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Nasal airflow engages central olfactory processing and shapes olfactory percepts

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Binding of airborne odour molecules to olfactory receptors at the top of the nasal cavity gives rise to our rich olfactory experience. Whether airflow plays a role in human olfactory perception beyond the transportation of odorants is scantly known. Combining psychophysical measures with strict controls of nasal flow parameters, we demonstrate in four experiments that the perceived intensity of a unilaterally presented odour decreases systematically with the amount of contralateral nasal airflow, in manners that are independent of odour flow rate, nasal pressure, perceived sniff vigour or attentional allocation. Moreover, the effect is due to the sensed rather than the factual amount of nasal flow, as applying a local anaesthetic to the contralateral nostril produces the same effect as physically blocking it. Our findings indicate that nasal flow spontaneously engages central olfactory processing and serves as an integral part of the olfactory percept in humans.

1. Background

Inhaling and exhaling, we smell odours. In that time, odorants carried by airflow make contact with the olfactory epithelium on the roof of the nasal cavity, triggering olfactory sensory neurons, and trigeminal afferents innervating the nasal cavity generate the sensation of nasal airflow [1]. Whereas olfactory perception apparently depends on nasal flow, it is commonly held that such dependence is limited to the initial stage of odorant transportation. For instance, the velocity at which an odorant flows across the olfactory mucosa interacts with its sorption rate to shape the distribution pattern of the molecules along the epithelium, thereby influencing olfactory nerve responses and perception [2–5]. Nonetheless, recent studies in mice indicate that the detection of odour molecules and that of airflow are, to a certain extent, entwined at the periphery. Some olfactory sensory neurons respond not only to odorants but also to mechanical stimuli like airflow via a shared cAMP cascade [6,7]. This mechanosensitivity drives sniff-coupled oscillations in the olfactory bulb, which could facilitate phase coding of odour identity [8], but does not appear to be directly linked to one's subjective experience of nasal airstream [1]. Studies on central olfactory processing have seldom dealt with nasal flow, despite that sniffing pure air is reported to activate the piriform cortex, a well-documented substrate of odour object perception [9,10].

Do central olfactory processing and its perceptual outcome entail the processing of nasal flow? To tackle this issue, we exploit the anatomical lateralization of the olfactory system and set out to examine subjective odour intensity—an elemental feature of perception [11]. The two nostrils are separated by the nasal septum. Binaral inputs only converge downstream of the olfactory bulb [12,13], in which temporal patterns of mitral/tufted cells' responses are suggested to represent subjective intensity [14]. This enables us to dissociate odorant transportation from nasal airflow at the periphery by using unilateral presentation. In a series of experiments, we assess whether manipulations of the physical or the sensed amount of contralateral nasal airflow would impact the subjective intensity of unilaterally presented odours. Potential confounding factors, including

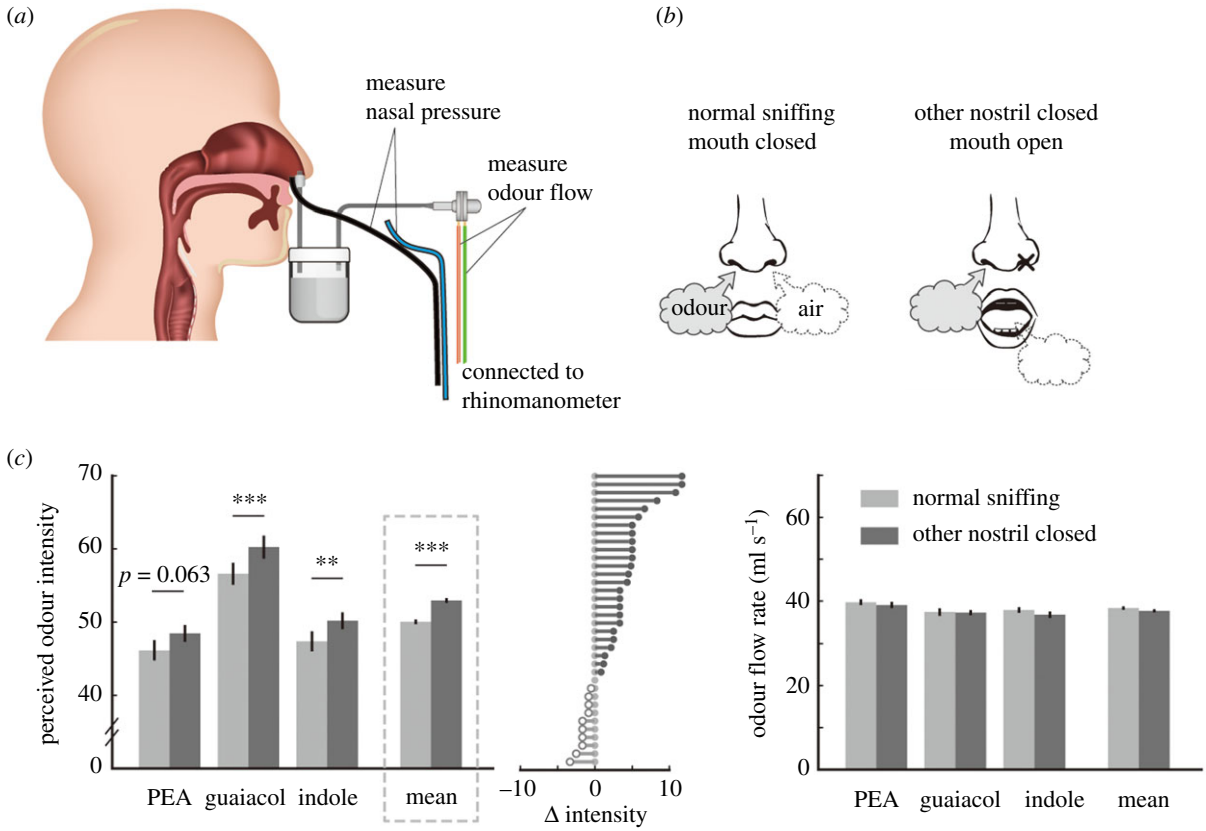

Figure 1. Elimination of contralateral nasal flow enhances perceived intensity of unilaterally presented odours. (*a*) Experimental set-up. The odours were presented unirhinally. Odour flow rate and nasal pressure in the exposed nostril were simultaneously recorded and fed back to participants while they were taking a sniff. (*b*) Participants in Experiment 1 sniffed the odours with the contralateral nostril naturally open and the mouth closed (normal sniffing) in half of the trials, and with the contralateral nostril pinched shut and the mouth slightly open in the other half of the trials. (*c*) The elimination of contralateral nasal airflow strengthened the perceived intensities of unilaterally presented odours (left), despite stable odour flow rates in the exposed nostril (right). Middle: ranked individual data for the overall difference in perceived odour intensity relative to normal sniffing highlighted by the dashed rectangle. PEA, phenylethyl alcohol. Error bars: s.e.m. values adjusted for individual differences. $^{**}p < 0.01$, $^{***}p < 0.001$. (Online version in colour.)

odour flow rate [15], nasal pressure [16], perceived sniff vigour [17], sniff duration [18] and attentional allocation are carefully controlled and monitored.

2. Methods

(a) Participants

A total of 144 healthy non-smokers participated in the main study, 36 (17 males, mean age ± s.d. = 22.1 ± 2.3 years) in Experiment 1, 36 (17 males, 21.9 ± 2.7 years) in Experiment 2, 36 (18 males, 21.7 ± 2.6 years) in Experiment 3 and 36 (18 males, 21.2 ± 2.3 years) in Experiment 4. Sample sizes were determined by G*Power to be adequate to detect a medium effect in odour intensity perception ($d \approx 0.5$), at a power of about 85%. All participants reported having a normal sense of smell, no nasal obstruction and no respiratory allergy or upper respiratory infection at the time of testing. All passed our initial screening where they were presented with each olfactory stimulus and asked whether an odour was clearly detectable. None had significant nasal septal deviation as assessed by nasal spirometry (GM Instruments, Glasgow, UK) (nasal partitioning ratios ranged between −0.4 and 0.4) [19]. They were blind to the purposes of the experiments.

(b) Olfactory stimuli

The olfactory stimuli in each experiment consisted of phenylethyl alcohol (PEA, a rose-like odour, 1% v/v in propylene glycol), guaiacol (smoky, 1% v/v in propylene glycol) and indole (faecal, 2% m/v in propylene glycol), which were supra-threshold to all participants. The odorants differ in valence and were generally perceived as pleasant, neutral and unpleasant, respectively, by an independent panel of 24 odour judges (12 males, 26.0 ± 2.9 years; mean valence ratings on a 100-unit visual analogue scale where 100 denotes extremely pleasant: 66.0, 47.1 and 40.9 versus neutral = 50, $p < 0.001$, $p = 0.40$ and $p = 0.016$, respectively). They also differ in trigeminality (degree to which the trigeminal nerve is stimulated) [20], water solubility (20, 18.7 and 3.56 mg ml^{-1} at 25°C, respectively), and hence likely mucosal sorption pattern [21]. They were presented unirhinally (to a single nostril) in identical 40 ml polypropylene jars. Each jar contained 10 ml of clear liquid and was fitted with a Teflon nosepiece and a separate tube whose free end was connected to the flowhead of a rhinomanometer (NR6, GM Instruments, Glasgow, UK) during odour presentation.

(c) Procedure

Experiment 1 employed a unilateral intensity judgment task, which allowed simultaneous recordings of odour flow rate and sniff pressure as an odour was sampled by using adapted anterior rhinomanometry [22]. As shown in figure 1*a*, during odour presentation, the odour jar was connected on one side with the nasal cavity (left or right) through a nosepiece and on the other side with the flowhead of a rhinomanometer. This ensured that the amount of odour flow sniffed into the nasal cavity was equivalent to that recorded by the rhinomanometer. Nasal pressure was measured at the same time with a pair of pressure tubes connected to the rhinomanometer: One (blue tube) was left in the air to sense atmospheric pressure, and the other (black tube) was attached to an anterior tube placed

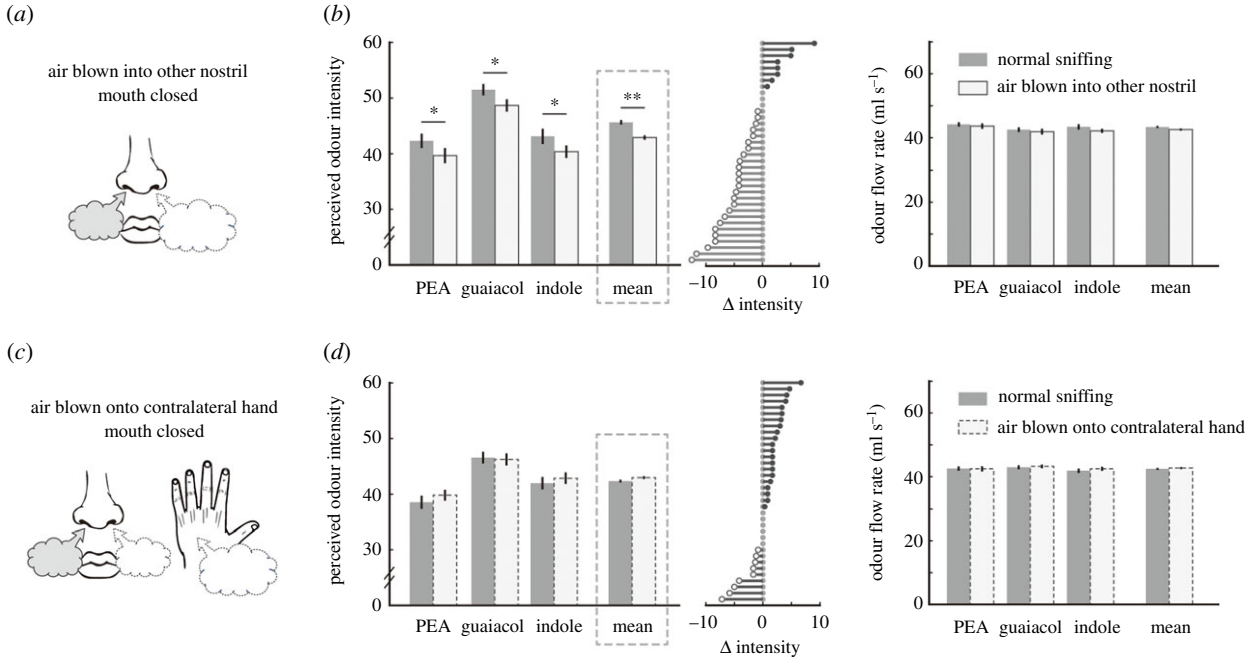

Figure 2. Augmentation of contralateral nasal flow reduces perceived intensity of unilaterally presented odours independent of attentional allocation. (a) Participants in Experiment 2 sampled the odours with the mouth closed. The unexposed nostril either was naturally open (normal sniffing) or had additional air blown in at 5 l min^{-1}. (*b*) The added contralateral nasal airflow diminished the perceived intensities of unilaterally presented odours without affecting odour flow rates in the exposed nostril. (*c*,*d*) In Experiment 3, the additional airflow was directed onto the hand contralateral to the exposed nostril (*c*), which produced no impact on perceived odour intensities or odour flow rates in the exposed nostril (*d*). Middle panels in (*b*,*d*) are as in figure 1*c*. PEA, phenylethyl alcohol. Error bars: s.e.m. values adjusted for individual differences. *$p < 0.05$, **$p < 0.01$.

inside the nasal cavity alongside the nosepiece to form a tight fit. Air leakage, if any, would be systematic and not affect the comparison of nasal pressure across different conditions. The output values of the rhinomanometer were instantaneously displayed in a 2D space spanned by odour flow rate and nasal pressure. Participants sampled the unilaterally presented odours either with the other nostril naturally open and the mouth closed (normal sniffing) or with the other nostril pinched shut (with the index finger) and the mouth slightly open (figure 1*b*). They were trained beforehand to control the openness of the mouth such that the amount of air inhaled through the mouth roughly matched that which would be inhaled through the closed nostril during normal sniffing. Essentially, the goal was to have comparable odour flow rates and nasal pressures in the odour-exposed nostril between the two manners of sniffing. They were asked to regulate their sniff vigour, always exhale through the mouth, and to produce a consistent time course of the 2D display, including the starting and highest points, every time they took a sniff, and, as mentioned, were given training to do so prior to the actual experiment. In each trial, they followed auditory prompts—three 1.5 s beeps with a 2.5 s gap in between—to take three sniffs of an odour, each 1.5 s long, and then rated its perceived intensity on a 100-unit visual analogue scale, with 100 representing extremely intense. A natural sniff episode during odour perception comprises several sniffs, the first of which seems to be most informative and the subsequent ones confirmatory [23]. Subjective odour intensity generally does not change with sniff duration beyond 1 s and is unaffected by the number of sniffs [18,23]. Hence, three sniffs each 1.5 s long with a 2.5 s gap in between should be sufficient for one to reach an accurate intensity judgement while also ensuring that sniff rates are constant across olfactory and airflow conditions. Participants then assigned a number from 1 to 10 to indicate how vigorously they had sniffed, with 10 denoting extremely vigorously. To facilitate comparison, each odour was presented to each nostril in two consecutive trials that differed only in how the odour was sampled. Each participant completed 12 trials of the unilateral intensity judgment task (3 odours × 2 nostrils × 2 manners of sniffing), with a break of at least 30 s in between two trials. The order of odour, nostril of exposure and manner of sniffing was balanced within and across participants.

In Experiments 2 and 3, participants sampled the unilaterally presented odours with the mouth closed. In half of the trials, there was no additional airflow and participants sniffed normally; in the other half, purified air was blown either into the unexposed nostril (Experiment 2, figure 2*a*) or onto the hand contralateral to the exposed nostril (Experiment 3, figure 2*c*) at 5 l min^{-1} via a computer-controlled olfactometer (Emerging Tech Trans, PA, USA). This additional airflow was synchronized with the auditory sniffing prompts and presented briefly and intermittently for 4.5 s per trial (three sniffs of 1.5 s each) in half of the trials. The procedures were otherwise identical to those in Experiment 1. None of the participants in Experiment 2 reported experiencing coldness, pain, nasal congestion, or increased nasal secretion—symptoms previously associated with prolonged exposures to additional airflow introduced to the nasal cavity [24].

Participants in Experiment 4 each completed four test sessions held on two consecutive days, including one session before and one session 5 min after drug administration on each day. In each session, they performed 12 trials of the unilateral odour intensity judgment task (four trials per odour in random order) and rated on a 10-point Likert scale how clear each nasal passage felt, with 10 representing extremely clear. The partitioning of airflow between the two nasal passages was then measured by a rhinospirometer (NV1, GM Instruments, Glasgow, UK). Across the sessions, the olfactory stimuli were always presented to the same nostril (left for half of the participants, right for the other half), and the participants always sampled them with the other nostril naturally open and the mouth closed (figure 3*a*,*b*). The unexposed nostril was treated with 2% g ml^{-1} tetracaine hydrochloride (tetracaine HCl, saline solution, two puffs via a regular nasal spray bottle, each puff with about 3 mg tetracaine HCl), a local anaesthetic, or saline, one on each day in a counterbalanced order, after the baseline session was over.

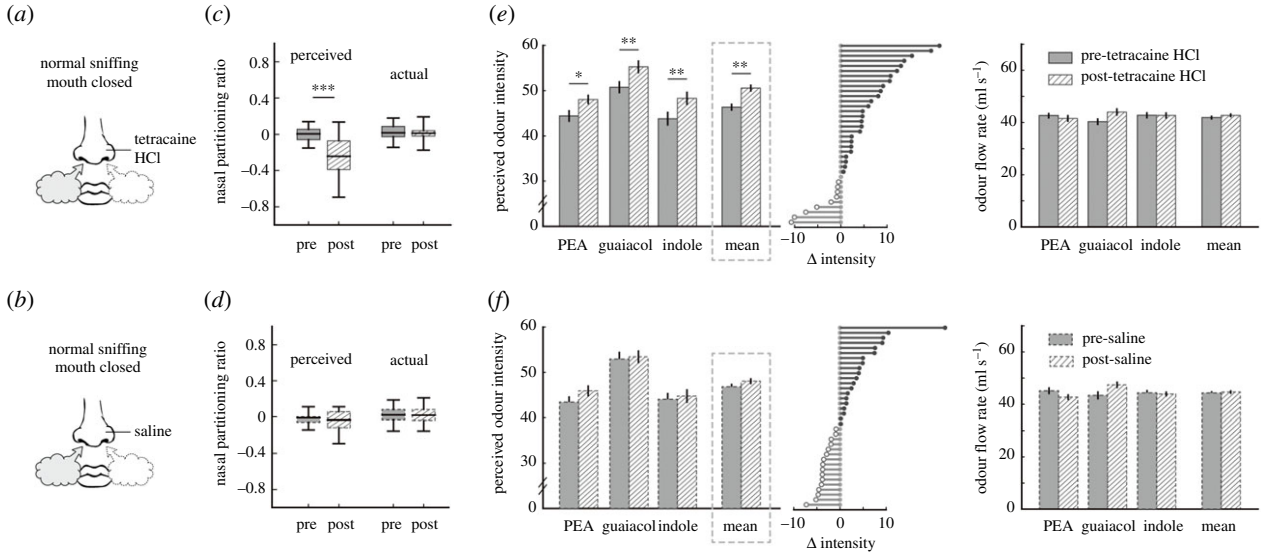

Figure 3. Odour intensity perception weights perceived as opposed to physical nasal flow. (*a*,*b*) Participants in Experiment 4 were tested on two days. They always sampled the unilaterally presented odours with the other nostril naturally open and the mouth closed. The unexposed nostril was treated with either tetracaine HCl (*a*) or saline (*b*), one on each day in a counterbalanced order. (*c*,*d*) Tetracaine HCl (*c*), but not saline (*d*), caused a significant decrease in the sensation of airflow in the treated nostril. Neither altered the actual nasal partitioning ratio. In each box and whisker plot, the central line denotes the mean, and the bottom and top edges of the box indicate the 25th and 75th percentiles. The ends of the whiskers represent 90% CI. (*e*,*f*) The unilaterally presented odours were perceived as more intense following the administration of tetracaine HCl (*e*), but not saline (*f*), to the contralateral nostril. Odour flow rates in the exposed nostril remained unchanged. Middle panels in (*e*,*f*): ranked individual data for the overall difference in perceived odour intensity relative to baseline (pre-drug treatment) highlighted by the dotted rectangles. PEA, phenylethyl alcohol. Error bars: s.e.m. values adjusted for individual differences. $*p < 0.05$, $**p < 0.01$, $***p < 0.001$.

(d) Analyses

We were primarily interested in whether the subjective intensity of unilaterally presented odours would be influenced by different manipulations of the physical (Experiments 1 and 2) or the sensed (Experiment 4) amount of contralateral nasal airflow or general attentional state (Experiment 3). To this end, we performed a series of paired sample *t*-tests to compare overall odour intensity ratings and the ratings for individual odours between (1) different manners of sniffing in Experiments 1 and 2, (2) the presence and absence of a distracting airflow in Experiment 3, and (3) pre- and post-drug treatment (tetracaine HCl or saline) in Experiment 4. For Experiments 1 to 3, self-reported sniff vigour, averaged odour flow rate and averaged nasal pressure in the exposed nostril during sniffing were analysed in separate repeated measures ANOVAs, using odour (phenethyl alcohol versus guaiacol versus indole) and airflow condition (Experiment 1: normal versus eliminated contralateral nasal flow; Experiment 2: normal versus augmented contralateral nasal flow; Experiment 3: absence versus presence of additional airflow blown onto hand) as the within-subject factors. In Experiment 4, we also compared the pre- and post-drug values of the actual and the perceived nasal partitioning ratios (NPRs) in separate paired-sample *t*-tests. NPR was calculated as

$$\mathrm{NPR} = \frac{(V_{\mathrm{treated}} - V_{\mathrm{untreated}})}{(V_{\mathrm{treated}} + V_{\mathrm{untreated}})},$$

where V_{treated} and $V_{\mathrm{untreated}}$, respectively, represent the volumes of airflow in the drug-treated and drug-untreated nostrils (actual NRP) or the subjective ratings of how clear the treated and the untreated nasal passages felt (perceived NRP). In addition, we conducted a series of repeated measures ANOVAs with actual NPR, perceived NPR, overall perceived odour intensity, self-reported sniff vigour, as well as odour flow rate and nasal pressure in the exposed nostril during sniffing as the dependent variables, respectively, and drug treatment and test session (pre- versus post-drug application) as the within-subject factors. All statistical tests were two-tailed.

3. Results

(a) Contralateral nasal airflow impacts unilateral odour intensity perception independently of attention

In Experiment 1, 36 healthy non-smokers performed a unilateral intensity judgement task devised to maximize control over sniff parameters (see Methods, figure 1*a*). In each trial, they unilaterally sampled phenethyl alcohol, guaiacol or indole, which differed in valence, trigeminality [20] and plausibly mucosal sorption pattern [21], while the odour flow rate and sniff pressure in the exposed nostril were simultaneously recorded. They then rated the perceived intensity of the odour and their subjective sniff vigour. Each odour was sampled in two different manners (figure 1*b*), i.e. with the other nostril naturally open and the mouth closed (normal sniffing) or with the other nostril pinched shut and the mouth slightly open (to compensate for the amount of air that would be inhaled through the closed nostril during normal sniffing, see Methods for details).

Regardless of the olfactory stimulus, the presence or absence of contralateral nasal airflow did not significantly influence the average odour flow rate (main effect: $F_{1,35} = 0.66$, $p = 0.42$; interaction: $F_{2,70} = 0.32$, $p = 0.73$; figure 1*c*) or sniff pressure (main effect: $F_{1,35} = 0.33$, $p = 0.57$; interaction: $F_{2,70} = 0.28$, $p = 0.76$) in the exposed nostril during sniffing. There was a trend that the participants self-reported to have sniffed more vigorously with the other nostril pinched shut and the mouth slightly open (main effect: $F_{1,35} = 3.42$, $p = 0.073$; interaction: $F_{1.72,60.17} = 0.65$, $p = 0.50$), which was likely related to the unnaturalness of this manner of sniffing. According to the perceptual constancy model proposed by Teghtsoonian and colleagues [16,17,25], an increase in perceived sniff effort without an actual change in odour flow rate would reduce the perceived odour strength. What we observed, however, was

the opposite (figure 1*c*). The elimination of contralateral nasal airflow relative to normal sniffing led to a robust increase in subjective odour intensity across the olfactory stimuli (t_{35} = 4.54, $p < 0.001$, Cohen's $d = 0.76$) that was unrelated to the difference in sniff vigour ($r_{36} = -0.25$, $p = 0.14$). The pattern held for guaiacol ($t_{35} = 3.84$, $p = 0.001$) and indole ($t_{35} = 3.16$, $p = 0.003$), and trended towards significance for phenethyl alcohol (t_{35} = 1.92, $p = 0.063$). In other words, the perceived intensity of a unilaterally presented odour was enhanced by a reduction in contralateral nasal airflow, despite overall stable odour flow rate and nasal pressure in the exposed nostril. We wondered if the reverse would also be true—that unilateral odour intensity would be diminished by augmented contralateral nasal airflow—and carried out Experiment 2.

The participants in Experiment 2 either sniffed the olfactory stimuli normally with the other nostril naturally open or had additional purified air blown into the other nostril at 5 l min^{-1} (figure 2*a*). They always sniffed with the mouth closed. The procedures were otherwise identical to those of Experiment 1. As expected, the added contralateral nasal airflow produced a significant drop in perceived odour intensity as compared with normal sniffing ($t_{35} = -3.43$, $p = 0.002$, Cohen's $d = 0.57$; figure 2*b*) for all of phenethyl alcohol ($t_{35} = -2.66$, $p = 0.012$), guaiacol ($t_{35} = -2.66$, $p = 0.012$) and indole ($t_{35} = -2.64$, $p = 0.012$). Meanwhile, between the two manners of sniffing, there was no significant difference in the average odour flow rate ($F_{1,35} = 1.44$, $p = 0.24$; figure 2*b*) or nasal pressure ($F_{1,35}$ = 0.74, $p = 0.40$) in the exposed nostril during sniffing irrespective of the olfactory stimulus (interactions: $F_{2,70} = 0.14$ and 0.99, $p = 0.87$ and 0.38, respectively). The participants' perceived sniff vigour also remained unaltered (main effect: $F_{1,35} = 0.69$, $p = 0.41$; interaction: $F_{1.72,60.23} = 1.64$, $p = 0.20$). To facilitate comparison, electronic supplementary material, figure S1a highlights the central tendencies of the percentage changes in subjective odour intensity in Experiments 1 and 2. The bootstrapped sample means formed two distinct normal distributions with the centres 12.7% apart. That is, the different manipulations of contralateral nasal airflow (elimination versus augmentation) overall induced a substantial 12.7% change in the intensity perception of unilaterally presented odours, in spite of the fact that the participants were explicitly aware that the odours remained unchanged. We also verified through prolonged testing of three volunteers that unilateral odour intensity decreased monotonically with the amount of contralateral nasal airflow (electronic supplementary material, supplementary experiment, figure S1b). Collectively, these data pointed to a systematic impact of nasal airflow on olfactory intensity perception that goes beyond mere transportation of odorants.

One could argue, however, that the observed effects were mediated by attentional allocation rather than nasal airflow *per se*. For instance, the participants in Experiment 2 could have been distracted by the additional airflow in the contralateral nostril and failed to pay sufficient attention to the unilaterally presented odours, which caused them to perceive the odours as weaker. To address this possibility, we recruited another 36 participants in Experiment 3 and tested them with the same procedures as in Experiment 2, except that the additional airflow of 5 l min^{-1} was directed onto the contralateral hand instead of into the contralateral nostril (figure 2*c*). A large portion of the somatosensory cortex is dedicated to the processing of information from the hands [26], and the participants all clearly noticed the additional airflow on the hand (when present) during sniffing. Nonetheless, their odour intensity ratings were unaffected by its presence or absence (overall: $t_{35} = 1.28$, $p = 0.21$; phenethyl alcohol, guaiacol and indole: $t_{35} = 1.58$, −0.42 and 1.10, $p = 0.12$, 0.68 and 0.28, respectively; figure 2*d*). The average odour flow rate (figure 2*d*) and sniff pressure in the exposed nostril, as well as the perceived sniff vigour, were also unaffected by its presence or absence across the olfactory stimuli (main effects: $F_{1,35} = 0.16$, 0.16 and 0, p = 0.69, 0.69 and >0.9; interactions: $F_{2,70} = 0.14$, 0.12 and $F_{1.51,52.77} = 0$, $p = 0.87$, 0.89 and >0.9, respectively). These results hence ruled out attention as a major contributing factor to the pronounced impact of contralateral nasal airflow on unilateral odour intensity perception (Experiments 1 and 2).

(b) Central computation of olfactory intensity weights sensed as opposed to physical amount of nasal flow

The sensation of nasal flow mainly takes place in the most anterior part of the nasal cavity called the nasal vestibule and can be blocked by local anaesthesia [27,28]. In Experiment 4, we examined whether the application of tetracaine hydrochloride (tetracaine HCl), a local anaesthetic, to the contralateral nasal vestibule would produce a similar effect to that of eliminating the nasal airflow, namely, enhancing the subjective intensity of unilaterally presented odours (Experiment 1). The participants were tested on two consecutive days in a within-subject crossover design and always sampled the olfactory stimuli normally with the contralateral nostril naturally open and the mouth closed. On each day, they performed the unilateral intensity judgement task both before and after the contralateral administration of tetracaine HCl or saline (figure 3*a*,*b*). Tetracaine HCl caused a marked decrease in the sensation of airflow in the treated nostril ($t_{35} = -6.58$, $p <$ 0.001, Cohen's $d = 1.10$) without affecting the actual nasal partitioning ratio ($t_{35} = -0.45$, $p = 0.65$; figure 3*c*), an index of the asymmetry of nasal airflow. In parallel, the participants indeed experienced a significant increase in the intensity of the odours presented to the untreated nostril ($t_{35} = 3.43$, p = 0.002, Cohen's $d = 0.57$; figure 3*e*), including phenethyl alcohol ($t_{35} = 2.46$, $p = 0.019$), guaiacol ($t_{35} = 3.05$, $p = 0.004$), as well as indole ($t_{35} = 2.91$, $p = 0.006$). Saline, on the other hand, had no effect (sensation of airflow: $t_{35} = -1.72$, $p = 0.095$; odour intensity: $t_{35} = 1.28$, $p = 0.21$; figure 3*d*,*f*). For the half of the participants who received tetracaine HCl on Day 1 and saline on Day 2, their odour intensity ratings prior to saline treatment on Day 2 were comparable to the pre-tetracaine HCl ratings ($t_{17} = -0.087$, $p = 0.93$) and significantly lower than the post-tetracaine HCl ratings on Day 1 ($t_{17} = -3.07$, $p = 0.007$, Cohen's $d = 0.72$). That is, their odour intensity perception returned to baseline after the anaesthetic effect washed out. Overall, the interaction between drug (tetracaine HCl versus saline) and test session (pre- versus post-drug treatment) was significant for both the sensation of airflow in the treated nostril ($F_{1,35} = 44.56$, $p < 0.001$, partial $\eta^2 = 0.56$) and the intensity perception of odours in the untreated nostril ($F_{1,35} = 5.11$, $p = 0.030$, partial $\eta^2 = 0.13$), but not for the actual nasal partitioning ratio ($F_{1,35} = 0.001$, $p = 0.98$), the perceived sniff vigour ($F_{1,35} = 1.79$, $p = 0.19$), or the average odour flow rate ($F_{1,35} = 0.16$, $p = 0.69$) or sniff pressure ($F_{1,35} = 0.49$, $p = 0.49$) in the odour-exposed untreated nostril during sniffing. In addition, careful inspections of the odour flow traces across all experiments showed

that detailed wave properties like peak, rise time and fall time were also unrelated to subjective odour intensity (electronic supplementary material, supplementary analysis, figure S2). We therefore concluded that the sensed rather than the physical amount of nasal flow is factored in the central computation of olfactory intensity independently of odorant transportation. Since tetracaine HCl was administered via regular nasal spray bottles, there was a small chance that olfactory sensory neurons in the treated nostril were also affected. Our data thus did not rule out the possibility that mechanosensation in the olfactory sensory neurons could contribute to the observed modulation of perceived odour intensity by nasal airflow.

4. Discussion

Classical psychophysical laws derived from studies of brightness, loudness and weight state that the subjective perception is proportional to the logarithm (Weber–Fechner Law) or exponential (Stevens's Power Law) of the stimulus' physical strength, i.e. a bijection exists between a stimulus' physical strength and perceived intensity. By employing the classical method of magnitude estimation [29], the current study provides clear evidence that such a bijection fails for the perception of odour intensity when nasal airflow is independently manipulated. Specifically, the subjective intensity of a unilaterally presented odour decreases systematically with the increase of sensed (as opposed to physical) contralateral nasal airflow in manners that are independent of odorant transportation, sniff vigour and attentional allocation. The findings indicate that central olfactory processing spontaneously involves the processing of airflow information, the latter mainly relayed from branches of the trigeminal nerve innervating the nasal vestibule. Moreover, subjective odour intensity, unlike brightness, loudness and weight, is inherently a bimodal (odorants and airflow) rather than unimodal percept. In doing so, the findings shed fresh light on the complex interplays between olfactory and trigeminal systems in nasal chemoreception [30], and could further understanding of olfactory functions in patients with conditions like nasal obstruction or empty nose syndrome [31].

For terrestrial animals, odour propagation relies on airflow dynamics, and the latter directly affects the number of odour molecules reaching the olfactory epithelium. To estimate the concentration of odorant at the source, the olfactory system conceivably needs to integrate information regarding airflow. Teghtsoonian and colleagues proposed about 40 years ago that this is achieved by utilizing perceived sniff vigour [16,17,25]. Our results challenge this view. In particular, we found that altering the sensation of nasal flow with a local anaesthetic, without influencing perceived sniff vigour or any other aspect of sniffing, was sufficient to change odour intensity perception (Experiment 4), which strongly argues that sensed nasal flow, rather than sniff vigour, is weighted in the central processing of olfactory intensity. As nasal flow dynamics are not entirely driven by sniff kinetics (e.g. nasal congestion, air puffed in, retronasal flow etc.), we note that it is also computationally more advantageous for the brain to estimate odour concentration based on the former than the latter.

At the neural level, the circuit basis of nasal flow's contribution to olfactory processing awaits further examination. Certain olfactory receptors cause the olfactory sensory neurons to respond to both chemical and mechanical stimuli by a shared second-messenger cascade [6,7]. Airflow-driven mechanical signals detected by olfactory sensory neurons have been proposed to facilitate phase coding of odour identity [8,32], yet the perceptual effect appears to be weak [33]. Some trigeminal ganglion cells with sensory endings in the nasal epithelium also have branches reaching directly into both the olfactory bulb and the spinal trigeminal complex [34], although whether they form a functional signalling pathway remains questionable [35]. Physiological studies in mice and rabbits have shown that sniffing in the absence of odour input drives activities of mitral and tufted cells in the olfactory bulb, which likely informs downstream centres of nasal flow dynamics [36,37], and blocking the trigeminal nerves at the level of the ganglia increases olfactory bulb excitability [38]. Furthermore, neurons in the piriform cortex, the largest of the central olfactory areas in terms of volume and also the major recipient of bulbar projections [39], seem to possess the capacity to encode for stimulus modality (olfactory versus trigeminal) by differential patterns of firing [40]. As bilateral olfactory tracts and piriform cortices are interconnected via the anterior olfactory nucleus [12,13,39,41], inter-hemispheric transfer of olfactory and/or mechanosensory information likely contributes to the perceptual outcome, whereby contralateral nasal airflow could influence the processing and perception of unilateral olfactory input. We hence conjecture that nasal flow information is extracted and analysed at multiple stages along the olfactory hierarchy. Critically, it engages high-order olfactory processing that synthesizes inputs from both nostrils, serving as an integral part of the olfactory percept [42].

Ethics. The study was approved by the Institutional Review Board at the Institute of Psychology, Chinese Academy of Sciences. Written informed consent and consent to publish were obtained from participants in accordance with the ethical standards of the Declaration of Helsinki (1964).

Data accessibility. All behavioural data are provided in the electronic supplementary material files.

Authors' contributions. F.Y. and W.Z. designed the study. Testing and data collection were performed by F.Y. and Y.Y. F.Y. performed the data analysis and interpretation under the supervision of W.Z., F.Y. and W.Z. wrote the manuscript.

Competing interests. The authors declare no competing financial interests.

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