Oxytocin facilitates communicative adjustment by upregulating broadband aperiodic neural activity

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Abstract

Oxytocin modulates various social behaviors. In humans, oxytocin has been shown to modulate recipient design, i.e. how communicators adjust to their presumed mutual knowledge. Here, we investigate electrophysiological correlates of the oxytocinergic modulation of recipient design. Fifty-one males were randomly assigned to receive double-blind intranasal administration of oxytocin or placebo. While acquiring magnetoencephalography, the participants engaged in a live communicative task with two different addressees, a child and an adult. Unbeknownst to the participants, a confederate played the role of both addressees, with matched communicative behaviors. All participants started the task by putting more emphasis on their communicative behaviors when they thought to be interacting with the child addressee. As the communication progressed, the oxytocin group reduced variability in their communicative adjustments, adapting more effectively than the placebo group to the matched communicative behavior of the addressees. The magnitude of this oxytocin-related dynamic communicative adjustment was proportional to increased broadband aperiodic power, an index of local synaptic activity, in the right prefrontal-temporal circuit. These findings indicate that oxytocin facilitates dynamic adjustments in recipient design by enhancing cortical signal-to-noise in a portion of the same cortical circuit known to support the production of novel communicative behaviors.

Key words:

recipient design; social interaction; oxytocin; aperiodic neural activity; ventral prefrontal

1 cortex

1 **Introduction**

2 Oxytocin is a pituitary neuromodulator that influences social behaviors by facilitating
3 perception of social cues and by regulating valuation and motivational processes (Bartz et al.,
4 2011; De Dreu, 2012; Gangopadhyay et al., 2021; Grinevich & Stoop, 2018; Ma et al., 2016;
5 Piva & Chang, 2018; Shamay-Tsoory & Abu-Akel, 2016). Recent works have focused on
6 oxytocin's ability to modulate mental state inferences and interpersonal coordination (Arueti
7 et al., 2013; De Coster et al., 2019; Domes et al., 2007; Levy, Goldstein, Zagoory-Sharon, et al.,
8 2016; Levy et al., 2017; Mu et al., 2016; Radke & de Bruijn, 2015; Spengler et al., 2017; Wu &
9 Su, 2015), which led to the suggestion that oxytocin might promote cognitive exploration of
10 a conceptual space (de Boer et al., 2017; De Dreu et al., 2014, 2015; Kapetaniou et al., 2021;
11 Ye et al., 2017). However, it remains unclear how this slow-acting neuromodulatory hormone
12 influences the electrophysiological activities implementing cognitive exploration, as often
13 required to achieve conceptual coordination during human communication.

14 Studies in human and non-human primates have explored electrophysiological correlates of
15 oxytocin administration, largely when other agents are perceived, monitored, or involved in
16 values-based decisions (de Bruijn et al., 2017; Festante et al., 2020, 2021; Geenen et al., 1988;
17 Levy et al., 2017; Levy, Goldstein, Influx, et al., 2016; Liu et al., 2013; Luo et al., 2017, 2019;
18 Mu et al., 2016, 2016; Perry et al., 2010; Ruissen & de Bruijn, 2015; Soriano et al., 2020; Wu
19 et al., 2013; Wynn et al., 2019; Ye et al., 2017; Zhuang et al., 2021). This study uses
20 magnetoencephalography (MEG) to explore electrophysiological correlates of how oxytocin

1 modulates human communicative actions, a fundamental dimension of human social
2 cognition largely ignored by previous studies of the oxytocinergic system. We exploit the fact
3 that human communicators adjust their referential signals to the presumed characteristics
4 and knowledge of the addressee of those signals ("recipient design"; Campisi & Özyürek,
5 2013; Clark & Murphy, 1982; Fussell & Krauss, 1989; Galati & Brennan, 2010; Levinson, 2006;
6 Newman-Norlund et al., 2009; Tomasello, 2008; Toni & Stolk, 2019; Yoon & Brown-Schmidt,
7 2014). Previous work has shown that oxytocin boosts participants' recipient design, driving
8 them to adjust their communicative behavior to the actual level of understanding
9 experienced in the addressees, and away from their expectations of the addressees' cognitive
10 abilities (de Boer et al., 2017). Following this hypothesis, we report a novel analysis of the
11 dynamics of recipient design adjustment produced by communicators, and investigate the
12 electrophysiological mechanisms supporting oxytocinergic effects on recipient design.

13 Previous studies have consistently shown the involvement of the temporal and ventral
14 prefrontal cortex in the right hemisphere during the production, comprehension, and
15 adjustment of communicative behaviors while adopting recipient design (Noordzij et al.,
16 2009, 2010; Stolk, Verhagen, et al., 2013; Stolk et al., 2015). Accordingly, we reasoned that, if
17 oxytocin modulates recipient design, then it is likely to happen at the level of the right-
18 hemispheric temporo-frontal upregulation of electrophysiological activity evoked by a
19 communicative challenge (Stolk, Verhagen, et al., 2013; Stolk et al., 2015). Furthermore, as
20 shown in a previous MEG study, selecting and understanding communicative actions evoked
21 matched broadband responses (30-250 Hz) in communicators and addressees (Stolk,

1 Verhagen, et al., 2013). In the last decade, accumulating evidence has pointed out that
2 broadband gamma electrophysiological activity is a mesoscopic correlate of action potentials
3 and dendritic currents in recurrent neural circuits, different in origin and functional
4 characteristics from narrowband oscillations (gamma: 60-80 Hz) evoked in superficial
5 cortical layers by feedforward processing of sensory stimuli (Bartoli et al., 2019; Bastos et al.,
6 2018; Buzsaki & Schomburg, 2015; Chaoul & Siegel, 2020; Donoghue et al., 2020; Leszczyński
7 et al., 2020; Manning et al., 2009; Miller et al., 2009, 2014; Pettersen et al., 2014; Ray et al.,
8 2008; Ray & Maunsell, 2011; Thivierge & Cisek, 2008; Touboul & Destexhe, 2010; Voytek et
9 al., 2015). Thus, in the current study, we carefully differentiated periodic and aperiodic
10 neural activities and tested their behavioral effects on communicative adjustment modulated
11 by oxytocin administration (Wen & Liu, 2016).

12 In sum, this study explored electrophysiological mechanisms supporting recipient design
13 adjustments, and the oxytocinergic modulation of those adjustments. After receiving
14 intranasal oxytocin or a placebo, electrophysiological activity was measured with MEG while
15 participants were engaged in live, non-verbal communicative interactions between a
16 communicator (the participant) and two presumed addressees (a confederate) (Tacit
17 Communication Game, Figure 1; de Boer et al., 2017; Newman-Norlund et al., 2009; Stolk et
18 al., 2015; Stolk, Hunnius, et al., 2013). We predicted that oxytocin regulates recipient design
19 through enhancement of broadband aperiodic neural responses in the right temporo-frontal
20 communication-related circuits.

1 **Methods**

2 ***Participants***

3 Fifty-eight male participants (mean $\pm$ SD = 22 $\pm$ 3 years), naïve to the experiment task, signed
4 informed consent according to the study protocol approved by the local ethics committee
5 (Committee on Research Involving Human Subjects, Region Arnhem-Nijmegen, the
6 Netherlands; study protocol registration number 37419.091.11). Assuming a medium-large
7 effect size (Cohen's d between .5 and .7) of oxytocin on communicative behavior, a sample
8 size between 26 and 49 in each of the two groups (oxytocin and placebo) was deemed to
9 provide adequate power (80%) to detect the presence of an effect of the intervention, at a
10 significance level of .05. The enrolled participants were asked to refrain from caffeine, alcohol
11 and/or cigarettes 24 hours prior to the experiment and from food or drink (except water) 2
12 hours prior to the experiment. Three participants were excluded during behavioral analysis
13 (One participant did not seem to understand the instructions of the task; one participant did
14 not believe our manipulation indexing communicative adjustment, one participant data
15 acquisition failed. see de Boer et al., 2017), and four other participants were excluded in the
16 current report due to lack of neuroimaging data (Two participants had no MRI recording, one
17 participant had no MEG recording and one participant had no head-location recording)
18 (*Supplementary 1*). 24 participants in the oxytocin group and 27 participants in the placebo
19 group remained in the final analysis.

Experimental Procedures and Pharmacological Application

The participants were randomly assigned to one of the two groups and double-blindly received self-administered intranasal sprays of either 24 IU (three puffs per nostril, each with 4 IU) oxytocin (Syntocinon, Novartis, Basel, Switzerland) or placebo (sodium chloride 8mg/ml and benzalkonium chloride .1mg/ml). The neuropeptide and saline solution were prepared and stored by the pharmacy of the Radboud University Medical Centre. After 45 minutes of the pharmacological administration, the participants sat into the MEG scanner, adjusted themselves to comfortable position, and then practiced for three trials to get familiar with the game-controller and the measuring environment. The MEG scanning preparation and experiment task lasted for about 30 minutes in total. Immediately afterwards, participants answered a questionnaire on the characteristics of the addressees (Newman-Norlund et al., 2009) and a 7-points Likert scale on their beliefs on the substance and dosage of the pharmacological treatment they had received. Furthermore, we verified that the two groups consisted of participants matched along study-relevant dimensions by measuring a number of physiological and psychological indexes (de Boer et al., 2017; Radke et al., 2017, *Supplementary 2*).

Experimental Task and Design

We used the TCG task to quantify communicative behavior between a communicator (a participant) and an addressee (the confederate) (de Ruiter et al., 2006, 2010; Newman-Norlund et al., 2009; Noordzij et al., 2009, 2010; Stolk, Verhagen, et al., 2013; Stolk, Hunnius,

et al., 2013; Stolk et al., 2015; de Boer et al., 2017). While sitting in separate rooms, each communicator-addressee pair jointly collected a target object (an acorn) from a location (one of the red-and-white circles) in a Target-Field (one of the nine grids) on a digital game board of 3-by-3 grids (Figure 1). Only the communicator was aware of the target's location, and only the addressee could collect the target. The communicator could move a bird token on the game board with a gamepad, in order to generate moving patterns that could be seen and interpreted by the addressee as signals for the target location. However, the bird token could only move to the center of each of the nine grids, and only through vertical or horizontal displacements (see Figure 1A, "2. MOVEMENT"). Such moving restrictions of the bird token required the communicator and the addressee to disambiguate one among multiple potential target locations within a grid, based on movements of the bird token. The addressee controlled a squirrel token with a computer mouse; and could move freely on the game board and had a direct access to any location when collecting the target (see Figure 1B, "4. MOVEMENT").

Figure 1. Time-course of a TCG trial. A) Communicator's view. **B)** Addressee's view. The TCG task required two players to collect targets (acorns) jointly from the nine-grid (fields) game board. Using MEG, we measured neural activity while participants played as communicators (in blue), following administration of either oxytocin or placebo. A confederate sat in a separate room and played the TCG as addressee (in green). Each trial consisted of six successive epochs illustrated with the time line of the figure. The trial starts with a blank game board visible to both players (3 secs, 0. BASELINE). Afterward, a bird appears on both player's screens, whereas the acorn is only visible on the communicator's screen. Meanwhile, the communicator plans how to solve the trial (A1. PLANNING). To this end, the communicator needs to indicate to the addressee the location of the acorn on the game board, and that can only be achieved by moving the bird. Namely, only the communicator can move the bird, vertically and horizontally, from the center of one field to the center of another adjacent field (A2. MOVEMENT). The bird cannot be overlaid on the red circle containing the acorn. Therefore, in those fields with multiple potential acorns' locations (red-and-white circles), the communicator needs to generate a moving pattern of the bird that is visible and understood by the addressee to disambiguate the relevant circle. In the example above, the communicator signals the relevant field to the addressee by holding the bird there for longer (Time on Target-Field) than the time spent on other fields of the board. Furthermore, the communicator would try to mark the relevant circle, e.g. by entering and exiting the field with the acorn from above, intersecting the relevant circle in the process. In the subsequent epoch, the bird disappeared and a squirrel become visible to both players, the communicator waits for the addressee's response (A3. WAIT). Only the addressee can move the squirrel, anywhere on the game board. After interpreting the referential motion pattern of the bird made by the communicator, the addressee indicates the inferred location of the acorn by moving the squirrel onto a circle. The communicator can see the movement of squirrel (A4. OBSERVATION). The trial ends with a feedback shown to both players (5. FEEDBACK).

1
2 Previous works have shown that, in this experimental setup, communicators spontaneously
3 generate communicatively specific adjustments towards younger presumed addressees, in
4 line with similar observations obtained during language use (Brand et al., 2002; Brodsky &
5 Edelman, n.d.; Grieser & Kuhl, 1988; Newman-Norlund et al., 2009; O'Neill et al., 2005; Stolk
6 et al., 2015; Stolk, Hunnius, et al., 2013; Warren-Leubecker & Bohannon, 1984). Building on
7 those observations, we informed the participants that they would complete the TCG task with
8 two addressees, an Adult and a Child, sitting in separate rooms with their own monitors to
9 watch the communicator's token moving on the game board. The task contained 50 trials that
10 were distributed into five Adult blocks and five Child blocks. Each block consisted of five
11 consecutive trials in which the communicator interacted with the same presumed addressee
12 (either Adult or Child). In reality, one confederate performed as addressee without knowing
13 the roles of Adult and Child, resulting in statistically matched addressee's performance
14 between Adult and Child (*Supplementary 2*). Therefore, the two types of blocks differed only
15 in terms of the communicator's expectations about the presumed addressees' abilities to
16 interpret the referential signals. Each participant randomly started with either an Adult or a
17 Child block, and then alternatively switched between the two types of blocks. Each
18 participant was informed about the addressee identity by a picture of adult or child, which
19 was displayed at the center of the screen for 5 seconds at the beginning of each block and in
20 the top-right of the screen throughout the block of five trials (Figure 2A).

Figure 2. Time-course of the experiment and quantification of communicative adjustment. **A)** Each communicator (participant) plays 50 trials, organized in ten blocks of five trials. A child and an adult alternated as presumed addressees between each block. In each block, the communicator (the participant) is informed about the identity of the presumed addressee by means of a photo shown at the beginning of each block and next to the game board throughout the trials. Unbeknownst to the communicator, a confederate played both presumed addressees without knowing the presumed addressee's identity. **B)** This experimental setup quantifies recipient design, with communicators spending longer time on the field containing the acorn (Time on Target-Field, see Figure 1) when they believe to be playing with the child than with the adult presumed addressee, despite matched performance of the confederate playing those two roles. We quantify recipient design with a dynamic index, Communicative Adjustment (CA), namely, a block-by-block Time on Target-Field difference, positive when the average Time on Target-Field towards a child addressee was longer than the corresponding average in the preceding block with an adult addressee and negative when the average Time on Target-Field decreased during Adult-to-Child block transitions. The inverse block transitions (Child-to-Adult) were indexed with an inverse CA sign.

Indexing Dynamics of Communicative Adjustment

This experiment builds on several studies showing that the time spent by communicators on a communicatively relevant referent provides a quantitative index of recipient design, in both laboratory and naturalistic contexts (Sacks et al., 1978; Newman-Norlund et al., 2009, 2009;

1 Noordzij et al., 2009, 2010; Stolk, Hunnius, et al., 2013; Stolk, Verhagen, et al., 2013; Stolk et
2 al., 2015; de Boer et al., 2017; Murillo Oosterwijk et al., 2017; Liu et al., 2019). Following
3 previous studies using this experimental setup, we labeled the time spent by communicators
4 on the relevant referent as Time on Target-Field, i.e. the time interval between the bird token
5 entering and leaving the Target-Field (Newman-Norlund et al., 2009; Stolk, Hunnius, et al.,
6 2013; Stolk, Verhagen, et al., 2013; Stolk et al., 2015; de Boer et al., 2017). In case the Target-
7 Field was visited multiple times within a trial, then we conservatively averaged the intervals
8 on Target-Field (Figure 1A, “2. MOVEMENT”). In a previous analysis of the same behavioral
9 data considered here, De Boer et al. (2017) quantified recipient design as the difference
10 between the time communicators spent on Target-Field when interacting with a presumed
11 Adult or Child addressee. In that study, it was shown that, in contrast to the placebo group,
12 participants receiving oxytocin did not differentiate between the two presumed Addressees
13 (as indexed by the Time on Target-Field averaged across trials). Here, differently from the
14 previous analysis, we focused on new indices of recipient design that capture the dynamics
15 of communicative adjustment towards the two presumed addressees. We considered a
16 previously ignored feature of the experimental design, namely the difference between the
17 Time on Target-Field of successive experimental blocks, when communicators interacted
18 with different presumed addressees (Figure 2A). Building on the a priori knowledge that
19 communicators spend longer Time on Target-Field when believing they are interacting with
20 a presumed Child addressee than an Adult addressee, we considered a lengthened Time on
21 Target-Field for a Child-addressee block compared to the previous Adult-addressee block as

positive communicative adjustment, and a shortened Time on Target-field for a Child-addressee block compared to the previous Adult-addressee block as negative communicative adjustment (Figure 2B). Formally, we defined Communicative Adjustment [CA(t); *eq. 1*] as the absolute local difference of averaged Time on Target-Field (avgT) between two successive blocks (block_{t+1} and block_t) multiplied by a step function D(t) indicating the direction of addressee transitions (“+1” for Adult-to-Child transitions and “-1” for Child-to-Adult transitions).

$$CA(t) = |avgT(t + 1) - avgT(t)| \times D(t) \quad (eq. 1)$$

$$where, \{t|t \leq 9, t \in \mathbb{Z}^+\} and D(t) = \begin{cases} +1 & \text{if transitioning from Adult to Child} \\ -1 & \text{if transitioning from Child to Adult} \end{cases}$$

We reasoned that the sensitivity of CA(t) to local differences in Time on Target-Field makes CA(t) potentially sensitive to other factors changing differently across blocks but not directly related to recipient design, e.g. procedural learning effects in producing movements of the bird token. We considered planning time as a conservative index of the block-by-block changes in behavior due to procedural learning effects, conservative because planning time might also be influenced by recipient design and communicative difficulty effects. Therefore, the Time on Target-Field data used in computing CA(t) were residualized against the time spent planning the motor response in the corresponding trials.

The overall statistical model used to quantify influences of oxytocin on recipient design as indexed by CA(t) was a 2 (Treatment: Oxytocin or Placebo) * 9 (block-wise Addressees Transition within each participant) mixed factorial design. Furthermore, we implemented a

1 number of control analyses. First, we tested whether the average CA(t) across block
2 transitions confirmed the findings obtained in de Boer et al., 2017, assessing the presence of
3 a recipient design effect in the placebo group but not in the oxytocin group with an
4 independent-samples t-test. Second, we tested whether the two experimental groups had
5 similar communicative adjustments at the onset of the experiment, i.e. whether the CA(t)
6 measured in the first block of the experiment showed the presence of a positive recipient
7 design effect in both placebo and oxytocin groups, using an independent-samples t-test.
8 Third, we tested whether CA(t) between blocks variability changed in the course of the
9 experiment differently for the oxytocin and the placebo groups. This hypothesis was assessed
10 by considering the within-subject CA(t) standard deviation during the first- and second-half
11 of the experiment (1st to 4th and 6th to 9th block transitions), in a 2 (Treatment: oxytocin,
12 placebo) $\times$ 2 (Time: first-half, second-half) mixed-factorial ANOVA. The statistical inferences
13 were conducted with *stats*, a build-in package of *R* (Bolar, 2019; R Core Team, 2009). Besides
14 the half-split within-subject variance of CA(t), we also quantified point-by-point changing
15 rate of CA(t) time course (see *Supplementary 5* for further analyses and results). Following a
16 previous study (Stolk et al., 2015), we formulated the post-error communicative adjustment
17 as the difference of Time on Target-field between the trial after an unsuccessful
18 communication and the preceding trial, normalized by the sum of Time on Target-field of the
19 two trials. We considered this parameter as an index of overall communicative motivation.

MEG and MRI Data Acquisition

Participants' electrical brain activity during task performance was recorded with a whole-head CTF system with 275 axial gradiometers (VSM Med Tech, 1200 Hz sampling rate, 300 Hz low-pass filter). The communicator's head position relative to the MEG sensor array was continuously monitored using localization coils attached to the anatomical landmarks, i.e., the nasion and left and right ear canals (Stolk, Todorovic, et al., 2013). High-resolution T1-weighted images were obtained with a single-shot MPRAGE sequence with a Siemens Avanto 1.5 T scanner (176 sagittal slices, TR = 1730 msec, TE = 2.95 msec, flip angle = 7°, voxel size = 1×1×1 mm³, FOV = 256×256 mm²). During anatomical scanning, the communicator wore a pair of vitamin-E ear capsules at the same locations of the MEG fiducial coils, allowing for co-registration of MRI images and MEG sensors for source reconstruction.

MEG data analysis

The MEG data were analyzed using the FieldTrip toolbox (Oostenveld et al., 2010) with customized MATLAB R2016b (MathWorks, Natick, MA, USA) analysis scripts.

Preprocessing. The raw MEG data were high-pass filtered (> 1 Hz) to remove low-frequency noise and segmented into trials, from 2 secs before presentation of the target stimuli until disappearance of the feedback stimuli (To avoid potential residual effect from the previous trial, we did not consider the first second at the beginning of each trial. Figure 1A). Each trial was demeaned to the time window of 0.2 secs right before stimulus-onset for controlling the leakage of non-zero DC component. A Discrete Fourier Transform (DFT) filter was applied to

1 minimize 50 Hz line-noise and its harmonics of 100, 150, 200 and 250 Hz.

2 *Source reconstruction and aperiodic spectral analysis.* For each participant, we estimated
3 aperiodic source power by combining a Partial Canonical Correlation adaptive beamformer
4 (PCC, Gross et al., 2001; Schoffelen et al., 2008) and Irregular Resampling Auto-Spectral
5 Analysis (IRASA, Wen & Liu, 2016). There were three major steps. First, we computed
6 normalized lead field matrices with participant-specific volume conductors and source
7 models (Schoffelen et al., 2008). More precisely, the individual anatomical MRIs were co-
8 registered to the CTF coordinates system and then segmented (with 5 mm smoothing and
9 0.5 threshold) for constructing volume conductors using a single-shell model of the brain
10 surface (Nolte, 2003). The source model was constructed by linearly transforming a 3D
11 template grid (with 10 mm resolution, leading to 2982 dipole locations inside the brain
12 compartment) in MNI coordinates into the participant-specific coordinates based on the
13 individual MRIs. The inverse of the transformation positioned the grid points across
14 participants at identical locations in normalized MNI space. Second, we reconstructed time
15 series of interest at each source dipole position. To this end, we first segmented the
16 preprocessed trials of data into 500 msec time series, and then computed the Cross-Spectral
17 Density (CSD) matrix between all MEG sensor pairs with Fourier transforms of the time
18 series between 0 and 300 Hz. With the balanced length of time series between the two
19 treatment groups, we computed spatial filters at each grid dipole location inside the brain
20 (as well as one location for each eye and one location in between, *Supplementary 3*) for each
21 frequency independently. The Fourier transformed MEG data at each location were then

1 passed through the corresponding spatial filter. The products were then projected onto the
2 dipole direction of maximal power, given the singular value decomposition (SVD) to the real
3 part of the CSD. In order to increase sensitivity towards state-related effects known to be
4 evoked by the communicative demands of the TCG task in temporal-prefrontal circuits (Stolk,
5 Verhagen, et al., 2013), we focus the analysis on the epoch from 2 secs before stimulus
6 presentation until the onset of the communicator response (Figure 1A). Thus, we
7 reconstructed the source-level time series of these epochs by inversely transforming the real
8 part of the estimated maximal dipole moment back to the time-domain. Third, we use IRASA
9 to differentiate aperiodic from periodic components in the reconstructed signals. This
10 method resamples the MEG signal in time, such that periodic components are symmetrically
11 redistributed beside its original location in the power spectra after Fourier transformation.
12 IRASA takes the median of the resample pairs as an estimate of the aperiodic component. We
13 avoided effects of large amplitude low-frequency signals on IRASA performance (Donoghue
14 et al., 2020) by focusing on the 30-150 Hz frequency range (Bartoli et al., 2019; Manning et
15 al., 2009; Ray et al., 2008; Ray & Maunsell, 2011), previously shown to be involved in
16 interpersonal referential communication (Stolk, Verhagen, et al., 2013).

17 *Statistical inference on MEG effects of oxytocin intervention.* We implemented three nested
18 analyses. First, we implemented a whole-brain analysis over 30-150 Hz averaged aperiodic
19 source power, contrasting the oxytocin and placebo groups at each dipole location
20 throughout the brain, making inferences corrected for multiple-comparisons using non-
21 parametric cluster-based permutation tests with cluster-forming threshold $\alpha = .05$, two-

tailed and 1000 Monte Carlo runs (Maris & Oostenveld, 2007). Effects of intra-subject variance of head translations and rotations, as well as effects of head size, were removed from the data through a multiple regression analysis considering four principle components that explained more than 98% variance in those confounds. We verified oxytocinergic effect on averaged broadband aperiodic power at the two local maxima identified in the first analysis, located in the anterior temporal cortex and in the ventral prefrontal cortex, after removing further potential confounds of eye-movements by regressing out estimates of those movements (*Supplementary 3*). Second, at each local maximum, we tested whether the oxytocin-related effect identified in the first analysis was a manifestation of broadband or band-limited increases in aperiodic power. Accordingly, we contrasted between-group differences in aperiodic activity over 103 frequency bins over the 30-150 Hz range through one-tailed independent-samples t tests ($\alpha = .05$ uncorrected for multi-comparisons to privilege sensitivity in this particular analysis). Third, we explored the task-related dynamics of the broadband oxytocin-related effect identified in the first analysis. At each local maximum as mentioned above, we extracted aperiodic power (averaged over the 30-150 Hz) for each experimental block in each participant, contrasting the effects between the oxytocin and placebo groups.

Statistical inference on brain-behavior relations. We examined whether, in the ventral prefrontal cluster showing a main effect of oxytocin intervention, the averaged broadband aperiodic power was related to the degree of Communicative Adjustment (CA) across participants. We used a linear mixed-effects model, with three fixed effects: the

pharmacological treatment (Treatment: Oxytocin or Placebo), aperiodic power averaged over 30 ~ 150 Hz (Neural Activity), and the interaction between the two factors. We included Participant as the grouping variable and considered three random effects: the random intercept of Participant, the random slope of Transitions (i.e. the block-wise Addressees Transition within each participant, see *Indexing Dynamics of Communicative Adjustment*), and their interaction; and potential confounding factors (PCs, see eq. 2).

$$CA \sim \text{Neural Activity} * \text{Treatment} + (1 + \text{Transition} | \text{Participant}) + \text{PCs} \quad (\text{eq. 2})$$

To verify the spatial and spectral specificity of the brain-behavior correlation identified in the ventral prefrontal cortex, we implemented post-hoc control analyses over rhythmic power from control regions in middle occipital cortex (8-12 Hz, Jensen & Mazaheri, 2010; Klimesch, 2012; Zhigalov & Jensen, 2020), posterior parietal cortex (8-12 Hz and 15-25 Hz, Brinkman et al., 2014; Haegens et al., 2011; Stolk et al., 2019), and ventral prefrontal cortex (30-60 Hz, Bartoli et al., 2019; Manning et al., 2009; Ray et al., 2008; Ray & Maunsell, 2011) (*Supplementary 4*). The model was fitted with *lme4* (Bates et al., 2015); and the post-hoc comparisons were conducted with *emmeans* (Lenth et al., 2021). The p-values were computed by t test with Kenward-Roger approximation for denominator degrees of freedom (Halekoh & Højsgaard, 2014) for degrees of freedom.

Results

Oxytocin Facilitates Updating of Recipient Design

Here, differently from de Boer et al. (2017), we investigated the temporal dynamics of how communicators adjusted their referential signals towards the presumed different addressees. First, we verified that the novel CA index used in this study, when averaged across addressees' transitions, reproduced one of the main findings reported in (de Boer et al., 2017). Namely, that study found that the placebo group spent longer time than the oxytocin group holding their token on the Target-field when they believed to be communicating with a Child than with an Adult Addressee. Accordingly, we found that communicators who received placebo intranasal sprays revealed a significantly greater CA averaged across all addressee transitions than those who received oxytocin ($t_{49} = 1.92$, $p = .031$, Cohen's $d = .54$; Figure 3A). The averaged CA was significantly higher than zero for placebo group (mean = 78 msec, $t_{26} = 2.21$, $p = .018$, Cohen's $d = .43$), but not for oxytocin group (mean = -13 msec, $t_{23} = -.42$, $p = .661$). Second, we verified that, in the first transition between addressee blocks in the experiment, the two experimental groups started the experiment with comparable CA(1) ($t_{49} = .05$, $p = .963$, Figure 3B). All communicators showed a positive communicative adjustment (CA(1): mean = 97 msec, $t_{50} = 2.21$, $p = .022$, Cohen's $d = .29$). These results confirm that, at the onset of the experiment, communicators put additional emphasis in their communicative signals when targeting addressees whom they believe to be less cognitively capable. One of the main findings of this study is that, over the course of the experiment, the oxytocin group produced a different CA(t) temporal profile than the placebo group (Figure 3C; see

Supplementary 5 for individual data). In the oxytocin group, CA(t) fluctuated around zero. In the placebo group, there was no clear convergence. Specifically, differently from the placebo group, communicators in the oxytocin group significantly reduced their block-by-block variability in communicative adjustments (Figure 3D; $F_{1,49} = 7.38$, $p = .009$, $\omega^2 = .04$). More precisely, the standard deviation of CA(t) in the oxytocin group dropped in the second-half of the experiment ($t_{23} = -3.43$, $p = .001$, Cohen's $d = .70$), whereas that of the placebo group remained at the same level across first- and second-half ($t_{26} = .20$, $p = .578$). These observations indicated that, in contrast to placebo group, the oxytocin group was stabilizing the level of CA(t) over the experiment (see Supplementary 5 for further analyses on changing rate of communicative adjustment).

Figure 3. Communicative adjustment behavior. **A)** Averaged over the whole experiment, the placebo group (in blue) showed a positive Communicative Adjustment (CA), larger than the near-zero CA of the oxytocin group (in red). The curved areas illustrate kernel density estimates of data distribution in each group. Each red dot or blue square is single participant data. The black dots represent the group mean, the black boxes indicate the interquartile range (IQR), and the whiskers are maximum and minimum with 1.5 IQR. **B)** For the first block transition, the two treatment groups had comparable positive CA values. **C)** The CA dynamics of the two groups. Filled red dots and blue squares for oxytocin and placebo groups respectively, represent the averaged CAs over each addressee transition. **D)** The within-subject variability in CA dynamics differed between the two groups, decreasing in the oxytocin group between the first and the second half of the experiment, but not in the placebo group. Error bars: standard errors of the mean. **: $p < .01$; *: $p < .05$; n.s.: not significant.

Moreover, we verified that the two treatment groups showed matched Time on Target-Field in the first block (Welch's $t_{35} = 1.92$, $p = .06$, Levene's test of variances equality: $F_1 = 5.85$, $p = .02$). Thus, the effects reported above could not be attributed to responding bias or heterogeneity of the initial level of recipient design of the communicators. The effects on the CA(t) could not attributed to the possibility that oxytocin reduced participants' communicative motivation, as oxytocin group revealed a positive post-error communicative adjustment, i.e. lengthen the Time on Target-field after an unsuccessful trial ($t_{23} = 2.64$, $p = .007$, Cohens' $d = .54$), which was significantly higher ($t_{49} = 1.94$, $p = .029$, Cohens' $d = .54$) than that of the placebo group showing no difference from zero ($t_{26} = .17$, $p = .433$). Further control analyses ruled out the possibility that the between-groups differences in CA(t) could be driven by confederate's reaction time, joint error rate, or communicator's total moving time, time on non-target-field and movement steps (*Supplementary 2*). As reported previously (de Boer 2017), we did not observe any between-group differences in a number of physiological and psychological indexes (e.g. cortisol, testosterone and social traits) or

beliefs about the psychopharmacological operation (*Supplementary 2*). Neither did we found evidence that the participants did not believe to be playing with a child and an adult partner (*Supplementary 2*). Taken together, these observations indicate that, over the course of the experiment, the oxytocin group converged in spending comparable time on the Target-Field for the two addressees and across subsequent blocks of trials. In contrast, the placebo group continued to spend more time on the Target-Field when addressing a child, with substantial between-blocks variability in performance even by the end of the experiment.

Oxytocin tonically upregulates aperiodic broadband power in the right ventral prefrontal cortex

Participants receiving oxytocin showed stronger broadband (30-150 Hz) aperiodic power in a right-lateralized cluster spanning ventral prefrontal cortex (vPFC) and anterior temporal (TP), with two corresponding local maxima (Oxytocin > Placebo at vPFC: $t_{49} = 2.41$, $p = 0.018$; TP: $t_{49} = 2.43$, $p = .024$); and weaker power than the placebo group in the right posterior parietal cortex (Oxytocin < Placebo at PPC: $t_{49} = -2.26$, $p = .028$, Figure 4A). The oxytocin-related effects on averaged aperiodic power in vPFC ($t_{49} = 2.22$, $p = .021$) and PPC ($t_{49} = -2.13$, $p = .031$), but not TP, persisted after regressing out individual variances of eye-movement noise, head position, and head size. Moreover, the oxytocin-related effects in vPFC and PPC could not be explained by narrowband increases in aperiodic power (extending from 44 to 150 Hz and from 30 to 150 Hz respectively, Figure 4B). Exploration of the task-related dynamics of the oxytocin-related vPFC effect indicated a sustained difference between

groups across the ten experimental blocks (minimum t : $t_{49} = 1.81$, $p = 0.043$, Figure 4B), but not in PPC (minimum t : $t_{49} = -1.54$, $p = 0.077$). In sum, these results indicate that exogenous oxytocin induced a tonic upregulation of aperiodic broadband power in ventral prefrontal cortex.

The upregulation of aperiodic broadband power supports oxytocinergic modulation of recipient design

We asked whether the magnitude of broadband aperiodic power in vPFC was related to the magnitude of communicative adjustment. Besides a main effect of broadband aperiodic power on CA ($t_{403.84} = 2.73$, $p = .007$, Estimate(SE) = .15(.06), 95% CI = [.04, .26], Figure 4C), and a supra-threshold effect of treatment ($t_{49.09} = 1.78$, $p = .081$, Estimate(SE) = .22(.06), 95% CI = [-.03, .46]), there was a significant interaction between treatment and broadband aperiodic power on CA ($t_{432.96} = -2.70$, $p = .007$, Estimate(SE) = -.28(.10), 95% CI = [-.48, -.08], Figure 4B). The interaction indicates that oxytocin modulated the effect of broadband aperiodic power on communicative adjustment. Stronger broadband aperiodic power in vPFC was associated with stronger communicative adjustments for those participants who received oxytocin intranasal spray ($t_{404} = 2.73$, $p = .007$, Estimate(SE) = .15(.06), 95% CI = [.04, .26]). This effect was not significant for those participants who received placebo ($t_{432} = -1.46$, $p = .146$, Estimate(SE) = -.13(.09), 95% CI = [-.30, .04]). Post-hoc control analyses verified the spatial and spectral specificity of the brain-behavior correlation identified in the ventral prefrontal cortex. We did not observe similar behavioral consequences when

1 considering low-gamma rhythmic activity (30-60 Hz) in vPFC and PPC, nor alpha (8-12 Hz)
2 or beta (15-25 Hz) rhythmic power in PPC, and alpha power in middle occipital cortex,
3 respectively (*Supplementary 4*). In sum, these results indicate that the tonic oxytocin-related
4 upregulation of aperiodic broadband power in ventral prefrontal cortex leads to behavioral
5 consequences, as indexed by a dynamic metric of recipient design (CA(t)).

6

Figure 4. Electrophysiological consequences of oxytocin administration during communicative performance. A) Spatial distribution of oxytocin-related changes in aperiodic broadband power (averaged over 30-150 Hz) with local maxima in right ventral prefrontal cortex (vPFC), temporal pole (TP) and posterior parietal cortex (PPC). B) Spectral (on the right) and temporal (on the left) profile of the vPFC effect (mean $\pm$ SEM, grey area indicates frequencies and time points where oxytocin and placebo groups are statistically different, $p < .05$ uncorrected for multiple comparisons over frequencies and time points). Insets show spectrally averaged effects and data distributions, using the same conventions as in Figure 3. C) Oxytocin-related changes in vPFC aperiodic broadband power account for

inter-participant variation in Communicative Adjustment. The upper grey panels indicate estimates of the fixed coefficients $\beta_{1\sim 3}$ relative to the effects specified in the legend. Error bars: 95% CI of the mean. ***: $p < .001$; **: $p < .01$; *: $p < .05$.

Discussion

Every-day human communication requires more than habitual responses to salient social stimuli (e.g. the face of an in-group member). Interlocutors accommodate to each other using recipient design (Clark & Murphy, 1982; Levinson, 2006; Tomasello, 2008). Oxytocin has been found to modulate recipient design by promoting cognitive exploration (de Boer et al., 2017; Ye et al., 2017). Here, we report behavioral and electrophysiological correlates of such oxytocinergic effect on human communicators. Behaviorally, oxytocin administration led participants to progressively adapt more effectively than the placebo group to the communicative behavior of the addressees. Electrophysiologically, oxytocin administration induced stronger broadband aperiodic power in the right ventral prefrontal cortex, pointing to increased synaptic activity (Bartoli et al., 2019; Leszczyński et al., 2020; Manning et al., 2009; Miller et al., 2014; Ray & Maunsell, 2011). Broadband aperiodic power in the ventral prefrontal cortex was positively correlated with the magnitude of communicative adjustment in the oxytocin group, but not in the placebo group. These findings qualify how oxytocin modulates recipient design, and suggest a neurophysiological mechanism supporting that modulation. Oxytocin adaptively regulates recipient design by boosting explorative adjustments during early stages of the interaction, then reducing those adjustments to match the actual demands of the communicative interaction. Correspondingly, oxytocin upregulates

state-related activity in a cortical region known to guide communicative behavior by updating knowledge about an interlocutor (Stolk et al., 2015).

Oxytocin Modulates Dynamics of Recipient Design

By using a new behavioral measure, Communicative Adjustment (CA(t)), we could quantify the fine-grained dynamics of recipient design adjustment to the child and adult presumed addressees. One of the main findings of this study is that oxytocin administration led to larger within-subject variation of CA(t) in the first half of the experiment, followed by a reduction of CA(t) variation in the second half of the experiment. This observation suggests that, in the initial stages of the communicative interaction, oxytocin increased exploration of different adjustments to the two presumed addressees, followed by a convergence of the communicative adjustments directed to those addressees. It is known that communicators spontaneously integrate prior knowledge about their interlocutor with the ongoing communicative experience (Brennan et al., 2010; Clark & Krych, 2004; Hawkins et al., 2021; Kuhlen et al., 2012; Kuhlen & Brennan, 2010; Zhang et al., 2021). The behavioral finding of this study illustrates how oxytocin enhances that integration and facilitates the implementation of the Gricean Maxim of Quantity, namely producing utterances that convey no more information than necessary (Blokpoel et al., 2012; Brennan & Clark, 1996; de Ruiter et al., 2010; Galati & Brennan, 2010; Garrod & Anderson, 1987; Grice, 1975; Levinson, 2000; Liu et al., 2019; Yoon & Brown-Schmidt, 2018). More generally, the finding supports the notion that oxytocin promotes exploration not only of other social agents (e.g. in-group

members), but also of the conceptual space supporting social interactions (de Boer et al., 2017; Ye et al., 2017).

Collateral findings from control analyses rule out a number of alternative interpretations. At the beginning of the experiment, participants from both oxytocin and placebo groups revealed a positive communicative adjustment, an indication that oxytocin did not modify participants' initial belief of interacting with addressees of differing communicative abilities. It could be argued that oxytocin reduced participants' prosocial motivation (De Dreu, 2012; De Dreu et al., 2011; Declerck et al., 2020; Hurlemann & Marsh, 2017; Luo et al., 2017; Muroy et al., 2016; Nave et al., 2015). That possibility is not compatible with the observation that the oxytocin group showed larger post-error communicative adjustments than the placebo group. Other control analyses rule out the possibility that the differential dynamics of communicative adjustments in the oxytocin and placebo groups resulted from between-group differences in social-cognitive traits (e.g. social anxiety and empathy), endogenous hormone level (cortisol and testosterone), or evaluation of the addressees' abilities. Furthermore, the effect of oxytocin was specific to communicative components of the participants' behavior, being absent from instrumental components of the movement (e.g. time on non-target-field, *Supplementary 2*).

Oxytocin Modulates State-related Ventral Prefrontal Activity

We observed that oxytocin induced persistently stronger aperiodic activity in vPFC, over a broad spectral (30-150 Hz) and temporal range (throughout all experimental blocks). It has

1 been argued that aperiodic power reflects action potentials of a neuronal ensemble,
2 contributing to frequencies all over the spectrum (broadband effect), possibly leading to
3 changes in cortical state (Bartoli et al., 2019; Bastos et al., 2018; Buzsaki & Schomburg, 2015;
4 Leszczyński et al., 2020; Manning et al., 2009; Miller et al., 2009, 2014; Pettersen et al., 2014;
5 Ray et al., 2008; Ray & Maunsell, 2011; Thivierge & Cisek, 2008; Voytek et al., 2015).
6 Furthermore, oxytocin administration in acute rat hippocampal slices leads to upregulation
7 of fast-spiking interneurons and increases fidelity of spike transmission (Owen et al., 2013).
8 This neurophysiological mechanism could increase signal-to-noise in neuronal ensembles
9 responsible for sensing and integrating social information (Gangopadhyay et al., 2021),
10 leading to increased broadband aperiodic power. The long-lasting oxytocinergic effect on
11 aperiodic power might be related to the relatively long half-life and slow degradation of this
12 neuropeptide (Bethlehem et al., 2013; Ludwig & Leng, 2006), although it remains to be seen
13 whether similar effects can be evoked by endogenous oxytocin release triggering both
14 synaptic and non-synaptic events, such as dendritic calcium spikes that profoundly
15 contributes to the MEG signal (Brown et al., 2020; Buzsáki et al., 2012; Ludwig & Leng, 2006).
16 It is known that lesion of the medial aspect of vPFC impairs recipient design, as indexed by
17 communicators' ability to tune their communicative actions to prior knowledge and ongoing
18 behaviors of an addressee (Stolk et al., 2015). Furthermore, vPFC is known to support mental
19 state inference and trait attribution (Frith & Frith, 2006; Frith & Frith, 2003; Gallotti & Frith,
20 2013; Van Overwalle, 2011; Van Overwalle & Baetens, 2009), as well as to construct and
21 update associations between stimuli and (social) contextual frames (Bertossi et al., 2016;

1 Krueger et al., 2009; Spalding et al., 2015; Zink et al., 2008). Here, we extend that knowledge
2 by showing that the contribution of vPFC to recipient design is proportional to the
3 oxytocinergic upregulation of broadband aperiodic activity. Accordingly, the current finding
4 emphasizes the importance of oxytocinergic tone in vPFC for dynamically integrating prior
5 knowledge with on-going communicative experience. Given the presence of oxytocin
6 receptors in the post-orbital gyrus in human brain (Boccia et al., 2013), it is possible that
7 oxytocin regulates recipient design via a direct impact on vPFC neural activity. However,
8 oxytocin effects are also mediated through other neurotransmitter systems, such as the
9 dopaminergic and the serotonergic systems, influencing several prefrontal-amygdala
10 circuits (Froemke & Young, 2021; Gangopadhyay et al., 2021; Love, 2014; Ma et al., 2016;
11 Martins et al., 2021; Quintana et al., 2019; Sripada et al., 2013; Striepens et al., 2014). Future
12 studies might differentiate these potential mechanisms with high-precision M/EEG (Seeber
13 et al., 2019; Tzovara et al., 2019) and newly developed algorithms for source reconstruction
14 (Krishnaswamy et al., 2017; Pizzo et al., 2019) that are able to disentangle activities from
15 deeper limbic structures.

16 It is known that the vPFC region is particularly vulnerable to non-neural contributions to the
17 MEG signal (Carl et al., 2012). It might be argued that the between-groups differences in MEG
18 signal are partially due to systematic between-groups differences in participants' head size,
19 position, or motion during the task. However, there were no major systematic differences in
20 those parameters between groups, and the analytical procedures removed contributions of
21 those parameters from the neural signals (*Supplementary 3*). Furthermore, despite the lack

1 of gaze-related data due to testing time constraints, we estimated MEG consequences of eye
2 movements by source-reconstructing ocular muscles signals (*Supplementary 3*) and removed
3 it from inter-subject variance with linear regression. This approach offered a relatively
4 stringent control against eye movements confounds, at the cost of reduced sensitivity to task-
5 related effects. For instance, this control analysis largely removed the oxytocin-related effect
6 on aperiodic power observed in the right temporal pole, leaving only an effect at high
7 frequency (>120 Hz, *Supplementary 3*).

8 The MEG analyses focused on effects spanning several seconds, from two seconds before trial
9 onset until the start of the communicator's response. It might be argued that this choice
10 misses the opportunity to take advantage of the high temporal resolution of MEG. In fact, this
11 study uses the temporal resolution of MEG to estimate the spectral distribution of neural
12 effects evoked by the experimental interventions. We chose to extract those estimates from
13 a relatively long temporal window because oxytocin administration and referential
14 communication are both known to evoke state-related effects (Bethlehem et al., 2013;
15 Ludwig & Leng, 2006; Stolk, Verhagen, et al., 2013). However, to ensure a fair comparison
16 between the oxytocin and placebo groups, the spatial filter for source reconstruction was
17 computed using matched lengths of data based on all trial epochs (*Supplementary 3*).

18 **Conclusion**

19 Our findings indicate that oxytocin leads to faster recipient design, pushing participants to
20 consider recent communicative history and downweight their expectations about the

1 abilities of a child addressee. The finding support the notion that oxytocin promotes
2 exploration of the conceptual space supporting communicative interactions (de Boer et al.,
3 2017; Ye et al., 2017). This study opens the way to test whether the state-related
4 oxytocinergic upregulation of vPFC aperiodic power provides a general mechanism
5 supporting communication during multimodal linguistic face-to-face interactions (Holler &
6 Levinson, 2019; Levinson, 2006).

7

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Supplementary 1: Recruitment and data quality control

The recruitment rate prevented us from completing the full planned sample (40 in each group) given the time and resources available for data collection of this study (Figure S1.). Eighty-nine participants in total were assessed for eligibility, 31 out of them were excluded in the enrolment phase for not meeting the inclusion criteria or no suitable time slots for testing. All the participants matched the following criteria: 1) aged between 18 and 35; 2) right-handed; and had 3) no metal in the body; 4) no claustrophobia; 5) no psychiatric, endocrine, or neurological disorders; 6) no use of medicine (except for normal use of paracetamol), experience in other pharmacological studies or blood donation within the last two months; 7) no use of drugs, alcohol or more than five cigarettes per day; and 8) no working at night (> 3 days per month) or intercontinental flights. During analysis phase, three participants were excluded in the behavioral analysis (one participant did not seem to understand the instructions of the task; one participant did not believe our manipulation indexing communicative adjustment, one participant data acquisition failed.) (de Boer et al., 2017). Four other participants were excluded in the current report due to lack of neuroimaging data (Two participants had no MRI recording, one participant had no MEG recording and one participant had no head-location recording). 24 participants in the oxytocin group and 27 participants in the placebo group remained in the final analysis.

Figure S1. Consort flow diagram.

Supplementary 2: Control analyses on behavioral, physiological, and psychological confounding factors

Homogeneity between oxytocin and placebo groups with respect to the levels of steroid hormones, social traits, and beliefs about the psychopharmacological manipulation.

First, to measure cortisol and testosterone levels, we collected salivary samples for three times (i.e. before, 15 minutes after, and 100 minutes after pharmacological administration, with salicaps, Immuno-Biological Laboratories GmbH, Hamburg, Germany). We considered the concentration for each hormone averaged the three time points. Specifically, the samples were stored at -25° and analyzed in duplicate for both cortisol and testosterone. Hormone concentration was measured using luminescence Immunoassays (Immuno-Biological Laboratories GmbH, Hamburg, Germany). The intra-assay and inter-assay coefficients of variation were less than 4% for cortisol, and 0-2% and 5-9% for testosterone respectively.

Second, immediately after the pharmacological administration, we gave participants a list of self-report questionnaires for measuring empathy (Empathy Quotient; Baron-Cohen & Wheelwright, 2004; Interpersonal Reactivity Index; Davis, 1983), systemizing abilities (Revised Systemizing Quotient; Baron-Cohen et al., 2003), social anxiety (Liebowitz Social Anxiety Scale; Liebowitz, 1987) and need for cognition traits (Need for Cognition Scale; Cacioppo et al., 1984). Third, after completing the TCG task, the participants also rated on a 7-points Likert scale on their beliefs on the substance and dosage of the pharmacological

treatment they had received. The oxytocin and placebo groups revealed no statistical difference in the array of independent-sampled t tests (Table S1). These results verified the between-group homogeneity in the levels of steroid hormones, social traits, and beliefs about the psychopharmacological manipulation.

Table S1. The oxytocin and placebo groups were matched in social traits, hormone concentration, and beliefs about the psychopharmacological manipulation.

Indexes	mean $\pm$ SD		df	t	p
	Oxytocin	Placebo			
Empathy Quotient	33.52 $\pm$ 11.59	36.93 $\pm$ 12.47	49	-1.000	.322
Systemizing Quotient	56.83 $\pm$ 14.37	51.82 $\pm$ 14.81	49	1.225	.226
Need for Cognition Scale	8.583 $\pm$ 9.036	10.78 $\pm$ 8.05	49	-.917	.363
Liebowitz Social Anxiety Scale	30.50 $\pm$ 16.76	28.56 $\pm$ 19.10	49	.384	.703
Interpersonal Reactivity Index:					
Fantasy subscale	16.96 $\pm$ 4.39	16.52 $\pm$ 4.19	49	.366	.716
Empathic concern	15.08 $\pm$ 4.31	15.63 $\pm$ 3.41		-.504	.616
Perspective taking	15.75 $\pm$ 3.53	17.04 $\pm$ 4.87	49	-1.068	.291
Personal distress	11.21 $\pm$ 5.03	10.19 $\pm$ 3.25	49	.873	.387
Cortisol (nmol/l)	8.44 $\pm$ 3.74	9.11 $\pm$ 4.25	49	-.594	.555
Testosterone (pg/nl)	79.25 $\pm$ 42.48	97.32 $\pm$ 87.31	49	-.921	.362
Belief on the type of administration (-3 = placebo, 0 = not sure, 3 = drug)	-.13 $\pm$ 1.80	-.18 $\pm$ 1.41	49	.093	.927
Belief on the dosage of administration (-3 = low, 0 = not sure, 3 = high)	-.75 $\pm$ 1.60	-.67 $\pm$ 1.47	49	-.194	.847

Balanced confederate's response and communicator's instrumental behaviors across experimental conditions.

A series of mixed-factorial ANOVAs showed that the task manipulation did not exert systemic

influence on the total moving time, time on non-target-field and the number of movement steps made by the participants (Table S2). The results indicated that participants specifically adjusted their communicative behavior but not instrumental responding patterns.

We verified that the role-blind confederate provided comparable reaction times and joint correctness of performance in the Adult and Child conditions for the oxytocin and placebo groups, also by conducting mixed-factorial ANOVAs (Table S2). Moreover, we also tested whether the confederate's reaction time and joint error rate predicted Communicative Adjustment (CA). Specifically, we compared the null model with Treatment (i.e. model_0: CA ~ Treatment) with two alternative models: 1) Treatment, confederate's Reaction Time, and their interaction (i.e. model_1: CA ~ Treatment * Reaction Time), and 2) Treatment, Correct rate, and their interaction (i.e. model_2: CA ~ Treatment * Error). The results revealed that adding the extra predictor to Treatment did not help explaining residual variance in CA (model_0 vs. model_1: $F_2 = .31$, $p = .73$; model_0 vs. model_2: $F_2 = 1.44$, $p = .25$). Together, these results suggested that the confederate's response were matched across experimental conditions and unlikely to account for CA effects.

Table S2. Confederate's response was matched across conditions and indexes of task performance other than Communicative Adjustment were unaffected by experimental manipulation.

Indexes	Effects	df	F	p
Moving Time	Addressee	1/49	.990	.325
	Treatment	1/49	.058	.811
	Addressee*Treatment	1/49	.211	.648
Time on	Addressee	1/49	1.545	.220

Nontarget-Field	Treatment	1/49	3.335	.074
	Addressee*Treatment	1/49	.106	.746
Number of movement steps	Addressee	1/49	.286	.597
	Treatment	1/49	.059	.809
	Addressee*Treatment	1/49	1.160	.287
Reaction time of confederate	Addressee	1/49	.385	.538
	Treatment	1/49	< .001	.978
	Addressee*Treatment	1/49	1.297	.260
Error rate	Addressee	1/49	.221	.641
	Treatment	1/49	1.939	.170
	Addressee*Treatment	1/49	.221	.641

We gave participants a questionnaire on the characteristics of the presumed addressees right after the TCG task. We performed mixed-factorial ANOVAs on the continuous dependent variables (Table S3-A), contingent-table tests on the categorical measures without involving the within-subject factor (Table S3-B), and mixed-effect binomial logit regressions on the categorical measures involving both between-subject factor (Treatment: Oxytocin or Placebo) and within-subject factor (Addressee: Child and Adult) (Table S3-C). The mixed-effect binomial logit models included Treatment, Addressee and their interaction as fixed effects, coupled with the random intercept of Participant, the random slope of Transitions and their interaction for the maximal random structure (*eq. S1*). The p-values were computed by F test with Satterthwaite approximation for denominator degrees of freedom for unbalanced data. The model was fitted with the GLMM module IBM SPSS Statistics 25.0. In sum, no matter what treatment they took, participants of both groups estimated that the adult presumed addressee were much older (mean $\pm$ SD = 29.57 $\pm$ 5.95) than the child presumed addressee

(mean $\pm$ SD = 8.32 $\pm$ 2.07); thought that the former was more alike to themselves; and assigned more intellectual attributes to the adult presumed addressee as well. These results further verified that the two addressee conditions differed only in terms of the communicator's expectations about cognitive abilities of the partners to interpret the communicative signals.

$$DV \sim Treatment * Addressee + (1 + Addressee|Participant) \quad (eq. S1)$$

Table S3. Oxytocin administration did not bias participants' beliefs about ones' own responses, nor their evaluation about attributes of the presumed addressees.

A)

Indexes	Effects	df	F	p	ω^2
How old do you think your partner is?	Addressee	1/49	684.339	< .001***	.851
	Treatment	1/49	.463	.500	
	Addressee*Treatment	1/49	.240	.627	
How alike to you do you think your partner is?	Addressee	1/49	8.985	.004**	.069
	Treatment	1/49	2.167	.147	
	Addressee*Treatment	1/49	3.449	.069	

B)

Indexes	df	χ^2 (corrected)	p
Did you play differently for one of the partners?	2	.184	.912
Who do you think played better?	1	< .0001	1.000

C)

Indexes	Effects	df	F	p	Odds ratio
Used a computer before?	Addressee	1/98	.001	.975	
	Treatment	1/98	.001	.975	
	Addressee*Treatment	1/98	.356	.552	

Rides a bike?	Addressee	1/98	.066	.798	
	Treatment	1/98	.839	.362	
	Addressee*Treatment	1/98	.066	.798	
Counts to 100?	Addressee	1/98	1.303	.257	
	Treatment	1/98	.003	.958	
	Addressee*Treatment	1/98	.003	.958	
Can do multiplication problems?	Addressee	1/98	10.918	.001**	42.217
	Treatment	1/98	.752	.388	
	Addressee*Treatment	1/98	.752	.388	
Knows the rules for driving a car?	Addressee	1/0	-	-	Inf
	Treatment	1/0	-	-	
	Addressee*Treatment	1/0	-	-	
Owns stuffed toys/animals?	Addressee	1/98	28.426	<.001***	.010
	Treatment	1/98	.196	.659	
	Addressee*Treatment	1/98	.145	.704	
Plays video games?	Addressee	1/98	10.783	.001**	.059
	Treatment	1/98	.010	.919	
	Addressee*Treatment	1/98	.010	.919	
Goes to the store on their own?	Addressee	1/98	24.139	<.001***	79.938
	Treatment	1/98	<.001	.990	
	Addressee*Treatment	1/98	<.001	.990	
Has read the diary of Anne Frank?	Addressee	1/98	18.651	<.001***	42.217
	Treatment	1/98	.147	.702	
	Addressee*Treatment	1/98	.207	.650	
Can sing the song "zakdoekje leggen"?	Addressee	1/98	.167	.683	
	Treatment	1/98	.165	.685	
	Addressee*Treatment	1/98	3.020	.085	

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Supplementary 3: MEG data quality

We performed a series of independent-samples t tests on potential MEG confounds. Results are presented in Table S4. First, we verified the homogeneity between oxytocin and placebo groups in averaged head position and variation (movement displacement and rotation along x-, y- and z-axes, Figure S2A) in the MEG scanner. Second, we conducted beamforming source reconstruction with balanced epoch lengths between the two treatment groups. Third, we estimated eye-movement induced noise in the MEG signal by considering three dipoles placed on right and left inferior ocular muscles, and another one between the two eyes (Figure S2B). Figure S2C illustrates the spatial distribution of correlations between each of the eye-related dipoles and the MEG sensors. The spatial patterns suggest that the eye-related dipoles captured variance in the MEG signal related to eye-movements.

Figure S2. Control analyses of head position and eye-movement noise. **A)** Trial-by-trial head position of each participant. The red and blue ellipsoids represent average 95% CI of head position variance of oxytocin and placebo group, respectively. **B)** The source model that was used for beamforming. We included three dipoles outside the brain, i.e. two on right and left inferior ocular muscles based on the MNI coordinates in the plot and another one in the middle. **C)** Correlation maps between the estimated source activities of the three dipoles and signals of each MEG sensor. The panels represent correlation maps of four randomly selected participants (two from each treatment group). The r values below each topography indicates the range of correlation coefficients.

Table S4. Control analyses of head position and eye-movement noise.

Indexes	mean $\pm$ SD		df	t	p
	Oxytocin	Placebo			

Average position	2.58 ± .09	2.58 ± .09	49	.24	.812
3D variance	1.5e -5 ± 4.4e -5	2.9e -6 ± 5.2e -6	49	-1.37	.178
Displacement					
X	.13 ± .05	.13 ± .05	49	.40	.693
Y	.15 ± .04	.14 ± .05	49	-.85	.399
Z	-2.57 ± .09	-2.58 ± .09	49	-.25	.802
Rotation			49	.32	.749
X	93.06 ± 29.76	95.94 ± 33.68			
Y	100.04 ± 44.30	81.87 ± 37.97	49	-1.58	.121
Z	57.24 ± 27.13	56.67 ± 35.50	49	-.06	.950
Length of epochs for computing beamformer	3.88 ± .95	3.85 ± 1.05	49	-.12	.902
Estimated eye-movement noise	1.7e-28 ± 4.3e-29	1.6e-28 ± 5.1e-29	49	-1.12	.267

Supplementary 4: Correlation between Communicative Adjustment and Neural Activities in Control Brain Regions

We also tested the social saliency hypothesis of oxytocin, by correlating the CA with a biological marker of attentional gating, i.e. inhibition of alpha activity in middle occipital cortex ($t_{48.29} = 1.81$, $p = .076$, Estimate(SE) = .22(.12), 95% CI = [-.02, .46]). We predicted that oxytocin might increase the alpha power in the visual cortex and/or modulate the neural-behavioral relationship in a way that a lower alpha power might enhance communicative adjustment behavior. However, we did not observe main effect of treatment ($t_{410.70} = 2.41$, $p = .017$, Estimate(SE) = .23(.10), 95% CI = [.04, .42]). Moreover, though the model revealed a significant interaction effect between treatment and the alpha activity in middle occipital lobe on CA, the post-hoc test showed no significant correlation between the alpha activity and CA for the oxytocin group ($t_{405} = -.91$, $p = .36$, Estimate(SE) = -.06 (.06), 95% CI = [-.18, .07]). For the placebo group, stronger inhibition of alpha power actually decreased the level of CA ($t_{401} = 2.41$, $p = .017$, Estimate(SE) = .17(.07), 95% CI = [.03, .31]). We reasoned that the communicative adjustment behavior is very likely involving complex feedforward and feedback control processes, so the relationship between visual alpha may not be that straightforward as we postulated.

Figure S4. Electrophysiological consequences of oxytocin administration during communicative performance. **A)** Spatial distribution of oxytocin-related changes in aperiodic broadband power (averaged over 30-150 Hz) with local maxima in right ventral prefrontal cortex (vPFC), temporal pole (TP) and posterior parietal cortex (PPC); and middle occipital lobe as a control region. **B)** Spectral effects extracted from TP and PPC local maxima (mean $\pm$ SEM, grey area at frequencies and time points where oxytocin and placebo groups are statistically different, $p < .05$ uncorrected for multiple comparisons over frequencies and time points). Insets show spectrally averaged effects and data distributions, using the same conventions as in Figure 3. **C)** Oxytocin-related changes in aperiodic broadband power in the

PPC local maximum did not account for inter-participant variation in Communicative Adjustment. The grey panels indicate estimates of the fixed coefficients β_{1-3} relative to the effects specified in the legend. Error bars: 95% CI of the mean. ***: $p < .001$; *: $p < .05$.

Table S5. Fixed effects of neural activity and treatment on Communicative Adjustment (CA).

Neural property	Effects	Coef	SE	95% CI	t	df	p
vPFC (aperiodic broadband)	Activity	.15	.06	[.04, .26]	2.73	403.84	.007**
	Treatment	.22	.12	[.03, .46]	1.78	49.09	.081
	Activity*Treatment	-.28	.10	[-.48, -.08]	-2.70	432.96	.007**
vPFC (periodic gamma)	Activity	-.01	.06	[-.13, .11]	-.15	449.58	.883
	Treatment	.21	.12	[-.03, .46]	1.75	48.36	.087
	Activity*Treatment	-.06	.09	[-.25, .12]	-.68	445.33	.497
PPC (aperiodic broadband)	Activity	-.01	.08	[-.16, .14]	-.15	282.28	.885
	Treatment	.21	.12	[-.03, .46]	1.74	48.08	.088
	Activity*Treatment	.11	.10	[-.09, .31]	1.05	359.08	.296
PPC (periodic alpha)	Activity	-.00	.07	[-.13, .13]	-.05	433.56	.962
	Treatment	.21	.12	[-.03, .46]	1.76	48.46	.085
	Activity*Treatment	.06	.09	[-.12, .25]	.68	450.15	.494
PPC (periodic beta)	Activity	-.04	.07	[-.18, .09]	-.62	430.95	.537
	Treatment	.21	.12	[-.04, .46]	1.70	48.38	.096
	Activity*Treatment	.16	.10	[-.03, .35]	1.66	437.65	.097
OC (aperiodic alpha)	Activity	-.06	.06	[-.18, .07]	-.91	404.52	.361
	Treatment	.22	.12	[-.02, .46]	1.81	48.29	.076
	Activity*Treatment	.23	.10	[.04, .42]	2.41	410.70	.017*
OC (mixed alpha)	Activity	-.06	.06	[-.18, .07]	-.92	423.80	.360
	Treatment	.22	.12	[-.03, .46]	1.80	48.30	.078
	Activity*Treatment	.17	.10	[-.02, .36]	1.81	418.34	.072

Supplementary 5: Changing Rate of Communicative Adjustment and Correlation with Aperiodic Power in vPFC

Changing Rate of Communicative Adjustment (CRCA)

As the within-subject variance of Communicative Adjustment (CA) differed in the first- vs. the second-half of experiment (Figure S5A showed group averaged and each participant's CA time course), that oxytocin administration might facilitate learning the actual communicative performance of the presumed addressees. A possible consequence of oxytocin-related improved learning is that the oxytocin group became more certain about their communicative adjustment. This should result in greater consistency in CA time courses in the oxytocin than in the placebo group. Here, we tested that hypothesis by quantifying the Changing Rate of Communicative Adjustment (CRCA), computed as the gradient norm of CA(t) with respect to the number of addressee transitions t (eq. S2).

$$CRCA = \|\nabla CA(t)\| \quad (\{t \mid t \leq 9, t \in \mathbb{Z}^+\}) \quad (eq. S2)$$

The norm of the gradient indicates the greatest change of CA(t) at time point t . A smaller CRCA reflects stabilization of the CA(t) over addressee transitions, i.e. convergence of the CA(t) time course. Therefore, we predicted a steeper descent of CRCA in the oxytocin group than in the placebo group.

CRCA allowed us to further probe the question whether oxytocin made communicators more efficient in converging to a certain level of CA, suggesting more certainty in their prediction

about the addressees' response. We used a linear mixed-effects model to account for within subject variances, with three fixed effects: the pharmacological treatment (Treatment: Oxytocin, Placebo), transitions between addressees (Transition: nine addressee transitions), and the interaction between Treatment and Transition. We included Participant as the grouping variable and considered three random effects: the random intercept of communicators, the random slope of Transitions, and their interaction (eq. S3).

$$CRCA \sim Treatment * Transition + (1 + Transition|Participant) \quad (eq. S3)$$

Oxytocin group showed a faster converged time course of communicative adjustment than the placebo group.

First, we observed that Transition revealed a significant main effect ($t_{49} = -3.95$, $p < .001$, Estimate(SE) = $-.11(.03)$, 95% CI = $[-.16, -.05]$). Specifically, CRCA descended significantly as a function of Transition in oxytocin (Estimate(SE) = $-.11(.03)$, 95% CI = $[-.16, -.05]$) but not in placebo (Estimate(SE) = $-.01(.03)$, 95% CI = $[-.06, .04]$) groups. The results suggested that both groups gained information from the presumed addressees' responses during communication and made use of such information to update their adjustment behavior. Critically, the model revealed a significant interaction effect ($t_{49} = 2.67$, $p = .01$, Estimate(SE) = $.10(.04)$, 95% CI = $[.02, .18]$, Figure S2B). Consistent with our hypothesis, the oxytocin group showed a faster descent of CRCA over time than the placebo group did ($t_{49} = -2.67$, $p = .01$, Estimate(SE) = $-.10(.04)$). These results indicated that oxytocin facilitated convergence

1 towards a more efficient recipient design and developed a higher confidence in their
2 predictions of addressees' responses during communication. These results together
3 suggested that participant, who took oxytocin intranasal sprays, went through different
4 exploration for the optimal level of CA for the presumed addressees. The more certainty in
5 their prediction, the more likely communicators sustain the current level of CA, thus, smaller
6 CRCA. We expected that oxytocin promoted recipient design by fastening the exploration
7 process, leading to greater reduction of CRCA over time.

8
9 *The oxytocin-related upregulation of aperiodic broadband power in ventral prefrontal cortex*
10 *modulates convergence of communicative adjustment*

11 We extracted the random slope per participant from the analysis above, as an index of
12 convergence speed of CA time course, indicating how fast a communicator achieve a stable
13 level of communicative adjustment. Further, we examined the relationship between the
14 magnitude of broadband aperiodic power in vPFC and convergence speed of CA. The results
15 showed a significant main effect of broadband aperiodic power on the random slopes ($t_{402.20}$
16 $= -3.61$, $p < .001$, Estimate(SE) = $-.18(.06)$, 95% CI = $[-.28, -.08]$) and an interaction between
17 treatment and broadband aperiodic power on CA ($t_{429.79} = 2.50$, $p = .013$, Estimate(SE)
18 $= .24(.10)$, 95% CI = $[.05, .43]$, Figure S5C). Specifically, the interaction indicates that oxytocin
19 modulated the effect of broadband aperiodic power on the temporal dynamics of
20 communicative adjustment; namely, a larger broadband aperiodic power in vPFC predicted
21 faster convergence of the communicative adjustment time course (i.e. a steeper random

1 slope) in the oxytocin group ($t_{402} = -3.61$, $p < .001$, Estimate(SE) = $-.18(.05)$, 95% CI = $[-.28,$
2 $-.08]$). However, no significant correlation was observed in the placebo group ($t_{436} = .69$, p
3 $= .494$, Estimate(SE) = $.06(.08)$, 95% CI = $[-.10, .21]$). Moreover, we did not find association
4 between CRCA and neural activities in other brain regions and spectral domains as shown in
5 Table S6. In sum, these results indicate that the oxytocinergic upregulation of aperiodic
6 broadband power in ventral prefrontal cortex promotes a more efficient recipient design
7 during the course of communication, rather than stereotypically increase communicative
8 adjustment based on their initial prediction about the interacting partner.

Figure S5. Dynamics of communicative adjustment and its correlation with aperiodic neural activity in vPFC. **A)** The Communicative Adjustment (CA) dynamics. Each thinner line represents the CA time course per participant who was assigned to oxytocin (in red) or placebo (in blue) treatment group. The thicker lines represent group averaged CA. **B)** The oxytocin group showed steeper slope of Changing Rate of Communicative Adjustment (CRCA) over time, suggesting a faster stabilization of CA in the oxytocin group than the placebo group. The thinner lines represent predicted CRCA per participant by the mixed-effect model; the thicker lines represent predicted group averages of CRCA. The grey panel indicates the fixed coefficients $\beta_1 \sim 3$ relative to the effects specified in the legend. Error bars: 95% CI of the predicted mean. **C)** Oxytocin-related changes in aperiodic broadband power in the vPFC local maximum account for inter-participant variation in CRCA. ***, $p < .001$; *, $p < .05$.

1 **Table S6. Fixed effects of neural activity and treatment on Changing Rate of**
2 **Communicative Adjustment (CRCA).**

Neural property	Effects	Coef	SE	95% CI	t	df	p
vPFC (aperiodic broadband)	Activity	-.18	.05	[-.28, -.08]	-3.61	402.20	<.001***
	Treatment	.23	.15	[-.07, .53]	1.56	48.92	.125
	Activity*Treatment	.24	.10	[-.05, .43]	2.50	429.79	.013*
vPFC (periodic gamma)	Activity	-.06	.06	[-.18, .05]	-1.08	431.62	.281
	Treatment	.21	.16	[-.10, .53]	1.36	48.88	.180
	Activity*Treatment	.08	.09	[-.10, .25]	.87	436.03	.384
PPC (aperiodic broadband)	Activity	-.02	.07	[-.17, .12]	-.34	306.82	.733
	Treatment	.21	.15	[-.10, .52]	1.37	48.60	.177
	Activity*Treatment	.06	.10	[-.13, .25]	.63	417.25	.530
PPC (periodic alpha)	Activity	.03	.06	[-.09, .16]	.56	422.72	.575
	Treatment	.21	.15	[-.09, .52]	1.40	48.91	.168
	Activity*Treatment	-.03	.09	[-.20, .14]	-.32	431.93	.750
PPC (periodic beta)	Activity	-.02	.06	[-.14, .11]	-.48	431.91	.812
	Treatment	.21	.15	[-.10, .52]	1.39	48.63	.172
	Activity*Treatment	-.01	.09	[-.18, .17]	-.07	434.63	.947
OC (periodic alpha)	Activity	.07	.06	[-.04, .19]	1.25	390.09	.211
	Treatment	.20	.15	[-.10, .51]	1.34	48.53	.187
	Activity*Treatment	-.08	.09	[-.25, .10]	-.84	397.45	.401
OC (mixed alpha)	Activity	.06	.06	[-.06, .17]	.96	402.18	.337
	Treatment	.21	.15	[-.10, .51]	1.36	48.55	.180
	Activity*Treatment	-.03	0.09	[-.21, .14]	-.38	400.59	.700