate standard error of the mean (SEM). †Post-hoc analysis.

msec; p < 0.05; and vs. oxytocin 40 IU: 2.64 ($\pm$0.07) log-msec; p < 0.02].

4. Discussion

We examined oxytocin's effects on automatic imitation of finger movements in both a clinical and a control population. We found that: 1) PTSD-AUD was associated with higher automatic imitation than HC on the placebo day; and 2) intranasal oxytocin increased automatic imitation in the HC group, replicating prior work (De Coster et al., 2014). Given our pre-registered analysis, this replication supports the hypothesis that oxytocin administration increases automatic imitation

in healthy individuals. We also found that 3) oxytocin did not alter automatic imitation in PTSD-AUD. Therefore, our data does not support oxytocin as a treatment for imitative abnormalities in PTSD-AUD.

Further strengthening our finding that oxytocin increases automatic imitation in HC, we saw a similar magnitude of increase from both the 20 and 40 IU oxytocin dosages. While the mechanism remains unclear, De Coster et al. (2014) proposed oxytocin may alter self-other distinction, thereby increasing automatic imitation. Oxytocin has already been shown to shift focus to the "other," not only on imitation tasks, but also on decision-making tasks (Zhao et al., 2016). Oxytocin administration has also been shown to alter neural networks involved in self-other distinction and focus; the same networks that are activated with imitation changes on our imitation-inhibition task (Brass et al., 2009). Regardless of mechanism, our results imply that oxytocin administration increases motor imitation on an automatic (i.e., unconscious) level. This automatic motor effect may be one of many effects oxytocin induces when increasing more complex imitation, such as synchrony in body language (Ramseyer et al., 2019) and dance (Josef et al., 2019). Better understanding how and when oxytocin affects complex motor imitation, which increases closeness and empathy, brings us one step closer to understanding oxytocin's nuanced effects on social connection.

Given problematic social relationships in both PTSD and AUD (Grant et al., 2015; Ouimette et al., 2006), we expected less placebo-day imitation in PTSD-AUD compared to HC group, but we found the opposite. This may explain why oxytocin did not increase imitation in the PTSD-AUD group; patients may have been at a ceiling level. Additionally, as attachment style modulates oxytocin's effects (Bartz et al., 2011), and as trauma often affects attachment, PTSD-AUD participants may suffer from attachment disruption that blunts oxytocin's effects. Other confounding influences in the PTSD-AUD population compared to HC, including higher distractibility, mistrust, and sensitivity to threat and incongruent behavior, may also have contributed to higher CE and insensitivity to oxytocin. Further work is necessary to clarify baseline characteristics of, and moderators of oxytocin's effects on, automatic imitation in PTSD-AUD.

Our study has several limitations. Our analytic plan was pre-registered after data collection. Though pre-registration is ideally done prior to data collection, it is increasingly viewed as a continuum of beneficial efforts undertaken at varying times (Benning et al., 2019), including using preexisting data (Nosek et al., 2018). Our first author and statistician remained blinded and our pre-registration defined the analytic plan before observing outcomes. Thus, we suggest this remains a strength, albeit a limited strength. We tested automatic imitation at 135 min post-administration, which is later than in most prior work and may be after oxytocin's peak effects. However, oxytocin is likely still elevated in the CSF at this time (Spengler et al., 2017; Striepens et al., 2013), timing of oxytocin effect varies greatly by brain region, and effects may occur in a non-linear fashion (Martins et al., 2020; also see S4-2). In our overall model, oxytocin significantly affected CE without significant group-by-dosage interactions, though there was a trend for this interaction with the 40 IU dosage. However, we did find group-specific effects when each group was examined separately. We were likely underpowered to detect group-by-dosage interactions. Additionally, our study included only men to decrease the heterogeneity of a relatively small sample. Given male-female differences in both the oxytocin system and automatic imitation tendencies, larger studies should examine the influence of sex. Lastly, we used the placebo visit to examine group differences, but this may not represent a true baseline.

In sum, using a pre-registered approach, we replicated the finding that intranasal oxytocin increases automatic imitation in HC. Oxytocin's increase of automatic imitation of finger movements demonstrates an unconscious motor effect likely relevant to more complex forms of imitative movements, which have the potential to improve social connection. We also found, contrary to expectation, that PTSD-AUD was associated with higher CE compared to HC under the placebo condition, and that oxytocin did not increase imitation in our clinical group.

Future research should investigate oxytocin's effects on automatic imitation in both sexes, at peak oxytocin levels, and on increasingly complex forms of imitation.

Declaration of Competing Interest

None.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.psyneuen.2020.104787.

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