Chemosignals Communicate Human Emotions

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Psychological Science, in press

Author note

This research was supported by a NCU grant. We thank TJA van Aerts, MA van den Hout, A Toet, JHT Verbugt, S Sportman, and R Duinkerken for their contributions, and the suppliers of Neutral for free supply of their products.

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Abstract

Are humans able to communicate emotional states via chemical signals? This question was answered by examining the function of chemosignals in a framework furnished by embodied social communication theory. From this, we derived the hypothesis that the processes a sender experiences during distinctive emotional states are transmitted to receivers by means of the chemicals that the sender produces, thus establishing a multi-level correspondence between them. In a double-blind experiment, we examined facial reactions, sensory regulation processes, and visual search in response to chemosignals. We demonstrated that fear chemosignals generated a fearful facial expression and *sensory acquisition* (increased sniff magnitude and eye scanning) while disgust chemosignals evoked a disgusted facial expression and *sensory rejection* (decreased sniff magnitude, detection sensitivity, and eye scanning). These findings underline the neglected social relevance of chemosignals in regulating communicative correspondence outside of conscious access.

Keywords: Social communication, chemosignals, emotional contagion, olfaction, fear, disgust

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Chemical agents play an important role in affecting intraspecies' behavioral responses. These types of effects are not unique to animals, as they have also been reported in humans (Wysocki & Preti, 2004). The extent to which chemosignals[1] (Doty, 2010) serve a communicative function has remained unclear, mainly because hypotheses concerning the social aspect of human emotional chemosignaling have not been tested. Here, we investigate whether the inhalation of chemosignals emitted by a person during an emotional state induces the same state in another. Do recipients of chemosignals emulate the emotions experienced by their producers? We investigated this question by examining systematically whether a receiver reproduces not only the facial expression, but also the concomitant sensory regulation processes (i.e., sniffing behavior, detection sensitivity, and gazing behavior) that are associated with the emotional states involved in the production of the chemosignals. Our findings revealed a uniform and distinctive communicative impact of chemosignals.

Chemosignal detection was traditionally believed to require a fully functioning vomeronasal organ, allegedly absent in most humans (Wyatt, 2003). Following a more recent perspective, the main olfactory system is now believed to be actively involved in chemosignal communication in both animals and humans (Tirindelli, Dibattista, Pifferi, & Menini, 2009). Moreover, evidence has been identified that supports the so-called signaling (e.g., Kaitz, Good, Rokem, & Eidelman, 1987) and modulating (e.g., Zhou & Chen, 2009) effects of chemical emissions in humans. To date, interest has focused primarily on the *neural* and *behavioral* consequences of chemosignaling. Of special relevance is recent research examining the effects of fear chemosignals, from which two conclusions can be drawn: (1) Compared to control conditions (e.g., sport sweat), exposure to sweat excreted by donors experiencing fear enhanced

vigilance and caution (Chen & Haviland-Jones, 2000; Ackerl, Atzmueller & Grammer, 2002; Pause, Ohrt, Prehn, & Ferstl, 2004; Prehn, Ohrt, Sojka, Ferstl, & Pause, 2006; Chen, Katdare, & Lucas, 2006; Pause, Adolph, Prehn-Kristensen, & Ferstl, 2009; Zhou & Chen, 2009; Haegler et al., 2010; Albrecht et al., 2011; Zernecke et al., 2011); and (2) these effects have been shown to occur outside conscious awareness (Sobel et al., 1999; Lundström, Boyle, Zatorre, & Jones-Gotman, 2008; Mujica-Parodi et al., 2009). In sum, these studies clearly document the psychological and neural consequences of fear chemosignals.

Importantly, the social relevance of these recent developments has not been realized, presumably because the research focus has primarily been on the functional implications of chemosignals (e.g., does fear sweat cause a bias to recognize fear in ambiguous facial expressions), and not on their social communicative function, which is the core argument in the current research. Specifically, we hypothesized that chemosignals produced by a sender during an emotional state induce in a receiver a facial expression that reproduces the emotional state of the sender. The theoretical framework we advance here suggests that chemicals in bodily secretions serve the function of recruiting joint processes in sender and receiver by means of which correspondence is established. This communication perspective (Semin, 2000, 2007) invites thinking about emotional chemosignaling as a process entailing partial synchronization between sender and receiver and is probably a contributor to what has been termed as *emotional contagion* (Hatfield, Cacioppo, & Rapson, 1993). What emotional contagion entails is that the affective, behavioral, and perceptual processes observed in a receiver are a partial reproduction of the state in which a sender is.

The specific chemosignals we investigated here are produced by emotional states of fear and disgust. Obviously, the visual expressions of these emotions serve *communication*

universally (Ekman & Friesen, 1975). They are also *functional* because the adaptive features of facial expressions enhance the survival values for the person expressing the emotion (Susskind et al., 2008). Fear contagion, for instance, optimizes chances of survival by linking individuals multimodally and signals warnings about environmental danger (Susskind et al., 2008). Likewise, disgust contagion signals the avoidance of noxious chemical stimulation (Susskind et al., 2008). Thus, fear is associated with *sensory acquisition*, disgust with *sensory rejection*. This type of sensory regulation has been shown to be initiated by artificially induced facial expressions (Susskind et al., 2008). By taking on a fearful expression (i.e., opening the eyes), nasal inspiratory volume is increased, perception is enhanced, and eye movements during target localization are accelerated (Susskind et al., 2008). The opposite action pattern was observed after expressing disgust (i.e., eyebrow lowering and nose wrinkling) (Susskind et al., 2008).

Relying on this work, we advance the hypothesis that inhaling an emotional chemosignal is sufficient to induce the same consequences in a receiver as were experienced by a chemosignal producer. Hence, chemosignals were expected to affect a receiver's facial expression such that it corresponds to the emotional expression of the sender along with the adaptive function of such an expression that modulates perceptual, affective, and behavioral processes.

This general hypothesis was tested in a double-blind within-subjects experiment. Participants were exposed to sweat sampled from donors in specific emotional states (i.e., fear and disgust) and unused absorbent compresses (control condition). We expected that emotional contagion through fear chemosignals would generate a fearful facial expression (i.e., *medial frontalis* muscle activity) in a receiver, which would induce *sensory acquisition* reflected in an increased sniff magnitude, heightened target detection sensitivity and enhanced eye scanning. Emotional contagion through disgust chemosignals was hypothesized to generate a disgusted

facial expression (i.e., *levator labii* muscle activity) in a receiver. This in turn would induce *sensory rejection* expressed by a reduced sniff magnitude, dampened target detection sensitivity, and reduced eye scanning behavior. The anticipated results are unique in the sense that they underline a remarkable human capability, namely that chemicals excreted by one individual have social relevance by inducing the very same somatic states in another.

Method

Chemically mediated social communication constitutes a novel hypothesis that is optimally tested by using male senders and female receivers, because males produce stronger signals and females are more receptive to these signals (Wysocki et al., 2009).

Part I

Participants. Ten heterosexual males (M = 22.90 yrs, SD = 1.66 yrs) donated sweat prior to the experiment for €20. Emotions were induced by having the donors watch fear and disgust-evoking videos in two counterbalanced sessions separated by one week.

Procedure. Donors followed a strict protocol to avoid sweat contamination. For two days prior to the donation, odorous food, alcohol, smoking, and excessive exercise was prohibited. Donors used scent-free personal care products and detergents provided by the experimenter. After application of sterile absorbent compresses (Cutisorb BSN, Hamburg, Germany) under their armpits, donors were seated individually (temperature: 23˚C). Heart rate and skin conductance were assessed while donors watched pilot-tested 25-minute videos. Fear videos, (modeled after Zhou & Chen, 2009) contained horror scenes (e.g., the Shining; Rottenberg, Ray, & Gross, 2007), whereas MTV's Jackass induced disgust (de Jong, van Overveld, & Peters, 2011). Before and after the videos, donors filled in Spielberger's State-Trait Anxiety Inventory (van der Ploeg, Defares, & Spielberger, 1980) and rated their emotions on seven-point Likert

scales. Afterward, sweat pads were removed and stored (-22ºC). The same procedure was applied to unused absorbent compresses, which in our view constitute optimal control stimuli, since introducing other non-emotional bodily secretions (e.g., sport sweat) can potentially contain other chemosignals. Stimulus freezing does not affect pleasantness, intensity, attractiveness, and masculinity ratings (Lenochova, Roberts, & Havlicek, 2009).

Part II

Participants and design. Thirty-six right-handed females (M = 21.33 yrs, SD = 2.11 yrs) with a normal sense of smell (smell threshold: M = 11.26 binary dilution steps [3.25×10^{-3}% phenethyl alcohol], SD = 2.27) were exposed to sweat for €8. Participants enrolled in a double-blind counterbalanced odor exposure (fear sweat, disgust sweat, control pads) by visual search task (easy, difficult) within-subjects design.

Materials.

Stimulus composition. Sweat pads were cut into eight even pieces. Participants were presented vials that held four pads from four different donors. Half of the pads came from the armpit on one side of the body.

Intranasal cannula. Participants wore an unobtrusive nasal pressure monitoring cannula (PT, Sleep Sense) that was inserted 0.5 cm into the nostrils to measure sniffing.

Facial electromyography. Facial muscle activity was measured on the left side of the face (Dimberg & Petterson, 2000), using bipolar placements of Ag-AgCl surface-electrodes to measure fear (*medial frontalis*) and disgust (*levator labii*) (Fridlund & Cacioppo, 1986).

Eye tracking. Eye movements were recorded by an infra-red stereo camera at 120 Hz sampling rate (Tobii X120, Tobii Technology AB, Danderyd, Sweden).

Visual search. This task was adapted from Müller-Plath and Pollmann (2003). All items were equidistantly placed with 30˚ angular distance on an imaginary circle with a diameter of 8˚ visual angle. The target was in-between clearly visible and barely perceptible, as it varied only in shape from the distracters (width-to-height ratio: 0.83 vs. 1.00, respectively).

Stimulus rating and discrimination. Pleasantness and intensity were rated on 7-point Likert scales; vials were presented at a predetermined counterbalanced order. To assess participants' ability to discriminate odors, a forced-choice triangle test was used (see Meilgaard, Civille, & Carr, 1991, for details).

Smell threshold test. Olfactory threshold was assessed with Sniffin' Sticks (Burghart Instruments, Wedel, Germany). Participants were tested in an up-and-down staircase triple forced-choice test with a 7-reversal criterion while blindfolded (see Hummel, Sekinger, Wolf, Pauli, & Kobal, 1997, for details).

Awareness check. Funneled post-experimental debriefing (Bargh & Chartrand, 2000) revealed that participants were unaware of the purpose of the study and the source of the compounds.

Procedure. Since male experimenters have been shown to increase female participants' mood (Jacob, Hayreh, & McClintock, 2001), only females served as experimenters. Both experimenters (Verbugt and Sportman) and participants were blind to stimulus content and experimental condition, because vials were counterbalanced and coded with three-digit codes devised by Smeets and Kaldewaij. Experimenters did not disclose the nature of the study and were instructed to display only neutral expressions. The odor stimuli were defrosted 30 minutes prior to the experiment; each participant received a fresh container. Participants provided written informed consent and were seated in individual cubicles. A nasal pressure monitoring cannula

was attached, after which EMG electrodes were applied. Their heads were stabilized in a chin rest. Participants had to complete an automated eye tracking calibration procedure provided by Tobii Studio software. Then, a vial (2 cm deep) containing the chemosensory stimulus was clipped 2 cm below the subject's nose to the chin rest keeping the stimulus at a constant distance from subjects' noses. Participants wore nose clips to prevent preliminary sniffs. The nose clip was removed just before the start of the visual search task, at which time a marker was placed in the online registration of physiological data to mark the session's start. In the subsequent experimental task, run in Presentation (Neurobehavioral Systems Inc., Albany, CA), participants decided on target presence/absence amidst four and ten distracters. Ten practice trials had to be completed (minimum accuracy: 90%). Response keys were counterbalanced across participants. Participants were instructed to look at the fixation cross in the center at the beginning of each trial. Visual stimuli appeared after one second. The actual task consisted of two counterbalanced blocks (easy and difficult) of 48 trials per exposure condition (fear, disgust, control), with an inter-trial time of one second. Between the task and debriefing, participants completed tests and questionnaires. Each cubicle had an integral ventilation system (refreshment rate: 5 cycles/hour) that cleaned the air between testing sessions.

Results

We sampled sweat from *senders* in fearful and disgusted states serving as chemosensory stimuli for *receivers* in the main experiment. Based on physiological assessments, emotion induction was successful. Paired t-tests[2] revealed that donors had higher heart rates in the fear condition than the disgust condition ($t(9) = 3.17$, $p = .011$, $d = 1.42$), while skin conductance levels did not differ significantly ($t(9) = 2.02$, $p = .074$) (Table S1 online). The effects reported in the next section cannot have been due to significantly differing pleasantness and intensity ratings

of chemosensory stimuli by *receivers* (cf. supporting analyses and Table S2 online), as these indicators of hedonic valence and arousal were included in the analyses as covariates in which they proved to be not significant. Furthermore, Greenhouse-Geisser correction for sphericity violation (EMG data) did not affect the interpretation of the results.

We tested the social communicative function of chemosignals first by examining whether chemosignals were sufficient to induce in a *receiver* a facial muscle configuration that was experienced by a *sender* when producing the chemosignal. A 3x2x5 repeated measures ANOVA with odor exposure (fear sweat, disgust sweat, control), facial muscle activity (*medial frontalis*, *levator labii*), and time (baseline, 0-1s, 1-2s, 2-3s, 3-4s after exposure) as factors yielded a significant three-way-interaction ($F(8,280) = 4.74$, $p = .004$, $\eta^2 = .06$). Next, separate ANOVAs were conducted per exposure condition for facial muscle activity induced shortly after exposure (epoch 1: 0-4s) and during the complete exposure time (epoch 2: 0-420s). An increase in *medial frontalis* activity (Figure1A) from baseline reflected the distinctive facial muscle signature of fear that was activated (epoch 1: $F(4,108) = 8.76$, $p < .001$, $\eta^2 = .02$) and maintained (epoch 2: $F(1,27) = 6.89$, $p = .014$, $\eta^2 = .02$) after fear chemosignal exposure. Likewise, *levator labii* activity (Figure 1B) reflected a disgusted facial expression that was activated ($F(4,116) = 15.36$, $p < .001$, $\eta^2 = .02$) and maintained ($F(1,29) = 15.44$, $p < .001$, $\eta^2 = .01$) after disgust chemosignal exposure. Moreover, while fear chemosignals generated an expression of fear and not of disgust in a receiver ($F(4,108) = 3.82$, $p = .006$, $\eta^2 = .01$), disgust chemosignals induced a facial configuration of disgust rather than fear ($F(4,112) = 6.32$, $p < .001$, $\eta^2 = .01$), and neither fear ($F(4,100) = 2.04$, $p = .095$), nor disgust ($F(4,104) = 1.69$, $p = .159$) were evoked in the control condition (supplemental results online). Chemosignals thus served as a medium for

communication. Mere inhalation was sufficient to induce in *receivers* a facial expression that reflected the emotion experienced by *senders* while they produced the chemosignal.

Next, we examined whether chemosignal-induced facial expressions modulated sniffing behavior. Whereas the first sniff was expected to be reflexively elicited and exploratory, the subsequent sniff was modulated in magnitude (Mainland & Sobel, 2006), consistent with the communicated emotion. We analyzed ten sniffs to meaningfully chart the unfolding of sniffing magnitude over time. A 3x10 repeated measures ANOVA with factors odor exposure (fear sweat, disgust sweat, control) and sniff number (10 sniffs) revealed significant changes in sniff magnitude over sniffs as a function of the olfactory stimulus ($F(18,540) = 3.24$, $p < .001$, $\eta^2 = .05$) (Figure 2). A further examination of the first couple of sniffs revealed a significant interaction ($F(2,60) = 9.13$, $p < .001$, $\eta^2 = .11$), an effect that was not observed from the third sniff onward ($F(14,420) = 1.18$, $p = .287$). Follow-up paired t-tests on the first two sniffs indicated that the magnitude of the first sniff was lower for fear than disgust ($t(32) = -2.87$, $p = .021$), whereas the magnitude of the second sniff was lower for disgust than fear ($t(32) = -3.83$, $p = .003$). Exposure to emotional chemosignals thus modulated sensory regulation processes temporarily, after which adaptation seemed to have taken place. Figure 2 depicts that sniff magnitude gradually decreased after nose clip removal in the control condition. A cyclic pattern of air intake emerged after emotional chemosignal exposure, in which each substantial reduction in sniff magnitude seems to be compensated in the subsequent sniff. The reversed systematicity in air intake observed in the fear and disgust condition arguably occurred as a function of the type of chemosignal. By temporarily increasing the sniff magnitude in the fear condition, a larger number of chemical compounds could potentially reach the olfactory epithelium (i.e., *sensory*

acquisition). The opposite pattern (i.e., *sensory rejection*) was observed after exposure to disgust chemosignals, which presumably served a protective function.

Next, we examined whether changes in facial musculature induced by chemosignals altered perception. For this purpose, we used a visual search task to assess participants' ability to detect a target amidst distracters. The task consisted of an easy (four distracters) and difficult (ten distracters) part. A 3x2 repeated measures ANOVA with factors odor exposure (fear sweat, disgust sweat, control) and task (easy, difficult) demonstrated that detection sensitivity (*d*', Macmillan & Creelman, 2005) was significantly lower on the difficult task ($F(1,35) = 19.04$, $p < .001$, $\eta^2 = .07$) and varied significantly between exposure conditions ($F(2,70) = 5.37$, $p = .007$, $\eta^2 = .03$; interaction not significant: $F(2,70) = 2.82$, $p = .066$). As predicted, detection sensitivity was lower in the disgust condition compared to the control condition (post-hoc ANOVA: $p = .001$), while sensitivity was not affected by task difficulty in the disgust condition ($t(35) = 1.72$, $p = .282$). In the fear condition, differences in detection sensitivity were not significantly different from the disgust and control condition ($F < 1$) (Figure 3A). Follow-up analyses on difference scores, however, indicated that detection sensitivity dropped from the easy to the difficult task in the fear condition in comparison to other exposure conditions (control: $t(35) = 2.56$, $p = .015$; disgust: $t(35) = 1.82$, $p = .049$).Taken together, the data suggest that perceptual benefits from a fear state may interact with task difficulty.

In addition to detection sensitivity, response bias (β) also varied as a function of the task ($F(1,35) = 12.25$, $p = .001$, $\eta^2 = .07$). Response bias is an individual's decision rule in terms of the likelihood ratio that response A is given over B, with higher levels indicating an increased likelihood of reporting the absence of the target during the difficult search task. As can be observed in Figure 3B, response bias increased markedly in the fear condition when the task

became difficult ($t(35) = 3.62$, $p < .001$). Thus, fear chemosignals induced caution on the difficult part of the search task.

Exposure to disgust chemosignals thus reduced detection sensitivity (*sensory rejection*) under all circumstances. In the fear condition, however, detection sensitivity was higher (*sensory acquisition*) when targets were easily detectible, whereas it was lower when targets were embedded in excessive distracters. These combined results suggest that perceptual benefits associated with fear are limited to easy target-distracter configurations.

An examination of eye scanning behavior was conducted to corroborate the connection between detection sensitivity and the visual system. Eye scanning is facilitated by fear, as widely opening the eyes increases the visual field (Susskind et al., 2008). Two 3x2 repeated measures ANOVAs with odor exposure (fear sweat, disgust sweat, control) and task (easy, difficult) as factors revealed significant differences in the number of target fixations ($F(2,70) = 4.43$, $p = .016$, $\eta^2 = .03$) and fixation duration ($F(2,70) = 4.45$, $p = .015$, $\eta^2 = .03$) between conditions. Compared to the control condition, fear chemosignals induced *sensory acquisition* as evidenced by fewer target visits ($p = .014$) and faster target and distracter visits ($p = .011$) (supplemental analyses; Table S3). *Sensory rejection* was evidenced by avoidance behavior rather than a decrease in scanning speed and effectiveness. A facial muscle expression of disgust (i.e., raising the cheek) restricted the lower visual field that is already limited during neutral viewing conditions (Susskind et al., 2008). Exposure to disgust chemosignals specifically resulted in fewer overall fixations on visual stimuli ($F(2,70) = 5.40$, $p = .007$, $\eta^2 = .02$) compared to fear chemosignals ($p = .025$) and control ($p = .024$). In sum, fear chemosignals induced *sensory acquisition*, adopting a quick scan strategy of the entire visual field, whereas disgust chemosignals induced *sensory rejection*, decreasing the number of fixations.

Discussion

The current study's main aim was to seek evidence for the human capability to communicate emotions via chemicals embedded in bodily secretions. Our results directly supported this hypothesis. Chemosignals induced emotional contagion (Hatfield et al., 1993), as was evidenced by receivers' distinctive facial muscle configurations that changed in line with the specific emotion experienced by a sender while secreting the chemosignal. Specifically, exposure to fear chemosignals generated a facial configuration of fear (i.e., *medial frontalis* activity) and not of disgust (i.e., *levator labii* activity). In contrast, exposure to disgust chemosignals resulted in a facial configuration of disgust rather than fear. Moreover, fear induced *sensory acquisition* in a receiver. Conversely, disgust initiated *sensory rejection*. These consequences occurred both outside of receiver awareness and showed no relationship to receivers' judgments of the pleasantness and intensity of chemosensory stimuli. The results can be considered unique in that they reveal a remarkable human capability, namely that chemosignals of fear and disgust establish correspondence between a sender and a receiver.

The current contribution introduced an embodied social communication model (Semin, 2000, 2007) that is derived from the argument that communication can be achieved only when a receiver emulates the bodily state of the sender. The present study supports the core contention of this model, and has revealed that chemosignals have a socially significant function by constituting a medium by means of which two individuals are emotionally synchronized in a multi-modal fashion (i.e., facial mimicry, sensory regulation processes). Synchrony specifically entails the production of partial *parity* or *correspondence*, which occurs after a chemosignal receiver produces an internal representation of the emotional state communicated by a sender. Exposure to sweat from donors awaiting an examination, for instance, automatically activated in

a receiver a neural circuit (i.e., insula, cingulate cortex, precuneus) mapping the sender's state (Prehn-Kristensen et al., 2009). What we propose here is that chemosignals constitute a medium inducing emotional contagion by recruiting joint states and processes in sender and receiver.

The current data suggest that fear and disgust are not only distinctive emotions in the way they reflect in facial expressions and behavior, but that they are distinctive with respect to the biomarker profile deposited onto the skin while individuals—in this case sweat donors—are experiencing these respective emotions. It is not prudent to assume that there is one or even a few unique chemical compounds triggering the well-defined behavioral responses in receivers. Questions regarding the composition of the "emotional chemosignal fingerprints" of fear and disgust as well as the exact mechanisms involved in sensing the chemosignals have largely remained unanswered. Nevertheless, chemical analyses of stress-related odors revealed that male signals were stronger, whereas females displayed greater sensitivity to these signals (Wysocki et al., 2009). The present results show strong evidence that different emotions can be communicated from males to females by chemical signals.

These findings run in the face of the commonly accepted assumption that human communication runs exclusively via language or visual channels. Neuronal networks responsible for body odor processing are remarkably *similar* to those of auditory and visual processing (cf. Lundström et al., 2008); like emotional visual stimuli, body odors receive increased attention and differential processing (e.g., amygdala and insular cortex) compared to non-body odors (Lundström et al., 2008). The *difference*, however, is that chemosignals embedded in bodily secretions contribute to a close-distance emotional message. Although its ecological validity has to be substantiated, our research suggests that emotional chemosignals can be potential contributors to emotional contagion in situations involving dense crowds. Moreover, although

bodily secretions may be consciously registered due to their inherent stimulus intensity, chemosignal recipients could not discriminate between different chemosensory stimuli and were unable to access the processes induced by these chemosignals. The present research thus reported the human capability to communicate emotional states via chemosignals, and constitute an invitation to investigate the communicative function of other chemosignals produced under other emotional states such as happiness or anger.

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Footnotes

[1]We prefer not to use the term *pheromone*, as this concept is controversial and fraught with problems (Doty, 2010) partly due to widely varying and very strict definitions of what constitutes a pheromone. We use the term *chemosignal* instead, which according to Doty is less problematic when communication is the referent (Doty, 2010, p.186).

[2]To determine statistical significance, the Bonferroni correction was applied and statistical significance was set at $p < .05$.

Figure 1. Mean baseline subtracted facial muscle activity after chemosignal (fear sweat, disgust sweat) and control pad presentation as a function of exposure time. Error bars, ± SEM. (a) Mean *medial frontalis* activity (fear expression) was increased after fear sweat presentation, both in the first four seconds after exposure (t = 1st second, 2nd second, 3rd second, 4th second) and throughout the first (t = 0-210s) and second part (t = 210-420s) of the visual search task. (b) In a similar vein, mean *levator labii* activity (disgust expression) was elevated after exposure to disgust sweat.

Figure 2. Mean sniff magnitude (nasal air pressure in mmH_2O over time) on the first ten sniffs after chemosignal (fear sweat, disgust sweat) and control pad presentation. Error bars, ± SEM. Exposure to fear sweat resulted in *sensory acquisition*, reflected in an increased sniff magnitude on the second sniff. Exposure to disgust sweat induced *sensory rejection*, observed from a decreased sniff magnitude on the second sniff.

Figure 3. Mean sensitivity (*d*') and response bias (β) displayed per exposure condition and split by task difficulty (easy, difficult). Error bars, ± SEM. (a) Sensitivity dropped markedly from the easy to the difficult task in the fear condition. Compared to a control condition, sensitivity was lower (*sensory rejection*) after exposure to disgust chemosignals. (b) Response bias increased significantly in the fear condition, reflecting a more conservative and less accurate response tendency when the task became more difficult.

Figure 1

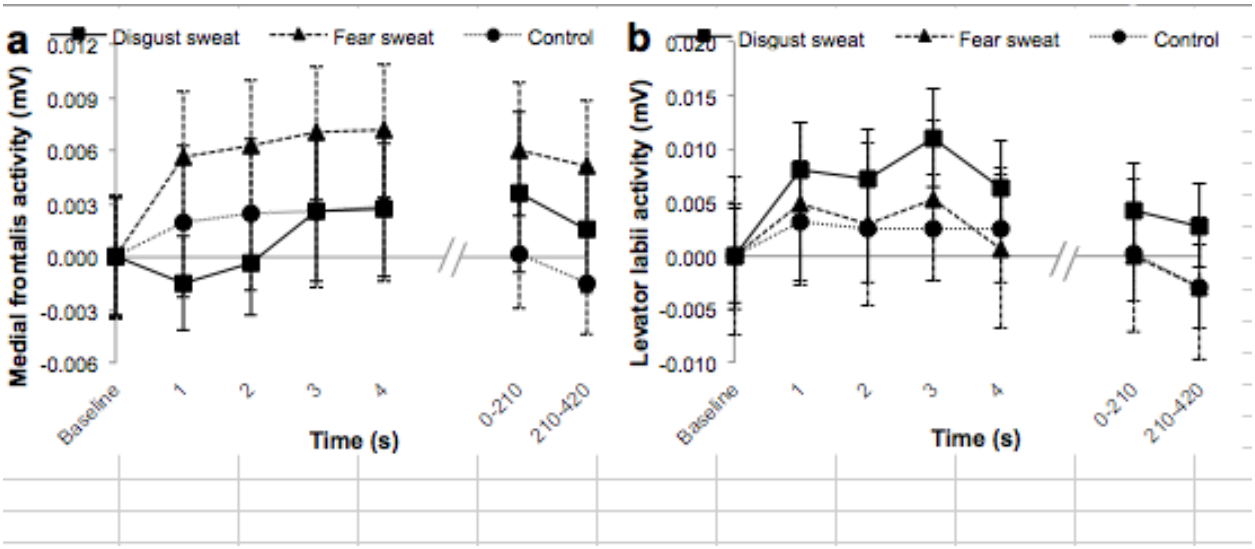

Figure 2

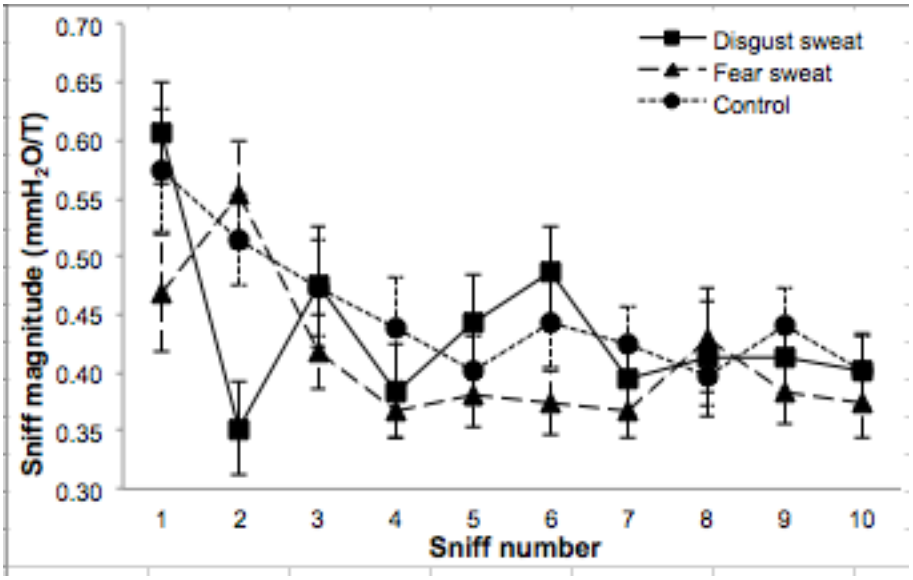

Figure 3

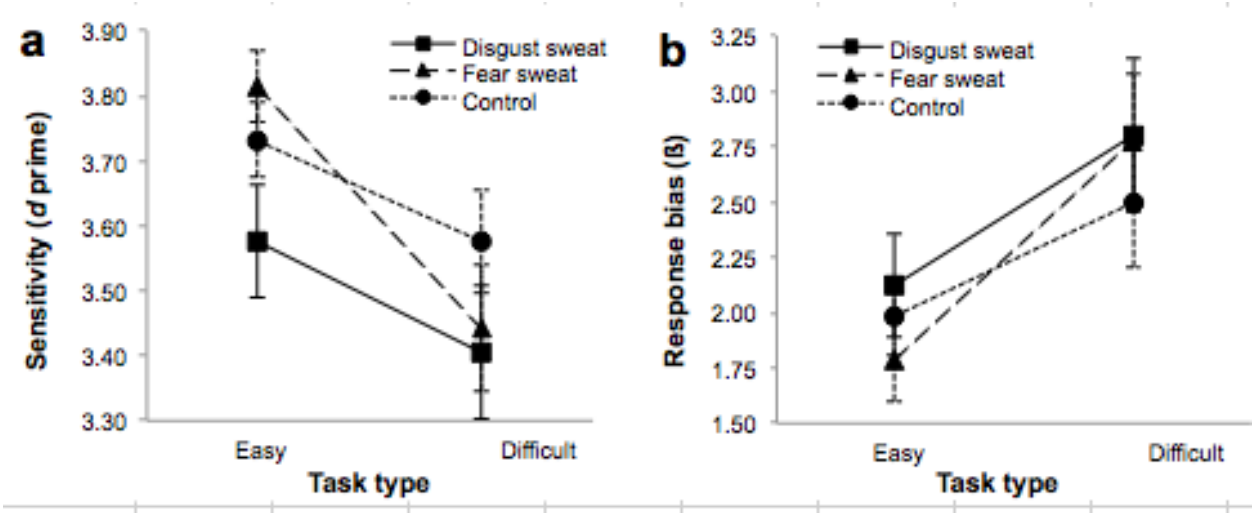
a
Sensitivity (d prime)
3.90
3.80
3.70
3.60
3.50
3.40
3.30
Disgust sweat
Fear sweat
Control
Easy
Difficult
Task type
b
Response bias (ß)
3.25
3.00
2.75
2.50
2.25
2.00
1.75
1.50
Disgust sweat
Fear sweat
Control
Easy
Difficult
Task type

Table S1

Physiological and subjective assessments of sweat donors

	Fear condition	Disgust condition	*p* value
Skin conductance level	2.60 (0.79-4.20)	2.22 (0.97-4.00)	.074
Heart rate	68.54 (48.74-85.50)	56.42 (48.67-65.95)	.011
Self-reported disgust	2 (1-5)	6 (4-7)	.005
Self-reported fear	2 (1-4)	1.5 (1-5)	.395
Self-reported anger	1.5 (1-3)	1 (1-4)	.785
Self-reported happiness	4 (3-6)	4 (2-7)	.719
Self-reported sadness	1 (1-4)	1 (1-7)	.414
Self-reported surprise	2.5 (1-6)	5 (3-7)	.024

Note. Skin conductance level was measured in microSiemens, heart rate in beats per minute, and self-report on 7-point Likert scales (1 = not at all; 7 = very). Data is presented as means (physiological data) and medians (subjective data), with ranges displayed between parentheses.

Table S2

Receivers' intensity and pleasantness ratings of sweat and unused pads

	Fear sweat	Disgust sweat	Control
Intensity	3.00 (1.31)	4.00 (1.45)	3.00 (1.37)
Pleasantness	4.00 (1.10)	3.00 (1.02)	4.00 (1.06)

Note. Evaluations were measured on 7-point Likert scales (1 = not at all; 7 = very) and are presented as medians. Standard deviations are displayed between parentheses.

Table S3

Eye tracking parameters per exposure condition as a function of task difficulty

	Fear sweat		Disgust sweat		Control	
	Easy	Difficult	Easy	Difficult	Easy	Difficult
Target fixations	0.69 (0.17)	0.76 (0.21)	0.73 (0.24)	0.74 (0.22)	0.78 (0.17)	0.82 (0.19)
Fixation duration	211.80 (37.37)	205.61 (29.35)	215.77 (38.79)	212.66 (27.02)	227.01 (47.82)	221.52 (45.13)
Overall fixations	5.58 (1.46)	11.17 (3.53)	5.55 (1.86)	9.94 (2.73)	5.67 (1.39)	11.22 (2.80)

Note. Depicted are the mean number of fixations on the target, mean fixation duration (ms), and mean number of overall fixations. Standard deviations are displayed between parentheses.

Additional analyses

Sweat donation (senders)

First, we checked whether emotion-induction in donors was successful. Because normality assumptions were violated for self-report data, non-parametric Wilcoxon signed-rank tests (df = 10) were used, which revealed that donors in the fear condition did not report significantly more fear than in the disgust condition (z = .85, p = .395) (Table S1). In the fear condition, self-reported state anxiety levels did not increase from baseline (z = 1.03, p = .305). These findings are consistent with the observation that self-report and physiological measures (cf. manuscript) often diverge in the assessment of fear in men (Pierce & Kirkpatrick, 1992). Compared to the fear condition, donors reported stronger feelings of disgust in the disgust condition (z = 2.83, p = .005, effect size (r) = .90), and they evaluated their feelings of surprise significantly higher in this condition (z = 2.25, p = .024, r = .71). As predicted, donors did not report increased levels of state anxiety after the disgust-inducing video compared to baseline (z = 1.58, p = .114).

Stimulus evaluation and discrimination (receivers)

We further examined how another group of participants evaluated sweat and control pads (Table S2). Wilcoxon signed-rank tests (df = 36) that compared disgust sweat, fear sweat, and control pad ratings revealed that disgust sweat was perceived as significantly more intense and less pleasant than fear sweat (intensity: z = 2.71, p = .007, r = .45; pleasantness: z = -2.00, p = .045, r = .33), and control pads (intensity: z = 3.14, p = .002, r = .52; pleasantness: z = -2.76, p = .006, r = .46). Differences between fear sweat and control pads in reported intensity (z = 1.14, p = .251) and pleasantness (z = -1.14, p = .213) were not significant. Despite these findings, participants were unable to discriminate between stimuli. With the minimum number of correct detections in the triangle tests being 18 for rejection of the no-discrimination hypothesis (given n

= 36, π = ⅓; Meilgaard, Civille, & Carr, 1991), participants could not discriminate between fear sweat vs. disgust sweat (13), control vs. disgust sweat (12), and control vs. fear sweat (9).

Facial expression modulation (receivers)

While exposure to unused control pads did not evoke emotional facial expressions, fear (disgust) chemosignals elicited a facial expression of fear (disgust). Fear chemosignals induced *medial frontalis* activity (cf. manuscript) that significantly increased from baseline in the third and fourth second after exposure (3rd second: $p = .032$; 4th second: $p = .028$) rather than the first two seconds (1st second: $p = .166$; 2nd second: $p = .083$), indicated by Bonferroni corrected post-hoc tests. Shortly after disgust chemosignal exposure, an early reduction in *medial frontalis* activity was followed by a marked increase, which contrasted the muscle activity pattern that was displayed in the fear condition ($F(4,116) = 3.12$, $p = .018$, $\eta^2 = .01$; p values > .817, for all 1s intervals). When adopting a time window that covered the complete visual search task (~7 minutes), *medial frontalis* activity was neither observed after disgust chemosignal exposure ($F(1,29) = 3.53$, $p = .071$), nor after control pad exposure ($F(1,25) = .27$, $p = .608$). Hence, fearful expressions were only reliably generated after fear chemosignal exposure.

Likewise, disgust chemosignals evoked a disgusted facial expression shortly after exposure that was maintained throughout the task (see manuscript). Post-hoc tests demonstrated a significant increase in *levator labii* activity from baseline up to the fourth second after exposure (p values < .001, for all 1s intervals). Although significant *levator labii* activity changes were shown after fear chemosignal exposure ($F(4,108) = 5.60$, $p < .001$, $\eta^2 = .01$), post-hoc tests revealed nonsignificant differences (p values > .077, for all 1s intervals). As predicted, a disgusted facial expression remained absent during the remainder of exposure to fear sweat

($F(1,27) = 1.29$, $p = .266$) and control pads ($F(1,26) = .73$, $p = .401$). Hence, disgusted expressions were only reliably generated after disgust chemosignal exposure.

Sniffing behavior (receivers)

Next to facial muscle activity, sniffing behavior was explored further. With regard to the first couple of sniffs, follow-up paired t-tests indicated that the magnitude of the first sniff neither differed between the control and fear condition ($t(31) = 2.05$, $p = .15$), nor between the control and disgust condition ($t(32) < 1$). The difference between the fear and control condition with respect to the magnitude of the second sniff was not significant ($t(31) < 1$), whereas a significantly lower sniff magnitude was indeed observed in the disgust condition relative to the control condition ($t(32) = 4.53$, $p < .001$). These combined findings (cf. manuscript) reflect the cyclic nature of air intake after chemosignal exposure. While fear chemosignals ostensibly induced rapid sensory acquisition, disgust chemosignals evoked sensory rejection.

Eye scanning (receivers)

Further support for chemosignal-induced sensory acquisition was obtained from eye tracking data. Exposure to fear sweat resulted in fewer fixations on the target (Table S3) compared to the control condition (post-hoc ANOVA: $p = .011$), but not the disgust condition ($p = .145$). Exposure to fear sweat furthermore led to shorter average fixation durations compared to the control ($p = .014$), but not the disgust condition ($p = .705$). The eye fixation modulating effects that were induced by fear chemosignals potentially reflected the employment of a quick scan search strategy of the entire visual field, rather than mere fixations on individual objects within a space.

Additional reference

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